

# Musculoskeletal Diseases

Diagnostic Imaging and Interventional Techniques

### Editors

J. Hodler G.K. von Schulthess Ch. L. Zollikofer





### Musculoskeletal Diseases

Diagnostic Imaging and Interventional Techniques

# MUSCULOSKELETAL DISEASES

DIAGNOSTIC IMAGING AND INTERVENTIONAL TECHNIQUES

37th International Diagnostic Course in Davos (IDKD) *Davos, April 2-8, 2005* 

*including the* Pediatric Satellite Course "Kangaroo" Davos, April 2-3, 2005

presented by the Foundation for the Advancement of Education in Medical Radiology, Zurich





J. HODLER Department of Radiology University Hospital Balgrist Zurich, Switzerland

CH. L. ZOLLIKOFER Kantonsspital Institut für Radiologie Winterthur, Switzerland G. K. VON SCHULTHESS Universitätsspital Nuklearmedizin Zurich, Switzerland

Library of Congress Control Number: 2005922183

ISBN 88-470-0318-0 Springer Milan Berlin Heidelberg New York

This work is subject to copyright. All rights are reserved, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, re-use of illustrations, recitation, broadcasting, reproduction on microfilms or in other ways, and storage in data banks. Duplication of this publication or parts thereof is only permitted under the provisions of the Italian Copyright Law in its current version, and permission for use must always be obtained from Springer. Violations are liable for prosecution under the Italian Copyright Law.

Springer is a part of Springer Science+Business Media

springeronline.com

© Springer-Verlag Italia 2005 Printed in Italy

The use of general descriptive names, registered names, trademarks, etc., in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

Product liability: The publisher cannot guarantee the accuracy of any information about dosage and application contained in this book. In every individual case the user must check such information by consulting the relevant literature.

Typesetting: Kley & Sebastianelli Srl, Milan Printing and binding: Grafiche Porpora, Cernusco sul Naviglio (Milan) Cover design: Simona Colombo

Printed in Italy

### Preface

The International Diagnostic Course in Davos (IDKD) offers a unique learning experience for imaging specialists in training as well as for experienced radiologists and clinicians wishing to be updated on the current state of the art and the latest developments in the fields of imaging and image-guided interventions.

This annual course is focused on organ systems and diseases rather than on modalities. This year's program deals with diseases of the musculoskeletal system. During the course, the topics are discussed in group seminars and in plenary sessions with lectures by world-renowned experts and teachers. While the seminars present state-of-the-art summaries, the lectures are oriented towards future developments.

This syllabus represents a condensed version of the contents presented under the 20 topics dealing with imaging and interventional therapies in the musculoskeletal radiology. The topics encompass all the relevant imaging modalities including conventional x-rays, computed tomography, nuclear medicine, ultrasound and magnetic resonance angiography, as well as image-guided interventional techniques.

The volume is designed to be an "*aide-mémoire*" for the course participants so that they can fully concentrate on the lectures and participate in the discussions without the need of taking notes. Additional information is found on the web page of the IDKD (http://:www.idkd.ch).

J. Hodler G.K. von Schulthess Ch.L. Zollikofer

## Table of Contents

### Seminars

Shoulder J. Beltran, M. Recht	3
Magnetic Resonance Imaging of the Elbow       C. Chung, L. Steinbach	7
Radiology of Hand and Wrist InjuriesA.J. Wilson1	3
Wrist and Hand L.A. Gilula	7
Imaging of the Painful Hip and PelvisC.W.A. Pfirrmann, C.A. Petersilge2	21
Imaging of the KneeD.A. Rubin, W.E. Palmer2	26
Imaging of the Foot and AnkleZ.S. Rosemberg, M. Zanetti3	9
Magnetic Resonance Imaging of MuscleM.N. Pathria, R.D. Boutin4	8
Soft Tissue Tumors and Tumo-Like Masses: A Systematic Approachto DiagnosisM.J. Kransdorf, M.D. Murphey5	54
Tumors and Tumor-Like Lesions of BoneM. Sundaram, D. Vanel6.	52
Imaging of Bone Marrow DisordersB. Vande Berg, J. Malghem, F. Lecouvet, B. Maldagu6	68
Bone Marrow DisordersA. Stäbler7	'3
Metabolic and Systemic Bone DiseasesJ. Freyschmidt8	3
Metabolic Bone Disease     J.E. Adams   8	\$9

<b>The Radiology of Hip and Knee Joint Prostheses</b> I. Watt, B.N. Weissman106
Traumas of the Axial SkeletonH. Imhof, G.Y. El-Khoury112
Trauma of the Appendicular SkeletonJ.J. Kaye, M.K. Dalinka121
Inflammatory Diseases of the SpineV. Jevtic, V. Pullicino127
Degenerative Diseases of the Spine D. Weishaupt, I. McCall
Osteomyelitis and Septic Arthritis D. Forrester, R.F. Kilcoyne
Peripheral Arthritis L.F. Rogers, C.S. Resnik
<b>Special Aspects of Musculoskeletal Imaging in Children</b> D. Jaramillo, G. Sebag
Musculoskeletal Sonography S. Bianchi, S. Marcelis

### Pediatric Satellite Course "Kangaroo"



The Spectrum of Non-Accidental Injury and Its Imitators in ChildrenP.K. Kleinman1	69
Contrast Enhancement of the Growing Skeleton: Rationale and Optimization in Pediatric MRI	
G. Sebag 1	75
Imaging the Osseous and Soft Tissue Tumors in the ChildA. Geoffray1	80
Imaging the Child's Inflammatory and Infectious Musculoskeletal Pathology	
S.G.F. Robben	85

# **SEMINARS**

### Shoulder

J. Beltran<sup>1</sup>, M. Recht<sup>2</sup>

<sup>1</sup> Department of Radiology, Maimonides Medical Center, Brooklin, NY, USA

<sup>2</sup> Department of E-Radiology, The Cleveland Clinic Foundation, Cleveland, OH, USA

### Introduction

This seminar places special emphasis on the MRI manifestations of shoulder pathology. The discussion includes the following topics:

1. Rotator cuff pathology and impingement lesions.

2. Glenohumeral instability and related lesions.

3. Miscellaneous shoulder conditions.

# **Rotator Cuff Pathology and Impingement Lesions**

Impingement syndrome is a clinical entity produced by compression of the supraspinatus tendon under the region of the acromial arch, and it can be related to abnormal morphology of the acromion process, thickening of the coracoacromial ligament, subacromial spurring, or degenerative arthritis of the acromioclavicular joint. Alternatively, it can be related to degeneration, repeated trauma or overuse during overhead exercise, such as swimming. Normal anatomical variants, such as type III undersurface of the acromion with a hooked configuration and os acromiale, have been described associated with rotator cuff impingement and tears.

There are two types of impingement syndrome: primary, associated with abnormalities in the coracoacromial arch; and secondary to rotator cuff dysfunction. The secondary form of rotator cuff impingement may be further subdivided into two types: internal and external. The internal type refers to the articular surface side of the rotator cuff and it is often termed posterosuperior impingement syndrome. The external variety occurs as a result of external compression of the anterior aspect of the cuff in the bursal side and includes the coracoid impingement syndrome. Posterosuperior impingement syndrome occurs in the throwing athlete as a result of continuous strain of the anterior capsular mechanism, which leads to laxity and anterior subluxation of the glenohumeral joint with the arm in abduction and external rotation. This situation produces impingement of the supraspinatus tendon at the level of its insertion in the greater tuberosity

of the humerus as well as small impaction fractures and posterosuperior labral lesions. The coracoid impingement syndrome may occur when the distance between the posterior aspect of the coracoid process and the humerus is decreased, producing compression of the rotator cuff, mainly the subscapularis tendon.

Inflammatory changes within the supraspinatus tendon can be seen during the early phases of the disease, along with subacromial bursitis, but this can progress into rotator cuff tear. Three histological stages of impingement syndrome have been described. In stage I, edema and hemorrhage of the subacromial soft tissues are present. In stage II, there is fibrosis and thickening, while in stage III, partial or complete rotator cuff tears are seen.

Full-thickness rotator cuff tears involve most often the supraspinatus tendon, but they can also extend to the infraspinatus and subscapularis tendons. Tear of the teres minor is very rare. Partial-thickness rotator cuff tears may involve the articular or the bursal surfaces, or they may be located within the substance of the tendon. Delaminating tears of the rotator cuff can be partial or full thickness. They extend in the longitudinal direction of the tendon fibers, and there may be different degrees of retraction of the various layers. Delaminating tears may be associated with fluid collections extending from the tear into the muscle (sentinel cyst). Full-thickness tears allow communication between the articular space of the glenohumeral joint and the subacromial-subdeltoid bursa, unless the tear is covered by granulation or scar tissue. On rare occasions, tears may involve the rotator cuff interval, with capsular disruption. Tears of the rotator cuff interval may be associated with lesions of the structures present within this anatomical space, namely, the long head of the biceps tendon, the coracoacromial ligament, the superior glenohumeral ligament and also the superior labrum.

### **Glenohumeral Instability and Related Lesions**

Restraints to anterior translation of the humeral head are provided by the capsule and the glenohumeral ligaments (GHL). The labrum is torn as part of the avulsion forces produced by the GHL at the time of the injury. Anteroinferior dislocation is the most frequent cause of anterior glenohumeral instability. A single event originates a constellation of lesions leading to other episodes of dislocation or subluxation. The lesions that may take place during an anteroinferior dislocation include anteroinferior labral tear, tear of the inferior GHL (IGHL) and/or capsular-periosteal stripping, fracture of the anteroinferior glenoid margin and compression fracture of the superior lateral aspect of the humeral head (Hill-Sachs lesion).

The classic Bankart lesion is the combination of anterior labral tear and capsuloperiosteal stripping. On arthroscopy, the Bankart lesion is seen as a fragment of labrum attached to the anterior band of the IGHL and to the ruptured scapular periosteum, "floating" in the anterior-inferior aspect of the glenohumeral joint. Extensive bone and soft-tissue damage and persistent instability may lead to multidirectional instability, resulting in episodes of posterior dislocation.

A number of variants of anterior labral tears have been described. The Perthes lesion is similar to the Bankart lesion, but without the tear of the capsule. Anterior labroligamentous periosteal sleeve avulsion (ALPSA) refers to a tear of the anteroinferior labrum, with associated capsuloperiosteal stripping. The torn labrum is rotated medially, and a small cleft or separation can be seen between the glenoid margin and the labrum. In contrast to the Bankart lesion, the ALPSA lesion can heal, leaving a deformed and patulous labrum. The glenoid labral articular disruption (GLAD) represents a tear of the anteroinferior labrum, attached to a fragment of articular cartilage, without associated capsuloperiosteal stripping.

Posterior shoulder dislocation more often occurs as a result of a violent muscle contraction, e.g., by electrical shock or seizures. After the acute episode of dislocation, the arm frequently remains locked in adduction and internal rotation. Posterior instability caused by repeated micro-trauma, without frank dislocation, may cause persistent shoulder pain in young athletes. Abduction, flexion and internal rotation are the mechanism involved in these cases (swimming, throwing, and punching). This may be also associated with posterior capsular laxity. Lesions that may occur during posterior dislocation or in cases of repeated micro-trauma include posterior labral tear, posterior capsular stripping or laxity, fracture, erosion, or sclerosis and ectopic bone formation of the posterior glenoid, and vertical impacted fracture of the anterior aspect of the humeral head (reverse Hill-Sachs, McLaughlin fracture).

Superior labral anterior and superior lesions (SLAP lesions) are not as rare as originally thought. These lesions involve the superior part of the labrum with varying degrees of biceps tendon involvement. Pain, clicking, and occasional instability in a young patient are the typical clinical manifestations. Four types of SLAP lesions were originally described based on arthroscopic findings. Type I is a partial tear of the superior part of the labrum with fibrillation of the LHBT. Type II is an avulsion of the LHBT with tear of the anterior and posterior labrum. Type III is a bucket-handle tear of the labrum and type IV is a bucket-handle tear of the labrum with longitudinal tear to the LHBT. More recently, up to ten types of SLAP lesions have been described, representing a combination of superior labral tears with extension into different areas of the labrum and gleno-humeral ligaments.

### **Miscellaneous Lesions**

The following lesions are discussed:

- a. Biceps tendon
- b. Compressive neuropathies
- d. Inflammatory and other miscellaneous lesions

### **Biceps Tendon**

Tendinosis or tenosynovitis of the LBT may occur in association with shoulder impingement syndrome and rotator cuff tears, where the intracapsular portion of the LBT is compressed between the humeral head, the acromion, and the coracoacromial ligament during abduction and rotation of the arm. Attritional tendinosis is associated with a narrow bicipital groove and hence it affects the extracapsular portion of the tendon. Magnetic resonance imaging (MRI) may demonstrate fluid in the joint extending into the bicipital grove, although this a non-specific sign unless the fluid completely surrounds the tendon, in the absence of a joint effusion. Trauma and degeneration may involve the LBT, producing swelling and increased signal intensity (SI) on T2 and T2\* pulse sequences.

Complete rupture of the LBT more often occurs proximally, at the level of the proximal portion of the extracapsular segment, within the groove. MRI demonstrates the absence of the LBT in the groove and its distal displacement. Intracapsular tears of the LBT are seen more often in patients with rotator cuff tears. Attritional tendinosis affecting the intertubercular portion of the LBT can progress to longitudinal splits within the tendon, resulting in thickening of the LBT with increased intrasubstance SI on T2-weighted images. A bifid LBT (normal variant) should not be confused with a partial longitudinal tear.

Biceps tendon dislocation occurs with tears of the subscapularis tendon and coracohumeral ligament. Two types of dislocation of the LBT have been described, depending on whether the tendon is located in front or behind the subscapularis tendon. In the first type, the insertional fibers of the subscapularis tendon are intact. In the second type, the subscapularis tendon is detached and the LBT is medially displaced, becoming entrapped intraarticularly.

### **Compressive Neuropathies**

The suprascapular nerve and its branches can become compressed or entrapped by stretching due to repetitive scapular motion, or they can be damaged by scapular fractures, overhead activities, soft-tissue masses or direct trauma. T2-weighted images can show hyperintensity of the involved muscle. Nerve thickening and muscle atrophy due to denervation may be noted in advanced cases. Ganglion cysts at the scapular incisura typically associated with posterior labral tears can be easily detected by MRI of the shoulder.

The quadrilateral space syndrome is caused by compression of the axillary nerve at the quadrilateral space. The teres minor and deltoid muscles and the posterolateral cutaneous region of the shoulder and upper arm are innervated by the axillary nerve. Proximal humeral and scapular fractures, shoulder dislocations, or axillary mass lesions can result in damage or compression of the axillary nerve. Entrapment of this nerve can also be produced by extreme abduction of the arm during sleep, hypertrophy of the teres minor muscle in paraplegic patients or by a fibrous band within the quadrilateral space. Patients may have shoulder pain and paresthesia. In advanced cases, atrophy of the deltoid and teres minor muscles can occur, but more often there is selective atrophy of the teres minor muscle.

Parsonage-Turner syndrome, also referred to as acute brachial neuritis, is clinically characterized by sudden onset of severe atraumatic pain in the shoulder girdle. The pain typically decreases spontaneously in 1-3 weeks, and is followed by weakness of at least one of the muscles about the shoulder. The exact etiology has not been established but viral and immunological causes have been considered. MRI findings in the acute stage include diffuse increased SI on T2-weighted images consistent with interstitial muscle edema associated with denervation. The most commonly affected muscles are those innervated by the suprascapular nerve, including the supra- and infraspinatus. The deltoid muscle can also be compromised in cases of axillary nerve involvement. Later in the course of the disease, there may be muscle atrophy, manifested by decreased muscle bulk.

#### Inflammatory and Other Miscellaneous Lesions

The manifestations of idiopathic synovial osteochondromatosis on MRI depend on the degree of calcification or ossification of the cartilaginous bodies. If no calcification is present, it may simulate a joint effusion, with low SI on T1-weighted images and high SI on T2-weighted images. However, high-resolution MRI may be able to demonstrate a signal that is more inhomogeneous than fluid. If calcifications are present, these will manifest themselves as multiple small foci of decreased SI on both T1- and T2-weighted pulse sequences, surrounded by high SI haloes on T2-weighted images, which represent the cartilaginous coverage. The presence of low-SI material mixed with hyperintense cartilage may mimic pigmented villonodular synovitis, especially if bone erosions are present. Other differential diagnostic considerations include entities that can produce multiple intra-articular bodies, such as osteocartilaginous loose bodies related to osteoarthritis or osteochondral trauma, and "rice bodies", such as those seen in rheumatoid arthritis and tuberculosis (see below).

The appearance of PVNS on MRI is quite distinct due to the paramagnetic effect of the hemosiderin deposits, which produces characteristic foci of low SI on T1- and T2-weighted sequences. An heterogeneous pattern is also frequently observed, due to the presence of areas of low hemosiderin deposition and associated joint effusion. The paramagnetic effect of hemosiderin is enhanced on gradient-echo pulse sequences. Associated ancillary findings, such as bone erosions and capsular distension, are often seen in the diffuse form of PVNS. The differential diagnosis of hypointense intra-articular material includes urate crystals of gout, synovial osteochondromatosis, and amyloid deposition.

MRI of rheumatoid arthritis shows joint effusion, subacromial-subdeltoid bursitis, rotator cuff tendinosis and tears secondary to the effect of the inflamed synovium on the undersurface of the tendons, and "rice bodies". Chronic articular inflammation evolves into proliferation of elongated synovial villi that become fibrotic and eventually detach, producing grains similar to polished rice. On MRI, these "rice bodies" manifest themselves as numerous rounded nodules of intermediate SI occupying the joint space and/or the subacromial bursa. Similar findings can be seen in tuberculous arthritis and even synovial chondromatosis.

### Suggested Readings

- Basset RW, Cofield RH (1983) Acute tears of the rotator cuff: the timing of surgical repair. Clin Orthop 175:18-24
- Beltran J, Bencardino J, Mellado J, Rosenberg ZS, Irish RD (1997) MR arthrography of the shoulder: Variations and pitfalls. Radiographics 17:1403-1412
- Beltran J, Rosenberg ZS, Chandanani VP, Cuomo F, Beltran S, Rokito A (1997) Glenohumeral instability: evaluation with MR arthrography. Radiographics 3:657-673
- Blacksin MF, Ghelman B, Freiberger RH, Salvati E (1990) Synovial chondromatosis of the hip: evaluation with air computed arthrotomography. Clin Imaging 14:315-318
- Bureau NJ, Dussault RG, Keats TE (1996) Imaging of bursae around the shoulder joint. Skeletal Radiol 25:513-517
- Burkhead WZ Jr (1990) The biceps tendon. In: Rockwood CA Jr, Matsen III FA (eds): The shoulder. WB Saunders, Philadelphia, p 791
- Campeau NG, Lewis BD (1998) Ultrasound appearance of synovial osteochondromatosis of the shoulder. Mayo Clin Proc 73:1079-1081
- Cervilla V, Schweitzer ME, Ho C, Motta A, Kerr R, Resnick D (1991) Medial dislocation of the biceps brachii tendon: appearance at MR imaging. Radiology 180(2):523-526
- Chung C, Coley BD, Martin LC (1998) Rice bodies in juvenile rheumatoid arthritis. Am J Roentgenol 170:698-700
- Chung CB, Dwek JR, Feng S, Resnick D (2001) MR arthrography of the glenohumeral joint: a tailored approach. AJR 177:217-219

- Crotty JM, Monu JU, Pope TL Jr (1996) Synovial osteochondromatosis. Radiol Clin North Am 34:327-342
- Deutsch A, Altchek DW, Veltri DM, Potter HG, Warren RF (1997) Traumatic tears of the subscapularis tendon. Clinical diagnosis, magnetic resonance imaging findings, and operative treatment. Am J Sports Med 25:13-22
- Dzioba RB, Quinlan WJ (1984) Avascular necrosis of the glenoid. J Trauma 24:448-451
- Erickson SJ, Fitzgerald SW, Quinn SF, Carrera GF, Black KP, Lawson TL (1992) Long bicipital tendon of the shoulder: normal anatomy and pathologic findings on MR imaging. AJR 158:1091-1096
- Farber JM, Buckwalter KA. (2002) Sports-related injuries of the shoulder: instability. Radiol Clin N Am; 235-249
- Fleckenstein JL, Watumull D, Conner KE et al (1993) Denervated human skeletal muscle: MR imaging evaluation. Radiology 187:213-218
- Greenan TJ, Zlatkin MB, Dalinka MK, Estehai JL (1993) Posttraumatic changes in the posterior glenoid and labrum in a handball player. Am J Sports Med 21:153-156
- Fritz RC, Helms CA, Steinbach LS, Genant HK. (1992) Suprascapular nerve entrapment: evaluation with MR imaging. Radiology 182:437-444
- Helms CA, Martinez S, Speer KP (1999) Acute brachial neuritis (Parsonage-Turner syndrome): MR imaging appearance-report of three cases. Radiology 207:255-259
- Jee WH, McCauley TR, Katz LD, Matheny JM, Ruwe PA, Daigneault JP (2001) Superior labral anterior posterior (SLAP) lesions of the glenoid labrum: reliability and accuracy of MR arthrography for diagnosis. Radiology 218:127-132
- Kramer J, Recht M, Deely DM, Schweitzer M, Pathria MN et al (1993) MR appearance of idiopathic synovial osteochondromatosis. J Comput Assist Tomogr 17:772-776
- Lin J, Jacobson JA, Jamadar DA, Ellis JH (1999) Pigmented villonodular synovitis and related lesions: the spectrum of imaging findings. Am J Roentgenol 172:191-197
- Linker CS, Helms CA, Fritz RC (1993) Quadrilateral space syndrome: evaluation of median nerve circulation with dynamic contrast-enhanced MR imaging. Radiology 188:675-676
- McCarty DJ, Halverson PB, Carrera GF et al (1981) "Milwaukee shoulder" – association of microspheroids containing hydrox-

yapatite crystals, active collagenase, and neutral protease with rotator cuff defects. I Clinical aspects. Arthritis Rheum 24:464-473

- Musgrave DS, Rodosky MW. (2001) SLAP Lesions: Current Concepts. Am J Orthop 1:29-38
- Neviaser TJ (1993) The anterior labroligamentous periosteal sleeve avulsion lesion: A cause of anterior Instability of the shoulder. Arthroscopy 9:17-21
- Parsonage MJ, Turner JWA (1948) Neuralgic amyotrophy. The shoulder-girdle syndrome. Lancet 1:973-8
- Pecina MM, Krmpotic-Nemanic J, Markiewitz AD. (1991) Tunnel syndromes in the upper extremities. In: Pecina MM, Krmpotic-Nemanic J, Markiewitz AD (eds) Tunnel syndromes. CRC, New York, 29-53
- Rokito AS, Bilgen OF, Zuckerman JD, Cuomo F (1996) Medial dislocation of the long head of the biceps tendon. Magnetic resonance imaging evaluation. Am J Orthop 25:314, 318-323
- Shanley DJ, Mulligan ME (1990) Osteochondrosis dissecans of the glenoid. Skeletal Radiol 19:419-421
- Snyder SJ: Shoulder Arthroscopy. MacGraw-Hill, New York, 1994, pp 121-124
- Tirman PF, Feller JF, Janzen DL, Peterfy CG, Bergman AG (1994) Association of glenoid labral cysts with labral tears and glenohumeral instability: radiologic findings and clinical significance. Radiology 190:653-658
- Tuckman GA (1994) Abnormalities of the long head of the biceps tendon of the shoulder: MR imaging findings. Am J Roentgenol 163:1183-1188
- Uetani M, Kuniaki H, Matsunaga N, Imamura K, Ito N (1983) Denervated skeletal muscle: MR imaging. Radiology 189:511-515
- Walch G, Nove-Josserand L, Boileau P, Levigne C (1998) Subluxations and dislocations of the tendon of the long head of the biceps. J Shoulder Elbow Surg 7:100-108
- Yu JS, Greenway G, Resnick D (1998) Osteochondral defect of the glenoid fossa: Cross-sectional imaging features. Radiology 206:35-40
- Zanetti M, Weishaupt D, Jost B, Gerber C, Hodler J (1999) MR imaging for traumatic tears of the rotator cuff: High prevalence of greater tuberosity fractures and subscapularis tendon tears. Am J Roentgenol 172:463-467

### Magnetic Resonance Imaging of the Elbow

C. Chung<sup>1</sup>, L. Steinbach<sup>2</sup>

<sup>1</sup> University of California, San Diego, and VAHCS, CA, USA

<sup>2</sup> Musculoskeletal Imaging, University of California San Francisco, San Francisco, CA, USA

Elbow injuries are common, especially in the athlete, and can be basically classified into acute or chronic injuries. The following discussion of magnetic resonance imaging (MRI) of the elbow will address variations in normal anatomy that represent pitfalls in imaging diagnosis, and commonly encountered osseous and soft-tissue pathology.

### **Osseous Anatomic Considerations and Pathology**

The lateral articulating surface of the humerus is formed by the capitellum, a smooth, rounded prominence that arises from its anterior and inferior surfaces. As it does so, its width decreases from anterior to posterior. This morphology of the capitellum (smooth surface), in conjunction with the knowledge that the adjacent lateral epicondyle (rough surface) is a posteriorly oriented osseous projection of the distal humerus, explains the pseudodefect of the capitellum which must be distinguished from a post-traumatic osteochondral lesion [1].

The articular surface of the proximal ulna is formed by the combination of the posterior olecranon and the anterior coronoid processes, with the articular surfaces taking the configuration of a figure of eight. At the waist of the eight, or junction between anterior and posterior aspects of the ulna, the articular surface is traversed by a cartilage-free bony ridge. This trochlear ridge is 2 to 3 mm wide and is at the same height as the adjacent cartilaginous surface. It should not be mistaken for a central osteophyte. The waist of the figure of eight is formed by the tapered central surfaces of the coronoid and olecranon processes both medially and laterally, forming small cortical notches devoid of cartilage. On sagittal MRI, these focal regions devoid of cartilage could be mistaken for a focal chondral lesion [2].

### **Osteochondral Lesions**

In the case of acute medial elbow injury, the involvement of a valgus force is usually described as one of the most common mechanisms of injury [3]. Subchondral bone and cartilage injuries that occur in this setting result from

impaction and shearing forces applied to the articular surfaces. The overall configuration of the humeroradial articulation, in this case, can be likened to a mortar and pestle, with the capitellar articular surface impacting that of the radius to result in a chondral or osteochondral lesion of the capitellar surface. These acute post-traumatic lesions are manifested on MRI as irregularity of the chondral surface, disruption or irregularity of the subchondral bone plate, and or the presence of a fracture line. The acuity of the lesion and a post-traumatic etiology are implied by the presence of marrow edema and joint effusion. Close inspection of the location of the lesion on coronal and sagittal MRI is of the utmost importance in order to distinguish a true osteochondral lesion from the pseudodefect of the capitellum. Correlation with presenting clinical history is also helpful in determining the etiology of imaging findings.

The entity of osteochondritis dissecans remains controversial, primarily due to debate over its etiology. The precise relationship of osteochondritis dissecans and an osteochondral fracture is unclear, but many investigators regard the former as a post-traumatic abnormality that may lead to osteonecrosis. Osteochondritis dissecans is thought to occur in immature athletes between 11 and 15 years of age, rarely in adults [4]. Osteochondritis dissecans of the elbow involves primarily the capitellum, but reports have described this process in the radius and trochlea [5].

Regardless of the etiology of the osteochondral injury, the role of imaging is to provide information regarding the integrity of the overlying articular cartilage, the viability of the separated fragment, and the presence of associated intra-articular bodies. Both computed tomography (CT) and MRI with and without arthrography can provide this information to varying degrees, although no scientific investigation has been performed to date that establishes specific indications for each study. MRI, with its excellent soft-tissue contrast, allows direct visualization of the articular cartilage, as well as of the character of the interface of the osteochondral lesion with native bone (Fig. 1). The presence of joint fluid or granulation tissue at this interface, manifested as increased signal intensity on fluid-sensitive MRI, generally indicates an unstable lesion. The in-





**Fig. 1.** A Conventional radiograph demonstrates a lytic osteochondral lesion in the capitellum (*arrow*). **B** This lesion is low signal intensity on a T1-weighted image and has a high signal intensity rim on a T2-weighted axial image, **C** suggesting instability (*arrow*)

troduction of contrast into the articulation in conjunction with MRI can be helpful in two ways: (1) to facilitate the identification of intra-articular bodies, and (2) to establish communication of the bone-fragment interface with the articulation by following the route of contrast, providing even stronger evidence for an unstable fragment [6, 7].

### **Ligament Pathology**

### Valgus Instability

The principle function of the ulnar collateral ligament complex is to maintain medial joint stability to valgus stress. The anterior bundle is the most important component of the ligamentous complex to this end, as it serves as the primary medial stabilizer of the elbow from 30 to 120 degrees of flexion. The most common mechanisms of ulnar collateral ligament insufficiency are chronic attenuation, as seen in overhead or throwing athletes, and post-traumatic, usually after a fall on the outstretched arm. In the case of the latter, an acute tear of the ulnar collateral ligament may be encountered.

With throwing sports, high valgus stresses are placed on the medial aspect of the elbow. The maximum stress on the ulnar collateral ligament occurs during the late cocking and acceleration phases of throwing [8]. Repetitive insults to the ligament allow microscopic tears that progress to significant attenuation or frank tearing within its substance (Fig. 2). While MRI facilitates direct



**Fig. 2.** Coronal FSE T2-weighted image with fat suppression shows a full-thickness tear of the anterior band of the ulnar collateral ligament at the attachment to the sublime tubercle (*arrow*)

visualization of the ligament complex, in chronic cases, the development of heterotopic calcification along the course of the ligament has been described [9].

#### Varus Instability

Lateral elbow instability related to isolated abnormalities of the lateral collateral ligament complex is not as well described as that on the medial side of the elbow. If it were to occur, the mechanism would be a stress or force applied to the medial side of the articulation, resulting in compression on that side, with opening of the lateral articulation and subsequent insufficiency of the radial collateral ligament. As the radial collateral ligament attaches on and is intimately associated with the annular ligament, an abnormality discovered in one of the structures obligates careful inspection of the other.

Varus stress applied to the elbow may occur as an acute injury, but rarely as a repetitive stress, as encountered on the medial side. While lateral collateral ligament injuries rarely occur as the result of an isolated varus stress, other causes can commonly lead to this injury, including dislocation, subluxation and overly aggressive surgery (release of the common extensor tendon or radial head resection).

Varus instability is also tested with the elbow in full extension and 30 degrees of flexion to unlock the olecranon. A varus stress is applied to the elbow while palpating the lateral joint line.

#### Posterolateral Rotary Instability and Elbow Dislocation

The subject of elbow instability is complex and has been a challenge due to the difficulty in establishing the mechanism of injury and reliable clinical tests for diagnosis. With the realization that elbow instability is more common than previously thought, marked advances in the understanding of this entity are occurring.

For recurrent instability, posterolateral rotary instability is the most common pattern. This type of instability represents a spectrum of pathology consisting of three stages, according to the degree of soft-tissue disruption. In stage 1, there is posterolateral subluxation of the ulna on the humerus that results in insufficiency of the lateral ulnar collateral ligament (Fig. 3) [10, 11, 12]. In stage 2, the elbow dislocates incompletely so that the coronoid is perched under the trochlea. In this stage, the radial collateral ligament, and anterior and posterior portions of the capsule are disrupted, in addition to the lateral ulnar collateral ligament. Finally, in stage 3, the elbow dislocates fully so that the coronoid rests behind the humerus. Stage 3 is subclassified into three further categories. In stage 3A, the anterior band of the medial collateral ligament is intact and the elbow is stable to valgus stress after reduction. In stage 3B, the anterior band of the medial collateral ligament is disrupted so that the elbow is unstable with valgus stress. In stage 3C, the entire distal humerus is stripped of soft tissues, rendering the elbow grossly unstable even when a splint or cast is applied with the el-



**Fig. 3.** Coronal-fat-suppressed T1-weighted image reveals fullthickness tears of the proximal aspects of the lateral ulnar collateral ligament and extensor tendon at the lateral epicondyle (*arrow*)

bow in a semi-flexed position. This classification system is helpful, as each stage has specific clinical, radiographic and pathologic features that are predictable and have implications for treatment [10].

Subluxation or dislocation of the elbow can be associated with fractures. Fracture-dislocations most commonly involve the coronoid and radial head, a constellation of findings referred to as the "terrible triad" of the elbow, as the injury complex is difficult to treat and prone to unsatisfactory results [10]. Radial-head fractures do not cause clinically significant instability unless the medial collateral ligament is disrupted. An important feature of elbow injuries to recognize is that the small flake fracture of the coronoid, commonly seen in elbow dislocations, is not an avulsion fracture. Nothing attaches to the very tip of the coronoid; rather, the capsule attaches on the downward slope of the coronoid, the brachialis even more distally. This fracture is a shear fracture and is likely pathognomonic of an episode of elbow subluxation or dislocation. A second consideration with respect to elbow dislocation is that, as the ring of soft tissues is disrupted from posterolateral to medial, the capsule is torn and insufficient. In the absence of an intact capsule, joint fluid dissects through the soft-tissue planes of the forearm, negating an indirect radiographic sign of trauma in the elbow, that of joint effusion.

### **Tendon Pathology**

The many muscles about the elbow can be divided into four groups: posterior, anterior, medial and lateral. The muscles of the posterior group are the triceps and anconeus. The muscles of the anterior group are the biceps brachii and brachialis. The muscles in the medial group are the pronator teres, the palmaris longus and the flexors of the hand and wrist. The muscles in the lateral group include the supinator, brachioradialis and extensor muscles of the hand and wrist. The vast majority of pathology encountered in the flexor and extensor groups will be isolated to the common flexor and common extensor tendons.

The classification of tendon injuries about the elbow can be organized by location, acuity and degree of injury. Tendon injury related to a single isolated event is uncommon, although exceptions to this rule do occur. More commonly, tendinous injuries in this location relate to chronic repetitive micro-trauma. MRI is particularly well suited, with its excellent soft-tissue contrast, to diagnose tendon pathology. This is done primarily by close inspection of signal intensity and morphology of the tendons. As elsewhere in the body, the tendons about the elbow should be smooth, linear structures of low signal intensity. Abnormal morphology (attenuation or thickening) can be seen in tendinosis or tear. If signal intensity becomes bright or increased on fluid-sensitive sequences within the substance of a tendon, a tear is present. Tears can be further characterized as partial or complete. A complete tear is diagnosed by a focal area of discontinuity (Fig. 3).

#### **Epicondylitis and Overuse Syndromes**

Chronic stress applied to the elbow is the most frequent injury in athletes, and a spectrum of pathology can exist with varying degrees of severity. The frequency of involvement of the common flexor and extensor tendons to the medial and lateral epicondyles, respectively, has led to the designation of "epicondylitis" as a general term applied to these overuse syndromes. Anatomically, they are classified by location and are further associated with sports that incite the pathology. The injury is believed to result from extrinsic tensile overload of the tendon, which, over time, produces microscopic tears that do not heal appropriately.

Although these overuse entities about the elbow have been termed "epicondylitis" for the purpose of clinical diagnosis, inflammatory osseous changes rarely occur. The imaging findings are those reflecting chronic change in the tendon, as evidenced by tendinosis alone, or in conjunction with partial or complete tear. As previously mentioned, the distinction between types of pathology is made by consideration of both morphology and signal intensity changes.

Medial epicondylitis involves pathology of the common flexor tendon and is associated primarily with the sport of golfing. It has also been reported with javelin throwers, racquetball and squash players, swimmers and bowlers. The pronator teres and flexor carpi radialis tendons are involved most frequently, resulting in pain and tenderness to palpation over the anterior aspect of the medial epicondyle of the humerus and origin of the common flexor tendon. The mechanism of injury includes repetitive valgus strain with pain resulting from resisting pronation of the forearm or flexion of the wrist [13]. The imaging findings encountered can include tendinosis, or tendinosis with superimposed partial- or full-thickness tear. When assessing the tendon, it is necessary to closely scrutinize the underlying ulnar collateral ligament complex to ensure integrity.

Lateral epicondylitis is the most common problem in the elbow in athletes, and has been termed tennis elbow. This term may be somewhat inappropriate as 95% of cases of the clinical entity of lateral epicondylitis occur in non-tennis players [14]. Moreover, it has been estimated that 50% of people partaking in any sport with overhead arm motion will develop this process [15].

It is associated with repetitive and excessive use of the wrist extensors. The pathology most commonly affects the extensor carpi radialis brevis at the common extensor tendon. A number of investigators have described the pathology encountered in the degenerated tendon of this disease process. Histologically, necrosis, round-cell infiltration, focal calcification and scar formation have been shown [16]. In addition, invasion of blood vessels, fibroblastic proliferation, and lymphatic infiltration, the combination of which are referred to as angiofibroblastic hyperplasia, occur and ultimately lead to mucoid degeneration as the process continues [17, 18]. The absence of a significant inflammatory response has been emphasized repeatedly, and may explain the inadequacy of the healing process.

The imaging findings in this process are exactly those encountered in the clinical entity of medial epicondylitis (Fig. 4). As on the medial side, when pathology is encountered in the tendon, close scrutiny of the underlying ligamentous complex is necessary to exclude concomitant injury. In particular, thickening and tears of the lateral ulnar collateral ligament have been encountered with lateral epicondylitis [13].



**Fig. 4.** Coronal T1-weighted (*left*) and fat-suppressed FSE T2-weighted images show thickening and intermediate signal intensity in the common extensor tendon (*arrows*), consistent with tendinosis (lateral epicondylitis)

### **Biceps Tendon**

Rupture of the tendon of the biceps brachii muscle at the elbow is rare and constitutes less than 5% of all biceps tendon injuries [19]. It usually occurs in the dominant arm of males. Injuries to the musculotendinous junction have been reported, but the most common injury is complete avulsion of the tendon from the radial tuberosity. Although the injury often occurs acutely after a single traumatic event, the failure is thought to be due to pre-existing changes in the distal biceps tendon, due to intrinsic tendon degeneration, enthesopathy at the radial tuberosity, or cubital bursal changes. The typical mechanism of injury relates to forceful hyperextension applied to a flexed and supinated forearm. Athletes involved in strength sports, such as competitive weightlifting, football and rugby, often sustain this injury.

Clinically the patient describes a history of feeling a "pop" or sudden sharp pain in the antecubital fossa. The classic presentation of a complete distal biceps rupture is that of a mass in the antecubital fossa due to proximal migration of the biceps muscle belly. Accurate diagnosis is more difficult in cases of the rare partial tear of the tendon, or more common complete tear of the tendon without retraction. The latter can occur with an intact bicipital aponeurosis, which serves to tether the ruptured tendon to the pronator flexor muscle group.

MRI diagnosis of biceps tendon pathology becomes important in patients who do not present with the classic history or mass in the antecubital fossa, or for evaluation of the integrity of the lacertus fibrosus. MRI diagnosis of tendon pathology, as previously mentioned, is largely dependent on morphology, signal intensity and the identification of areas of tendon discontinuity (Fig. 5). In the



**Fig. 5.** Axial-fat-suppressed T2-weighted image shows complete disruption of the distal biceps at the radial tuberosity (*arrow*)

case of the biceps tendon, an important indirect sign of tendon pathology is the presence of cubital bursitis.

### **Triceps Tendon**

Rupture of the triceps tendon is quite rare. The mechanism of injury has been reported to result from a direct blow to the triceps insertion, or a deceleration force applied to the extended arm with contraction of the triceps, as in a fall. Similar to the pathology encountered in the distal biceps tendon, most ruptures occur at the insertion site, although musculotendinous junction and muscle belly injuries have been reported. Complete ruptures are more common than partial tears. Associated findings may include olecranon bursitis, subluxation of the ulnar nerve, or fracture of the radial head. Accurate clinical diagnosis relies on the presence of local pain, swelling, ecchymosis, a palpable defect, and partial or complete loss of the ability to extend the elbow. With more than 2 cm of retraction between the origin and the insertion, a 40%loss of extension strength can result [19].

For MRI diagnosis of triceps tendon pathology, it is imperative to be aware that the triceps tendon appearance is largely dependent on arm position. The tendon will appear lax and redundant when imaged in full extension, whereas it is taut in flexion. The MRI features of a tear are similar to those associated with any other tendon.

#### **Entrapment Neuropathy**

The ulnar, median and radial nerves may become compressed at the elbow, leading to symptoms of entrapment neuropathy. Abnormal nerves may have increased signal intensity on T2-weighted images, focal changes in girth, and deviation that may result from subluxation or displacement by an adjacent mass.

Ulnar nerve entrapment most commonly occurs in the cubital tunnel. Nerve compression may be caused by a medial trochlear osteophyte or incongruity between the trochlea and olecranon process [20]. Anatomic variations also contribute. The absence of the triangular reticulum, the anatomic roof of the cubital tunnel, occurs in about 10% of cases, permitting subluxation of the nerve with flexion. It is necessary, therefore, to include axial images of the flexed elbow in patients suspected of this disorder.

The presence of the anomalous anconeous epitrochlearis muscle over the cubital tunnel causes static compression of the nerve. In addition, there are many other causes of ulnar neuritis, including thickening of the overlying ulnar collateral ligament, medial epicondylitis, adhesions, muscle hypertrophy, direct trauma, and callus from a fracture of the medial epicondyle. MRI can be used to identify these abnormalities and to assess the ulnar nerve itself. When compressed, the nerve may become enlarged and edematous. If conservative treatment fails, the nerve can be transposed anteriorly, deep to the flexor muscle group, or more superficially, in the subcutaneous tissue. One can follow these patients with MRI postoperatively if they become symptomatic to determine whether symptoms are secondary to scarring or infection around the area of nerve transposition.

Compression of the median nerve may be seen with osseous or muscular variants and anomalies, soft-tissue masses and dynamic forces. In the pronator syndrome, compression occurs as the median nerve passes between the two heads of the pronator teres and under the fibrous arch of the flexor digitorum profundus.

The radial nerve can become entrapped following direct trauma, mechanical compression by a cast or overlying space-occupying mass, or a dynamic compression as a result of repeated pronation, forearm extension, and wrist flexion, as is seen in violinists and swimmers. Motor neuropathy of the hand extensors is a dominant feature when the posterior interosseous nerve is entrapped [21].

### References

- Rosenberg ZS, Beltran J, Cheung YY (1994) Pseudodefect of the capitellum: potential MR imaging pitfall. Radiology 191(3):821-823
- Rosenberg ZS, Beltran J, Cheung Y, Broker M (1995) MR imaging of the elbow: normal variant and potential diagnostic pitfalls of the trochlear groove and cubital tunnel. Am J Roentgenol164(2):415-418
- Pincivero DM, Heinrichs K, Perrin DH (1994) Medial elbow stability. Clinical implications. Sports Med 18(2):141-148
- Bradley JP, Petrie RS (2001) Osteochondritis dissecans of the humeral capitellum. Diagnosis and treatment. Clin Sports Med 20(3):565-590
- Patel N, Weiner SD (2002) Osteochondritis dissecans involving the trochlea: report of two patients (three elbows) and review of the literature. J Pediatr Orthop 22(1):48-51
- Steinbach LS, Palmer WE, Schweitzer ME (2002) Special focus session. MR arthrography. Radiographics 22(5):1223-1246
- 7. Carrino JA, Smith DK, Schweitzer ME (1998) MR arthrogra-

phy of the elbow and wrist. Semin Musculoskelet Radiol 2(4):397-414

- Phillips CS, Segalman KA (2002) Diagnosis and treatment of post-traumatic medial and lateral elbow ligament incompetence. Hand Clin 18(1):149-159
- Mulligan SA, Schwartz ML, Broussard MF, Andrews JR (2000) Heterotopic calcification and tears of the ulnar collateral ligament: radiographic and MR imaging findings. Am J Roentgenol 175(4):1099-1102
- O'Driscoll SW (2000) Classification and evaluation of recurrent instability of the elbow. Clin Orthop 370:34-43
- Potter HG, Weiland AJ, Schatz JA, Paletta GA, Hotchkiss RN (1997) Posterolateral rotatory instability of the elbow: usefulness of MR imaging in diagnosis. Radiology 204(1):185-189
- Dunning CE, Zarzour ZD, Patterson SD, Johnson JA, King GJ (2001) Ligamentous stabilizers against posterolateral rotatory instability of the elbow. J Bone Joint Surg Am 83-A(12):1823-1828
- Bredella MA, Tirman PF, Fritz RC, Feller JF, Wischer TK, Genant HK (1999) MR imaging findings of lateral ulnar collateral ligament abnormalities in patients with lateral epicondylitis. Am J Roentgenol 173(5):1379-1382
- Frostick SP, Mohammad M, Ritchie DA. Sport injuries of the elbow. Br J Sports Med 199933(5):301-311
- Field LD, Savoie FH (1998) Common elbow injuries in sport. Sports Med 26(3):193-205
- Nirschl RP, Pettrone FA (1979) Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg Am 61(6A):832-839
- Regan W, Wold LE, Coonrad R, Morrey BF (1992) Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med 20(6):746-749
- Nirschl RP (1992) Elbow tendinosis/tennis elbow. Clin Sports Med 11(4):851-870
- 19. Rettig AC (2002) Traumatic elbow injuries in the athlete. Orthop Clin North Am 33(3):509-522
- 20. Kim YS, Yeh LR, Trudell D, Resnick D (1998) MR imaging of the major nerves about the elbow: Cadaveric study examining the effect of flexion and extension of the elbow and pronation and supination of the forearm. Skeletal Radiol 27:419-426
- Yanagisawa H, Okada K, Sashi R (2001) Posterior interosseous nerve palsy caused by synovial chondromatosis of the elbow joint. Clin Radiol 6(6):510-514

### **Radiology of Hand and Wrist Injuries**

A.J. Wilson

University of Washington, Harborview Medical Center, WA, USA

### Introduction

Musculoskeletal trauma is common and the distal upper extremity is one of the most frequent sites of injury. Imaging of hand and wrist injuries should always begin with conventional radiographs. While computed tomography (CT) and magnetic resonance imaging (MRI) are very helpful in some cases, their overall impact on trauma imaging in the hand and wrist is small. Radiographs remain the primary diagnostic modality. It is therefore essential for radiologists who work in a trauma and emergency setting to be familiar not only with the normal radiographic anatomy of the hand and wrist but also with the range of injuries that can occur. Our learned colleague, Lee F. Rogers, put it all quite simply in a few statements that can be called "Rogers' Rules": Rule #1, make the diagnosis; Rule #2, avoid embarrassment; Rule #3, stay out of court. In order to meet these objectives, we must get adequate radiographs and we must interpret them correctly. Thus, not only should we know where to look when there is nothing obvious at first glance but we must also know where *else* to look when there are obvious findings.

### **Normal Anatomy**

Before considering injury patterns and mechanisms, it essential to have a working knowledge of the normal radiographic anatomy. The standard trauma series for the hand includes three views, which should cover the anatomy from the radiocarpal joint to the finger tips. These views are a pronated frontal view (PA), a pronated oblique view and a lateral view. For wrist injuries, these same three projections are used but are centered and collimated to cover the wrist area, from the metadiaphyses of the distal radius and ulna to the proximal metacarpal diaphyses. A fourth view, the so-called scaphoid view, should always be included in the wrist trauma series. This is a PA view, more tightly collimated than the other three, that is centered on the scaphoid, with the wrist in maximum ulnar deviation. This view rotates the scaphoid about its short axis, presenting the waist of the bone in profile.

When evaluating radiographs of the wrist, several anatomic points are important to observe. First, look at the soft tissues. On the lateral view, convexity of the dorsal soft-tissue margin represents soft-tissue swelling around the carpus and distal radius. It is often a sign of subtle underlying bone or joint injury. Also on the lateral view is the pronator fat pad, which lies parallel to the palmar cortex of the distal radius in most normal individuals. When the distal radius is fractured, the pronator fat pad will be deformed and displaced, becoming convex in a palmar direction. A second but less frequently present fat pad is the scaphoid fat pad. When present, it should be relatively straight and lateral and parallel to the scaphoid bone. If the scaphoid fat pad is convex laterally, a scaphoid fracture should be suspected.

There are several lines and angles that can be drawn in and around the carpus that are helpful in detecting injuries which may otherwise be overlooked. On the PA view, the three carpal arcs (of Gilula) are smooth curves that will be disrupted in injuries to the intercarpal joints. Arc I is drawn across the proximal surfaces of the proximal carpal row. Arc II is drawn across the distal surfaces of the proximal carpal row. Arc III is drawn across the proximal surfaces of the distal carpal row (Fig. 1). The long axis of the capitate, drawn on the PA view, should bisect the third metacarpal shaft regardless of the degree of ulnar or radial deviation (Fig. 1).

The second through fifth carpometacarpal joints should be seen in profile on a good-quality PA view, forming a "lazy M" shape on the radiograph (Fig. 1). While it may not always be possible to see the entire lazy M, most of it should be visible if the wrist is positioned correctly. The key to the carpometacarpal joints is to look at those joint surfaces that have been profiled by the Xray beam. If one side of a joint (carpal or metacarpal) is seen in profile, the other side of that same joint should be seen in profile and parallel to its mate. When only one side is profiled or the articular surfaces are overlapping or not parallel, the joint is either subluxed or dislocated.

On the lateral view, the distal radial articular surface and proximal lunate articular surface should form parallel curves. Similarly, the distal lunate and proximal capi-



Fig. 1. The arcs of Gilula, lazy M and capitate axis

tate should form parallel curves (Fig. 2). If one or more of these articulations are not parallel, the carpus has been dislocated or subluxed. By determining the long axes of the scaphoid, lunate and capitate on the lateral view and measuring the angles between them, the presence of various carpal instabilities and/or ligament injuries can be predicted. The normal scapholunate angle lies between 30 and 60°. The normal capitolunate angle is  $\pm 30^{\circ}$  (Fig. 3).



**Fig. 2.** The radial, lunate and capitate articulations

**Fig. 3.** The scapholunate and capitolunate angles

An increase in the scapholunate angle indicates a dorsal intercalated segment instability (DISI). A decrease in the scapholunate angle indicates a palmar intercalated segment instability (PISI). In both DISI and PISI, the capitolunate angle will usually be increased.

The articular cartilage has approximately the same thickness throughout the carpus. If the apparent space between any two carpal bones appears wider than the apparent space between the others, a ligament disruption has probably occurred. The joints most commonly affected by ligament injuries are the scapholunate and lunotriquetral joints. Therefore, the apparent space between the lunate and scaphoid and the lunate and triquetrum should always be carefully evaluated.

### **Injury Patterns and Mechanisms**

The majority of upper-extremity injuries are the result of a fall onto the out-stretched hand (FOOSH). Many of these FOOSH injuries are concentrated around the wrist and some involve the hand. Those around the wrist are somewhat agedependent. In very small children, whose bones are relatively soft, buckle or torus fractures of the distal radius are the most common injuries. While most of these are obvious, the findings may be limited to very subtle angulation of the cortex, seen only on the lateral view. These injuries are often associated with similar fractures of the distal ulna.

As adolescents enter the growth spurt associated with puberty, their physes become weaker and subject to fracture. The commonest FOOSH injuries in this age group are physeal fractures of the distal radius, which may or may not be associated with ulnar fractures, particularly of the styloid process. These physeal fractures are described in the Salter-Harris classification as follows: type 1, physeal shear injury; type 2, physeal shear with marginal metaphyseal fracture; type 3, physeal shear with epiphyseal fracture; type 4, epiphyseal, physeal and metaphyseal fractures; type 5, physeal crush injury. In general, these injuries are displaced and easy to recognize, with exception of type 5 injuries. However, in some patients, partial auto-reduction may make a type 1 or 2 fracture difficult to find on the radiographs. Secondary signs, such as displacement the pronator fat pad, may be helpful.

In young adults, the bones are at their strongest. This puts the ligaments at increased risk. The center of most frequent injury moves to the carpus, where fractures and dislocations are most likely to occur in the so-called zone of vulnerability (Fig. 4). This zone runs in a curved manner across the radial styloid, scaphoid, capitate, triquetrum and ulnar styloid. The commonest injury within the zone of vulnerability is a scaphoid fracture. The second commonest is an avulsion fracture of the dorsal triquetrum. Next in frequency are various dislocations and fracture dislocations, involving predominantly the midcarpal joint. Scaphoid fractures are important to consider in all injured wrists for two reasons. First, they have a high incidence of nonunion and ischemic necrosis. Second, they tend to be truly nondisplaced and may be difficult to see on radi-



Fig. 4. The zone of vulnerability

ographs taken on the day of injury. Follow up radiographs, after 2 weeks, will often show these occult fractures. If prompt diagnosis is needed, MRI is much more sensitive in revealing nondisplaced fractures than radiography.

In older adults, as osteoporosis sets in and the bones become weaker, the distal radius once again becomes the commonest site for FOOSH injuries. The most common variety of distal radial fracture is one in which the distal fracture fragment is displaced and angulated in a dorsal direction. This fracture was first described by Abraham Colles, in 1814, and now bears his name. Since Colles described this fracture 81 years before the discovery of X-rays, he did not know the detail or radiographic manifestations of this injury. His real contribution was to point out that these are fractures, not dislocations. He showed that they could be reduced and splinted and could heal with excellent results. When the deformity is in the opposite direction (palmar) we refer to the injury as a Smith's fracture. When there is no deformity, the injury should be described simply as a nondisplaced, distal, radial fracture. Fractures of the ulnar styloid commonly occur in association with distal radial fractures but are not always present. Their presence does not change the designation as a Colles', Smith's or nondisplaced fracture. One of the most important findings to observe in these fractures is extension into the distal radial articular surface. Intra-articular fractures often require surgical repair and should be further evaluated with CT.

When fractures of the distal radius are associated with radiocarpal dislocations, they are referred to as "Barton's fractures". If the dorsal lip is fractured, the carpus will be displaced dorsally. This is referred to as a "dorsal Barton's fracture". Conversely, if the palmar lip of the radius is fractured, the carpus will be displaced palmarly. This is referred to as a "palmar Barton's fracture". While pure dislocations of the radiocarpal joint can occur without radial lip fractures, they are much less frequent than Barton's fracture-dislocations.

### **Carpal dislocations**

Most carpal dislocations involve the midcarpal joint, which is between the proximal and distal carpal rows. On the lateral view, these injuries show disruption of the normal relationship between lunate and capitate, usually with dorsal displacement of the capitate. The distal articular surface of the lunate is "empty". On the PA projection, the lunate takes on a triangular shape as it rotates about its horizontal axis. Arcs I and II are disrupted, while arc III is normally intact. These dislocations usually occur around the lunate and are therefore called "perilunate" dislocations. The majority of perilunate dislocations are associated with fractures through the scaphoid waist but any fracture within the zone of vulnerability is possible. Perilunate dislocation without an associated fracture is not uncommon. The description of the injury includes the fractures and the words "perilunate dislocation". For example: a trans-radial, trans-scaphoid, trans-capitate, perilunate dislocation would be one of these dislocations with fractures through the radial styloid, scaphoid waist and capitate neck. Ulnar styloid fractures are frequently present but are usually not included in the descriptive classification. When the lunate is displaced from the radial articular surface in a midcarpal joint disruption, it is called a "lunate dislocation". "Midcarpal dislocation" is the term used to describe the intermediate position, when the capitate is dislocated from the lunate and the lunate is subluxed from the radius. This term is confusing, since all of these patterns are dislocations of the midcarpal joint.

Other, less-common, carpal dislocations include the longitudinal variety. These are the result of high-energy trauma and separate the carpus into medial and lateral portions. They are usually obvious radiographically and frequently require surgical repair.

### **Carpometacarpal dislocations**

Perhaps the most commonly missed serious injury to the hand and wrist is dislocation along the carpometacarpal joint. These injuries can be surprisingly subtle on initial radiographs. In spite of this, they are serious injuries that usually require surgical repair. There are two keys to finding them:. (1) they are frequently associated with avulsion fractures of the distal carpals or proximal metacarpals; (2) on at least one of the standard views, the affected carpometacarpal joints will show loss of parallelism. On the lateral radiograph, dorsal displacement of the metacarpal bases may be apparent. So, the important point to remember is: any time a fracture at the carpometacarpal junction is seen, a dislocation must be assumed, until proven otherwise.

CT or fluoroscopy may be required to resolve this issue.

### **Metacarpal Injuries**

While metacarpal fractures may occur in FOOSH, they are more frequent when the fist is closed. In other words, they are most commonly associated with punching, usually during a fist fight. A well placed punch will line up the second metacarpal with the radius, often resulting in a fracture of the second metacarpal neck. However, most barefisted fighters have not been trained to punch correctly and strike glancing blows with the ulnar aspect of the fist. These blows frequently result in fractures of the fifth metacarpal neck. This has been called the "boxers fracture" but would be more accurately defined as the "amateur street-fighter's fracture". The head of the metacarpal is typically displaced and angulated in a palmar direction. If the fracture is allowed to heal in this position, the next time the individual participates in a fist fight, a fracture of the fourth metacarpal neck is likely, as the fifth is now depressed and allows the fourth to receive the maximum force of the punch. In indirect trauma from FOOSH or other mechanisms, twisting injuries to the metacarpal may occur, resulting in spiral, diaphyseal, fractures.

### **Finger Injuries**

Finger fractures can occur from FOOSH but are more commonly the result of direct trauma to the fingers. As in the metacarpals, twisting injuries will result in spiral, diaphyseal, phalangeal, fractures. Direct dorsal blows to the finger tip, such as hitting with a hammer, result in burst fractures of the terminal tuft. These are typically comminuted but minimally displaced. Injuries in which the finger is bent backward may result in dislocation of the interphalangeal joint or avulsion of the volar plate. The volar plate is a fibrocartilaginous structure at the insertion of the short flexor tendon, at the palmar base of the middle phalanx. When the finger is acutely bent backwards, this plate may be avulsed and often takes a small fragment of bone with it. These injuries can be subtle and may be visible only on the lateral view. When the finger is stuck directly on its tip, as in a failed attempt to catch a hard ball, the tip of the finger is forced palmarly against tensed flexor and extensor tendons. This results in avulsion of the extensor tendon insertion, at the dorsal base of the distal phalanx, sometimes with a small avulsed fragment of bone. Detachment of the extensor tendon produces a characteristic finger deformity in which there is persistent slight flexion of the distal interphalangeal joint. This deformity has been variously described as "mallet finger" or "baseball finger". It is readily diagnosed, both clinically and on the lateral radiograph, with or without an avulsion fracture.

### **Penetrating injuries**

Penetrating injuries to the hand and wrist result from stab wounds, gunshot injuries and explosions with the grasp. The latter are most commonly seen around times of celebration with fireworks. In the United States, these injuries most frequently occur around July Fourth and New Year's Eve. Penetrating injuries are very variable, depending on the location and force of penetration. They are often devastating, resulting in multiple fractures, severe soft-tissue loss and a hand beyond repair. The radiologist's job is simple: describe what is broken and what is missing. Penetrating trauma rarely presents the same challenges as blunt trauma.

### **Advanced Imaging**

As stated earlier, CT and MRI have a limited role in diagnosing hand and wrist trauma. However, in certain situations, they can prove invaluable.

CT often provides the best method for characterizing complex injuries. It is far more reliable than radiography for the assessment of fracture healing. CT is the most reliable method for evaluating alignment of the distal radioulnar joints in suspected instability, dislocation or subluxation. In pre-operative planning, CT gives the most reliable assessment of comminution, displacement or involvement of articular surfaces. It is also helpful in calculating the volume of bone graft that is needed for surgical repair.

MRI remains the most sensitive and accurate method for excluding occult fractures. With radiography, 2 weeks of immobilization may be required before an occult fracture can be reliably excluded. By contrast, with MRI, a definitive decision can usually be made on the day of injury. In professional athletes and others, whose occupations do not lend themselves to prolonged or unnecessary immobilization, such prompt diagnosis is important.

### **Suggested Reading**

- Fisher MR, Rogers LF, Hendrix RW (1983) Systematic approach to identifying fourth and fifth carpometacarpal dislocations. AJR 140:319
- Gilula LA (1990) The traumatized hand and wrist. WB Saunders, Philadelphia, pp 94-97
- Gilula LA (1990) The traumatized hand and wrist. WB Saunders, Philadelphia, pp 287-314
- Gilula LA, Yin YM (1996) Imaging of the wrist and hand. WB Saunders, Philadelphia, pp 43-224
- Gilula LA, Yin YM (1996) Imaging of the wrist and hand. WB Saunders, Philadelphia, pp 311-318
- Hill N (1970) Fractures and dislocations of the carpus. Orthop Clin North 1:275
- Rawles JG (1988) Dislocations and fracture at the carpometacarpal joints of the fingers. Hand Clin 4:103
- Rogers LF (2001) Radiology of skeletal trauma, 3rd Edition. Churchill Livingstone, Philadelphia, pp 813-855
- Rogers LF (2001) Radiology of skeletal trauma, 3rd Edition. Churchill Livingstone, Philadelphia, pp 904-929
- Wagner CJ (1959) Fracture-dislocations of the wrist. Clin Orthop 15:181

### Wrist and Hand

### L.A. Gilula

Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, MO, USA

### Introduction

This chapter will emphasize general principles when assessing a variety of lesions of the hand and wrist. An approach to analyzing the wrist and hand bones will be provided, followed by a discussion of applications of these principles with respect to trauma, infection, neoplasia, arthritis, and metabolic bone disease. Obviously it is impossible to cover all of musculoskeletal imaging and pathology in a short article; however, some major points will be emphasized in each of these different areas, with the most emphasis placed on complex carpal trauma.

### **Overview of Analysis**

As described by D. Forrester [1], looking at the musculoskeletal system anywhere can be evaluated by the "A, B, C, D, 'S" system. "A" stands for alignment, "B" for bone mineralization, "C" for cortex, cartilage and joint space abnormalities, "D" for distribution of abnormalities, and "S" for soft tissues. Utilizing these principles will help keep one from missing major observations. Starting with "S" for soft tissues will keep one from forgetting to evaluate soft tissues. Recognizing soft-tissue ("S") abnormalities will point to an area of major abnormality and should trigger a second or third look at the center of the area of soft-tissue swelling to see whether there is an underlying abnormality. The soft tissues dorsally over the carpal bones are normally concave. When the soft tissues over the dorsum of the wrist are straight or convex, swelling should be suspected. The pronator fat line volar to the distal radius suggests deep swelling when it is convex outward, as normally it should be straight or concave [2]. Soft-tissue swelling along the radial and ulnar styloids may be seen in synovitis or trauma. Swelling along the radial or ulnar side of a finger joint can indicate collateral ligament injury. Exceptions to this statement exist along the radial side of the index finger and the ulnar side of the small finger. Focal swelling circumferentially around one interphalangeal or metacarpophalangeal joint is highly suggestive of capsular or joint

swelling. Another cause for diffuse swelling along one side of the wrist or finger can be tenosynovitis.

The evaluation of alignment ("A") allows deviations from normal to be recognized. Angular deformities are commonly seen in arthritis. Dislocations and carpal instabilities manifest as abnormalities in alignment.

In evaluating bone mineralization ("B"), different patterns are evident. Acute bone demineralization presents as subcortical bone loss in the metaphyseal areas and at the ends of bones, in regions of increased vascularity of bones. A typical example is the young person who has an injured part of the body placed in a cast with subsequent development of rapid demineralization. Diffuse even demineralization commonly develops over longer periods of time and may be seen in older people with diffuse osteopenia of age and also from prolonged disuse. Focal osteopenia, especially associated with cortical loss, should raise the question of infection or a more acute inflammatory process in that area of local bone demineralization.

"C" reminds us to look at all the joint spaces as well as the margins of these joints and bones for cartilage space narrowing, erosions, and other cortical abnormalities.

"D" refers to the distribution of abnormalities. It is most vividly exemplified by the distribution of erosions, as may be seen distally in psoriasis and more proximally in rheumatoid arthritis.

Three major concepts relate to alignment: (1) parallelism, (2) overlapping articular surfaces, and (3) three carpal arcs [3-5]. All three can be especially applied to the carpal bones., while the first two can be applied throughout the body. Parallelism refers to the fact that any anatomic structure that normally articulates with an adjacent anatomic structure should show parallelism between the articular cortices of those adjacent bones. This is exactly how jigsaw puzzles work. If there is a piece of a jigsaw puzzle out of place, then that piece loses its parallelism to adjacent pieces. Anatomically, this would cause overlapping articular surfaces. Therefore, the concepts of parallelism and overlapping articular surfaces are related. If there is overlap of normally articulating surfaces, there should be dislocation or subluxation at the site of those overlapping surfaces. This does not apply if one bone is foreshortened or bent, as with overlapping phalanges on a PA view of a flexed finger. In that situation, one phalanx would overlap the adjacent phalanx, but in the flexed PA position one would not normally see parallel articular surfaces at that joint.

The third alignment concept refers to the fact that three carpal arcs can be drawn in any normal wrist when the wrist and hand are in a neutral position, i.e., the third metacarpal and the radius are coaxial. Arc I is a smooth curve along the proximal convex surfaces of the scaphoid, lunate and triquetrum. Arc II is a smooth arc drawn along the distal concave surfaces of these same three carpal bones. Arc III is a smooth arc that is drawn along the proximal convex surfaces of the capitate and hamate [3, 6]. When one of these arcs is broken at a joint, then something is probably wrong with that joint, as ligament disruption; or when broken at a bone surface, a fracture. Two normal exceptions to the descriptions of these arcs exist. In arc I, the proximal distal dimension of the triquetrum may be shorter than the apposing portion of the lunate. A broken arc I at the lunotriquetral joint is a congenital variation when this situation arises. Another congenital variation exists where there is a prominent articular surface of the lunate that articulates with the hamate, a type II lunate. (A type I lunate is the lunate with one distal smooth concave surface; in a type II lunate there is one concave articular surface that articulates with the capitate and a second concavity, the hamate facet of the lunate, which articulates with the proximal pole of the hamate). In a type II lunate, arc II may be broken at the distal surface of the lunate, where there is a normal concavity at the lunate hamate joint. Similarly, there can be a slight jog of arc III at the joint between the capitate and hamate in this type of wrist; however, the overall outer curvatures of the capitate and hamate are still smooth. At the proximal margins of the scapholunate and lunotriquetral joints, these joints may be wider due to curvature of these bones. Observe the outer curvature of these bones when analyzing the carpal arcs. Also, to analyze the scapholunate joint space width, look at the middle of the joint between parallel surfaces of the scaphoid and lunate to see whether there is any scapholunate space widening compared to a normal capitolunate joint width in that same wrist.

The hand and wrist can be analyzed very promptly after first surveying the soft tissues by looking at the overall alignment, bone mineralization and cortical detail as one looks at the radiocarpal joints, the intercarpal joints of the proximal carpal row, midcarpal joint, intercarpal joints of the distal carpal row, carpometacarpal joints, and interphalangeal joints. Analyzing these surfaces and bones evaluated on all views leads to a diagnosis. It is preferable to carefully analyze the PA view of the wrist first as this view will provide the most information. The lateral and oblique views are merely used for confirmation and clarification of what is actually present on the PA view. An exception to this comment is the need to closely evaluate the soft tissues on the lateral as well as the PA view. The following sections will discuss applying these principles to more specific abnormalities.

### Trauma

Traumatic conditions of the wrist basically can be classified as fractures, fracture-dislocations, and soft-tissue abnormalities, which include ligament instabilities. Analysis of the carpal arcs, overlapping articular surfaces, and parallelism will help determine what exact traumatic abnormality is present. Recognizing which bones normally parallel each other also identifies which bones have moved together as a unit away from a bone that has overlapping adjacent surfaces. A majority of the fractures and dislocations about the wrist are of the perilunate type, in which there is a dislocation with or without adjacent fractures taking place around the lunate. The additional bones that may be fractured are named first with the type of dislocation mentioned last. For the perilunate type of dislocations, whatever bone centers over the radius (the capitate or lunate) is considered to be "in place". Therefore, if the lunate is centered over the radius, this would be a perilunate type of dislocation. If the capitate is centered over the radius and the lunate is not, this would be a lunate dislocation. Therefore, if there were fractures of the scaphoid and capitate, dorsal displacement of the carpus with respect to the lunate, and the lunate was still articulating or centered over the radius, this would be called a transscaphoid transcapitate dorsal perilunate dislocation. Another group of fracture-dislocations that occur in the wrist are the axial fracture-dislocations, in which a severe crush injury may split the wrist along an axis around a carpal bone other than the lunate. such as perihamate or peritrapezial axial dislocation, usually with fractures [7].

### Ligamentous Instability

There are many types of ligament instabilities, including very subtle types; however, there are five major types of ligament instabilities that can be recognized readily based on plain radiographs. These refer to the lunate as being an "intercalated segment" between the distal carpal row and the radius, similar to the middle or intercalated segment between two links in a three-link chain. Normally there can be a small amount of angulation between the capitate, lunate, and the radius on the lateral view. However with increasing lunate angulation, especially as seen on the lateral view, an instability pattern may be present. If the lunate tilts too far dorsally, it would be called a dorsal intercalated segmental condition; if the lunate tilts too far volarly, it would be called a volar or palmar intercalated segmental problem. Therefore, if the lunate is tilted too far dorsally (so that the capitolunate angle is more than 30° and/or the scapholunate angle is more than  $60^{\circ}$ - $80^{\circ}$ ), this would be called a dorsal intercalated segmental instability (DISI) pattern. If the lunate is tilted too far volarly or palmarly (a capitolunate angle of more than  $30^{\circ}$  or scapholunate angle of less than  $30^{\circ}$ ), this would be a volar intercalated segmental instability (VISI) or palmar intercalated segmental instability (PISI) pattern. When there is a "pattern" of instability, a true instability can be further evaluated with a dynamic wrist instability series performed under fluoroscopic control [8,9]. When there is abnormal intercarpal motion and abnormal alignment, this supports the radiographic diagnosis of carpal instability. By comparison with the opposite wrist, the questionable wrist can be evaluated for instability with lateral flexion, extension, and neutral views, PA and AP views with radial, neutral, and ulnar deviation views. Fist-compression views in the supine position may help widen the scapholunate joint in some patients. Ulnar carpal translation is a third type of carpal instability [8]. If the entire carpus moves too far ulnarly, as recognized by more than one-half of the lunate positioned ulnar to the radius when the wrist and hand are in neutral position, this would be an ulnar carpal translation type I. If the scaphoid is in the normal position relative to the radial styloid, but there is scapholunate dissociation and the remainder of the carpus moves too far ulnarly, as mentioned for ulnar carpal translation type I, this is called ulnar carpal translation type II. The fourth and fifth types of carpal instabilities relate to the carpus displacing dorsally and volarly off the radius. If the carpus, as identified by the lunate, has lost its normal articulation with the radius in the lateral view and is displaced dorsally off the radius, this is called dorsal radiocarpal instability, or dorsal carpal subluxation. It occurs most commonly following a severe dorsally impacted distal radius fracture. If the carpus is displaced palmarly off the carpus, as identified between the lunate and its articulation with the radius, and the remainder of the carpus has moved with the lunate, this would be called a palmar carpal subluxation. There are other types of carpal instability patterns that are better detected more by physical examination; these will not be covered here.

### Infection

Infection should be suspected when there is an area of cortical destruction with pronounced osteopenia. It is not uncommon to have patients present with pain and swelling, and clinically infection may not be suspected when it is chronic, as with an indolent type of infection such as tuberculosis. Soft-tissue swelling is a key point for this diagnosis as for other abnormalities of the wrist, as mentioned above. Therefore, the diagnosis of infection is most likely when there is swelling and associated osteopenia as well as cortical destruction ,or even early focal joint-space loss without cortical destruction.

### Neoplasia

When there is an area of abnormality, it helps to determine the gross area of involvement, then look at the center of the abnormality [10]. If the center of the abnormality is in

bone, then probably the lesion originated within the bone. When the center of abnormality is in soft tissues, a lesion originating in soft tissues should be suspected. When there is a focal area of bone loss or destruction or even a focal area of soft-tissue swelling with or without osteopenia. neoplasia is a major consideration. Whenever neoplasia is a concern on an imaging study, infection should also be considered. To analyze a lesion within a bone, look at the margins of the lesion to see whether it is well-defined and whether it has a thin to thick sclerotic rim. Evaluate the endosteal surface of the bone to see whether there is scalloping or concavities along the endosteal surface of the bone. Concavities representing endosteal scalloping are characteristic of cartilage tissue. This would be typical for an enchondroma, which is the most common intraosseous bone lesion of the hands. The matrix of the lesion should also be evaluated to see whether there are dots of calcium that can be seen in cartilage, or whether there is a more diffuse type of bone formation as occurs in an osseous type of tumor as from osteosarcoma. As elsewhere in the body, if a lesion is very well-defined and if there is bone enlargement, these are indicative of an indolent or a less aggressive type of lesion. The presence of cortical destruction supports the finding of an aggressive lesion, such as malignancy or infection. To determine the extent of a lesion, magnetic resonance (MR) is the preferred method of imaging. Bone scintigraphy can be very valuable to survey for osseous lesions throughout the body, as many neoplastic conditions spread to other bones or even to the lung.

When there is a lesion is in the soft tissue of the hand, especially with pressure effect on an adjacent bone, a giant cell tumor of the tendon sheath should be suspected. Ganglion is another cause for a focal swelling in the hand, but usually that occurs without underlying bone deformity. Glomus tumor is a less common, painful soft-tissue lesion that may be detected with ultrasound or MR imaging. Occasionally, a glomus tumor will cause a pressure effect on bone, especially on the distal phalanx under the nail bed.

### Arthritis

Using the above scheme of analyzing the hand, wrist, and musculoskeletal system [11], swelling can indicate capsular involvement as well as synovitis. The overall evaluation of alignment shows deviation of the fingers at the interphalangeal and metacarpophalangeal joints in addition to subluxation or dislocation at the interphalangeal, metacarpophalangeal, or intercarpal or radiocarpal joints. Joint-space loss, the sites of erosions, and the sites of bone production are important to recognize. When identifying the abnormalities, the metacarpophalangeal joint capsules, especially of the index, long and small fingers, should be examined carefully to determine whether they are convex, as occurs in for capsular swelling. This can help in establishing whether this is primarily a synovial arthritis, which in some cases exists in combination with osteoarthritis. Synovial arthritis is supported by findings of bony destruction from erosive disease. The most common entities to consider for synovial-based arthritis are rheumatoid arthritis, and then psoriasis. If there is osteophyte production, osteoarthritis is the most common consideration, whereas osteoarthritis associated with erosive disease, especially in the distal interphalangeal joints, is supportive of erosive osteoarthritis. Punched-out or welldefined lucent lesions of bone, especially about the carpometacarpal joints in well-mineralized bones, must also be considered for the robust type of rheumatoid arthritis. For deposition types of disease, gout is a classic example. Gout is usually associated with normal bone mineralization and "punched-out" lesions of bone. Gouty destruction depends somewhat on where the gouty tophi are deposited, whether they are intraosseous, subperiosteal, adjacent to and outside of the periosteum or intraarticular.

### **Metabolic Bone Disease**

A classic condition of metabolic bone disease in the hands is that seen with renal osteodystrophy. Metabolic bone disease is considered when there are multiple sites of bone abnormality throughout the body with or without diffuse osteopenia. However, some manifestations of metabolic bone disease may start first or be more manifest in the hands, in the feet or elsewhere in the body. There is a strong likelihood of renal osteodystrophy when there is subperiosteal resorption, typically along the radial aspect of the bases of the proximal or middle phalanges, but there also may be cortical loss along the tufts of the distal phalanges. Bone resorption can also take place intracortically and endosteally. Again, analysis of the bones involved and of the associated abnormalities present can help lead to the most likely diagnosis.

### Conclusions

Application of the the "A, B, C, D'S" system, together with an analysis of parallelism, abnormal overlapping articular surfaces and carpal arcs, can help analyze abnormalities encountered in the hand and wrist, which can help in making a most reasonable diagnosis for further evaluation of the patient.

### References

- 1. Forrester DM, Nesson JW (1973) The ABC'S of Arthritis (Introduction) In: Forrester DM, Nesson JW (eds) The radiology of joint disease. Philadelphia WB Saunders, Philadelphia, Pennsylvania, pp 3
- Curtis DJ, Downey EF Jr (1992) Soft tissue evaluation in trauma. In: Gilula LA (ed) The traumatized hand and wrist. Radiographic and anatomy correlation. WB Saunders, Philadelphia, Pennsylvania, pp 45-63
- Gilula LA (1979) Carpal injuries: analytic approach and case exercise. Am J Roentgenol AJR 133:503-517
- Yin Y, Mann FA, Gilula LA, Hodge JC (1996) Roentgenographic approach to complex bone abnormalities. In: Gilula LA. Yin Y (eds) Imaging of the wrist and hand. WB Saunders, Philadelphia, Pennsylvania, pp 293-318
- 5. Gilula LA, Totty WG (1992) Wrist trauma: roentgenographic analysis. In: Gilula LA (ed) The traumatized hand and wrist. Radiographic and anatomy correlation. WB Saunders, Philadelphia, Pennsylvania, pp 221-239
- Peh WCG, Gilula LA (1996) Normal disruption of carpal arcs. J Hand Surg (Am) 21:561-566
- Garcia-Elias M, Dobyns JH, Cooney WP, Linscheid RL (1989) Traumatic axial dislocations of the carpus. J Hand Surg 14A:446-457
- Gilula LA, Weeks PM (1978) Post-traumatic ligamentous instabilities of the wrist. Radiology 129:641-651
- Truong NP, Mann FA, Gilula LA, Kang SW (1994) Wrist instability series: Increased yield with clinical-radiologic screening criteria. Radiology 192:481-484
- Peh WCG, Gilula LA (1995) Plain film approach to tumors and tumor-like conditions of bone. Br J Hosp Med 54:549-557
- Forrester DM, Nesson JW (eds) (1973) The radiology of joint disease. WB Saunders, Philadelphia, Pennsylvania

### Imaging of the Painful Hip and Pelvis

C.W.A. Pfirrmann, C.A. Petersilge

Department of Radiology, Orthopedic University Hospital Balgrist, Zurich, Switzerland

Many exciting new advances in our knowledge of the hip and its pathologic processes have occurred during the past several years. With the use of magnetic resonance (MR), MR arthrography and with improvements in arthroscopy and surgery of the hip we continue to improve our understanding of the hip. Current topics of interest include imaging of the acetabular labrum, femoroacetabular impingement, fatigue and insufficiency fractures, bone-marrow edema syndromes, and abnormalities of the greater trochanter and its tendon.

### Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is a conflict occurring between the proximal femur and the acetabular rim. FAI is caused either by abnormalities of the proximal femur or the acetabulum. Often a combination of factors may lead to FAI. The repetitive mechanical conflict occurring in flexion and internal rotation will lead to lesions of acetabular labrum and the adjacent acetabular cartilage. The exact mechanism responsible for osteoarthritis (OA) of the hip has long been debated. There is emerging evidence that FAI may be an important etiologic factor for the development of early OA of the hip [1].

### **Types of FAI**

Two distinct types of FAI can be distinguished: "cam" and "pincer" impingement.

Cam impingement is caused by jamming of an abnormal junction of the femoral head and neck (usually a deficiency of the femoral waist at the anterolateral portion of the femoral neck) into the acetabulum during forceful flexion and internal rotation of the hip. This results in abrasion of the acetabular cartilage or its avulsion from the labrum and subchondral bone in a rather constant anterosuperior area. Chondral avulsion, in turn, leads to tear or detachment of the principally uninvolved labrum. Cam impingement is common in young and athletic males [2].

Pincer impingement is the result of linear contact between the acetabular rim and the femoral head-neck junction. The femoral waist is usually normal and the abutment is the result of an acetabular abnormality, often a general over-coverage (coxa profunda) or local anterior over-coverage (acetabular retroversion). The first structure to fail with the pincer impingement type is the acetabular labrum. Continued pincer impingement results in degeneration of the labrum and ossification of the rim, leading to additional deepening of the acetabulum and worsening of the over-coverage. Pincer impingement can result in chondral injury in the contrecoup region of the posteroinferior acetabulum. Chondral lesions in pincer impingement often are limited to a small rim area and therefore are more benign. This is in contrast to the deep chondral lesions and chondral avulsions seen with cam impingement. Pincer impingement is seen more frequent in middle-aged women [1].

FAI has been shown to cause labral and chondral lesions and leads to OA of the hip. Surgical treatment includes reshaping of the femoral waist or the acetabular rim and thus eliminating the main pathogenic factor of FAI [3].

Conventional radiographs are the basis for evaluating patients with FAI. Radiographs often appear normal at first. However, on detailed review some abnormalities may become apparent. In cam impingement, a bony prominence, usually at the anterior head and neck junction, is often present and is seen best on cross-table lateral radiographs. Other findings include a reduced waist of the femoral neck and head junction, and changes at the acetabular rim, such as os acetabuli, or herniation pits at the femoral neck (Fig. 1). In pincer impingement, acetabular findings include conditions with a relatively too-large anterior wall of the acetabulum, such as the coxa profunda/protrusio acetabuli (Fig. 2) or the retroversion of the acatebulum (crossing sign between the lateral outlines of the anterior and posterior acetabular wall).



**Fig.1.** Femoroacetabular impingement (FAI), cam impingement. Cross-table lateral radiograph showing a marked bony prominence at the anterior head and neck junction (*curved arrow*). Note the os acetabuli (*arrowhead*)



**Fig. 3.** FAI: cam impingement. Transverse oblique magnetic resonance (MR) image parallel to the femoral neck showing a marked bony prominence at the anterior head and neck junction (*arrow*). Note contrast material (*arrowhead*) in a cartilage defect of the anterior acetabulum



**Fig. 2.** FAI, pincer impingement. AP radiograph of the hip demonstrating a marked protrusio acetabuli. The medial border of the acetabulum (*black arrowheads*) extends medial to the Ilio-ischial line (*arrow*). Note the ossification of the acetabular rim (*white arrowheads*)

*MR arthrography* is the best way to evaluate a hip with suspected FAI syndrome. Transverse oblique (Fig. 3) or radial images maybe helpful for the evaluation of the femoral waist contour. Most patients present with anterosuperior labral tears and degeneration of the labrum associated with cartilage lesions at the anterosuperior acetabulum. Herniation pits and os acetabuli are frequent findings. It has been postulated that the former are indicative of FAI.

The *a*-angle helps to identify and quantify an abnormal contour of the anterior femoral head-neck junction



**Fig. 4.** a-Angle. Transverse oblique MR image parallel to the femoral neck. A circle is drawn along the contour of the femoral head. The angle is measured between the line drawn from the center of the circle of the femoral head to the point where the circle leaves the outline of the anterior contour of the femoral head-neck junction (*curved arrow*) and a line drawn parallel through the center of the femoral neck and the center of the circle of the femoral head

(Fig. 4). Transverse oblique MR images parallel to the femoral neck are obtained and the *a*-angle is measured on the central slice. First, a circle is drawn along the contour of the femoral head. Then two lines are drawn: The first line is drawn from the center of the circle of the femoral head to the point where the circle leaves the anterior contour of the femoral head-neck junction.

The second line is drawn parallel through the center of the femoral neck and the center of the circle of the femoral head. The *a*-angle is then measured between the two lines. An angle over  $55^{\circ}$  indicates a significant abnormal contour of the anterior femoral head-neck junction [4].

### The Greater Trochanter

The hip joint, much like the glenohumeral joint, has one of the widest ranges of motion in the human body. The greater trochanter serves as the main attachment site for very strong tendons, facilitating complex movement such as postural gait. This complex motion is achieved by the sophisticated attachment architecture of the abductor mechanism in the trochanteric surface and its three interposed bursae. The integrity of the greater trochanteric structures is therefore important for normal gait.

The attachments of the abductor tendons about the greater trochanter of the hip can be divided into three groups. The main tendon of the gluteus medius muscle has a strong insertion covering the posterosuperior aspect of the greater trochanter. The lateral part of the gluteus medius tendon insertion is obliquely orientated. It runs from posterior to anterior and inserts at the lateral aspect of the greater trochanter. Parts of the gluteus medius run anteriorly and cover the insertion of the gluteus medius tendon is usually thin and may be almost purely muscular. The main tendon of the gluteus minimus attaches to the anterior part of the trochanter.

Part of the gluteus minimus insertion is muscular and inserts in the ventral and superior capsule of the hip joint [5].

Although pain over the lateral aspect of the hip has been commonly attributed to trochanteric bursitis, the spectrum of pathology about the hip has broadened with the identification of entities such as "rotator cuff tears of the hip", referring to a tear of the gluteus medius (Fig. 5) or minimus tendon [6]. Despite similar clinical presentations, treatment of these processes can be quite different, emphasizing the need for accurate diagnosis. The typical appearance of this tear is a circular or oval defect in the gluteus minimus tendon that extends posteriorly into the lateral part of the gluteus medius tendon. MR imaging is useful for the diagnosis of either tendinosis or tendon tears of the abductors [7].

### Abductor Tendons After Total Hip Arthroplasty

Primary total hip arthroplasty (THA) is the second most common joint-replacement performed in the United States after primary total knee replacement, and over 200,000 procedures are done per year. Reasons for residual pain after total hip replacement include hardware failure, such as mal-alignment or loosening of the prosthesis, and soft-tissue abnormalities, including infection, joint instability, trochanteric bursitis and ectopic bone formation. The imaging workup usually focuses on evaluating hardware failure; however, especially if a transgluteal approach has been used, soft-tissue defects, such as tendon tears



**Fig. 5.** Coronal T1weighterd spin-echo image (*left image*) and T2weighted fat saturated (*right image*) demonstrating a complete tear (*curved arrow*) of the gluteus medius tendon (*arrowheads*)

(Fig. 6), muscle atrophy, and bursitis, are often the underlying reason for trochanteric pain and limping.

Traditionally, MR imaging (MRI) has played a very limited role in the evaluation of patients after THA, primarily because of susceptibility artifacts related to the metallic implants. Modifications of traditional MR sequences can be used to such artifacts. Optimized image quality can be achieved in spin echo imaging by using a high bandwidth (at least 130 Hz/pixel), a high-resolution matrix (512×512), sequences with multiple refocusing pulses, and a frequency-encoding axis parallel to the long axis of the prosthesis.

It is important to recognize that, although more frequent in symptomatic patients, many MR findings, such as altered signal and diameter of the abductor tendons, bursal fluid collections and fatty atrophy of the anterior gluteus minimus muscle, are frequently found in asymptomatic patients after THA through a lateral transgluteal approach. However, defects of the abductor tendons (Fig. 6) and fatty atrophy of the gluteus medius and the posterior part of the gluteus minimus muscle are uncommon in asymptomatic patients after THA and are therefore clinically relevant.

MRI is a valuable diagnostic tool in patients with trochanteric pain or weakness after THA.



**Fig. 6.** Coronal T2-weighted fast spin-echo MR image in a patient after total hip arthroplasty. Note detachment of the gluteus medius tendon (*arrowheads*) with retraction and a large fluid collection (*arrow*)

### Imaging of the Acetabular Labrum

The acetabular labrum is a fibrocartilaginous structure that is firmly attached to the acetabular rim. At the anteroinferior and posteroinferior margins of the joint, the labrum joins with the transverse ligament, which spans the acetabular notch. The labrum is normally of triangular morphology and typically has low signal intensity on all imaging sequences [8]. However, variations in signal intensity and morphology do occur, including rounded and flattened labra as well as absent labra [9-11]. Variations in signal intensity are most common in the superior labrum and may be seen on any imaging sequence [9, 10, 12].

Labral detachments and intrasubstance tears are frequently identified in patients with symptoms of mechanical hip pain without any radiographically identifiable abnormality [13-16]. Labral pathology is also commonly seen in patients with developmental dysplasia and those with femoroacetabular impingement. The use of MR arthrography and joint distention significantly increases the sensitivity and specificity for detection of labral abnormalities. Tears are recognized by the intrasubstance collection of contrast material while detachments are recognized by the presence of contrast at the acetabular labrum interface. These abnormalities are most commonly located at the anterosuperior margin of the joint.

Pitfalls in interpretation include the sulcus at the junction of the labrum and the transverse ligament at the anteroinferior and posteroinferior portions of the joint as well as the presence of a cleft or groove between the articular cartilage and the labrum.

### **Stress and Insufficiency Fractures**

Stress and insufficiency fractures commonly involve the pelvis. Stress fractures are commonly identified in the proximal femur and typically occur along the medial aspect of the femoral neck. Pubic rami stress fractures are one cause of groin pain, and imaging will help to differentiate these injuries from injuries to the anterior abdominal wall musculature and the adductor muscle origins [17, 18].

Insufficiency fractures are also frequently seen in the pelvis. Common sites include the sacrum, pubic rami, and the ileum, including the supra-acetabular ileum. Insufficiency fractures of the subchondral portion of the femoral head have recently been recognized [19-21]. Previously, these lesions were often diagnosed as transient osteoporosis of the hip. On MRI, an ill-defined low-signal-intensity line is visible on T1-weighted images, and is variably visible on T2-weighted images. These lesions may be one underlying cause of rapidly destructive OA of the hip [22].

### **Bone-Marrow Edema Syndrome**

This syndrome is a global term used to describe the MR findings of low signal on T1-weighted images and bright signal on fluid-sensitive sequences that involve the femoral head with varying degrees of extension into the femoral neck. Etiologies include transient osteoporosis of the hip, early avascular necrosis, insufficiency fracture, and infection [23, 24]. The clinical scenario often helps to differentiate these various entities.

### References

- Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA (2003) Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop 112-120
- Ito K, Minka MA, 2nd, Leunig M, Werlen S, Ganz R (2001) Femoroacetabular impingement and the cam-effect. A MRIbased quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br 83:171-176
- Lavigne M, Parvizi J, Beck M, Siebenrock KA, Ganz R, Leunig M (2004) Anterior femoroacetabular impingement: part I. Techniques of joint preserving surgery. Clin Orthop, pp 61-66
- Notzli HP, Wyss TF, Stoecklin CH, Schmid MR, Treiber K, Hodler J (2002) The contour of the femoral head-neck junction as a predictor for the risk of anterior impingement. J Bone Joint Surg Br 84:556-560
- Pfirrmann CW, Chung CB, Theumann NH, Trudell DJ, Resnick D (2001) Greater trochanter of the hip: attachment of the abductor mechanism and a complex of three bursae – MR imaging and MR bursography in cadavers and MR imaging in asymptomatic volunteers. Radiology 221:469-477
- Kagan A, 2nd (1999) Rotator cuff tears of the hip. Clin Orthop 135-140
- Chung CB, Robertson JE, Cho GJ, Vaughan LM, Copp SN, Resnick D (1999) Gluteus medius tendon tears and avulsive injuries in elderly women: imaging findings in six patients. AJR Am J Roentgenol 173:351-353
- 8. Petersilge CA (2001) MR arthrography for evaluation of the acetabular labrum. Skeletal Radiology 30:423-430
- 9. Cotten A, Boutry N Demondion X et al (1998) Acetabular labrum: MRI in asymptomatic volunteers. J Comp Assist Tomogr 22:1-7

- Lecouvet FE, Vande Berg BC, Malghem J et al (1996) MR imaging of the acetabular labrum: variations in 200 asymptomatic hips. AJR 167:1025-1028
- Abe I, Harada Y, Oinuma K et al (2000) Acetabular labrum: abnormal findings at MR imaging in asymptomatic hips. Radiology 216:576-581
- Hodler J, Yu JS, Goodwin D, Haghighi P, Trudell D, Resnick D (1995) MR arthrography of the hip: improved imaging of the acetabular labrum with histologic correlation. AJR 165:887-891
- Czerny C, Hofmann S, Neuhold A et al (1996) Lesions of the acetabular labrum: accuracy of MR imaging and MR arthrography in detection and staging. Radiology 200:225-230
- Czerny C, Hofmann S, Urban M et al (1999) MR arthrography of the adult acetabular-labral complex: correlation with surgery and anatomy. AJR 173:345-349
- Petersilge CA, Haque MA, Petersilge WJ, Lewin JS, Lieberman JM, Buly R (1996) Acetabular labral tears: evaluation with MR arthrography. Radiology 200:231-235
- McCarthy JC, Day B, Busconi B (1995) Hip arthroscopy: applications and technique. J Am Acad Orthop Surg 3:115-122
- Kerr, R (1997) MR Imaging of sports injuries of the hip and pelvis. Sem Musculoskeletal Radiol 1:65-82
  Tuite MJ, DeSmet AA (1994) MRI of selected sports injuries:
- Tuite MJ, DeSmet AA (1994) MRI of selected sports injuries: muscle tears, groin pain, and osteochondritis dissecans. Sem US, CT, MRI 15:318-340
- Miyanishi K, Yamamoto T, Nakshima Y et al (2001) Subchondral changes in transient osteoporosis of the hip. Skeletal Radiol 30:225-261
- Yamamoto T, Kubo T, Hirasawa Y, Noguchi Yasuo, Iwamoto Y, Sueishi K (1999) A clinicopathologic study of transient osteoporosis of the hip. Skeletal Radiol 28:621-627
- Vande Berg BC, Malghem J, Goffin EJ, Duprez TP, Maldague BE (1994) Transient epiphyseal lesions in renal transplant recipients: presumed insufficiency stress fractures. Radiology 191:403-407
- 22. Yamamoto T, Bullough PG (2000) The role of subchondral insufficiency fracture in rapid destruction of the hip joint. Arthritis & Rheumatism 43:2423-2427
- Hayes CW, Conway WF, Daniel WW (1993) MR imaging of bone marrow edema pattern: transient osteoporosis, transient bone marrow edema syndrome, or osteonecrosis. Radiographics 13:1001-1011
- Watson RM, Roach NA, Dalinka MK (2004) Avascular necrosis and bone marrow edema syndrome. Radiol Clin North Am 42:207-219

### Imaging of the Knee

D.A. Rubin<sup>1</sup>, W.E. Palmer<sup>2</sup>

<sup>1</sup> Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, MO, USA

<sup>2</sup> Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

### Introduction

This article addresses the spectrum of imaging modalities that are commonly used in the knee, and describes their roles in the evaluation of particular knee disorders. Emphasis is placed on magnetic resonance imaging (MRI) and its value in knee trauma and on the biomechanical approach to understanding patterns of injury.

### **Imaging Modalities**

Conventional radiographs are the initial radiologic study in most suspected knee disorders. Radiographs demonstrate joint spaces and bones, but are relatively insensitive to soft-tissue conditions (except those composed largely of calcium or fat), destruction of medullary bone, and early loss of cartilage. A minimum examination consists of an AP and lateral projection. In patients with acute trauma, performing the lateral examination cross-table allows identification of a lipohemarthrosis, an important clue to the presence of an intraarticular fracture [1]. The addition of oblique projections increases the sensitivity of the examination for nondisplaced fractures, especially those of the tibial plateau [2]. For the early detection of articular cartilage loss, a PA radiograph of both knees with the patient standing and knees mildly flexed is a useful adjunct projection. A joint space difference of 2 mm side-to-side correlates with grade III and higher chondrosis [3]. The tunnel projection is useful to demonstrate intercondylar osteophytes. In patients with anterior knee symptoms, an axial projection of the patellofemoral joint, such as a Merchant view, can evaluate the patellofemoral joint space and alignment [4].

Bone scintigraphy with an agent such as  $Tc^{99m}$ -MDP can screen the entire skeleton for metastatic disease. Scintigraphy also has a role in the detection of other radiographically occult conditions, such as nondisplaced fractures, and early stress fractures, osteomyelitis, and avascular necrosis, especially with three-phase technique. Bone scanning is a useful adjunct in the evaluation of painful knee arthroplasties [5]. Evaluation of a potential-

ly infected arthroplasty usually requires combining the bone scan with an additional scintigraphic examination, such as a sulfur colloid, labeled white blood cell, or inflammatory agent scan [6].

Sonography is largely limited to an evaluation of the extraarticular soft tissues of the knee but, with careful technique, at least partial visualization of the synovium and ligaments is also possible [7]. Ultrasound is useful in the evaluation of overuse conditions of the patellar tendon [8]. Also, sonography easily demonstrates popliteal (Baker's) cysts [9].

Computed tomography (CT) is used most frequently to evaluate intraarticular fractures about the knee, for planning complex orthopedic procedures, and for post-operative evaluation. Maximal diagnostic information may necessitate reformatting the transversely acquired dataset into orthogonal planes and/or 3D projections [10]. To facilitate reconstructions, multidetector-row helical acquisitions with thin collimation (sub-millimeter, if possible) are preferred [11]. Combining helical CT with arthrography makes it a viable examination for the detection of internal derangements, including meniscal and articular cartilage injuries [12, 13].

Magnetic resonance imaging has emerged as the premier imaging modality for the knee. It is the most sensitive, noninvasive test for the diagnosis of virtually all bone and soft-tissue disorders in and around the knee. Additionally, MRI provides information that can be used to grade pathology, guide therapy, prognosticate conditions, and evaluate treatment for a wide variety of orthopedic conditions in the knee. MR arthrography following the direct intraarticular injection of gadolinium-based contrast agents increases the value of the examination in selected knee conditions, including evaluation of the post-operative knee, detection and staging of chondral and osteochondral infractions, and discovery of intraarticular loose bodies [14, 15, 16].

High-quality knee MRI can be performed on high- or low-field systems with open, closed, or dedicated-extremity designs, as long as careful technique is used [17 18]. Use of a local coil is mandatory to maximize signal-tonoise ratio [19]. Images are acquired in transverse, coronal, and sagittal planes, often with mild obliquity on the sagittal and coronal images to optimize visualization of specific ligaments [20, 21]. A combination of different pulse sequences provides tissue contrast. Spin-echo T1weighted images demonstrate hemorrhage, as well as abnormalities of bone marrow, and extraarticular structures that are bounded by fat [22, 23]. Proton-density-weighted (long repetition time, short effective echo time) sequences are best for imaging fibrocartilage structures like the menisci [24]. T2- or T2\*-weighted images are used to evaluate the muscles, tendons, ligaments, and articular cartilage [25, 26]. These fluid-sensitive sequences can be obtained using spin-echo, fast spin-echo, or gradient-recalled techniques. Suppressing the signal from fat increases the sensitivity for detecting marrow and soft-tissue edema [27, 28]. 3D gradient-recalled acquisitions can provide thin contiguous slices for supplemental imaging of articular cartilage [29, 30]. To consistently visualize the critical structures in the knee, standard MRI should be done with a field-ofview no greater than 16 cm, 3- or 4-mm slice thickness, and imaging matrices of at least 192×256. Depending on the MR system and coil design, in order to achieve this spatial resolution with adequate signal-to-noise, other parameters, like the number of signals averaged and the receive bandwidth, may need to be optimized [31, 32].

### **Specific Disorders**

### **Bone and Articular Cartilage**

Osseous pathology in the knee encompasses a spectrum of traumatic, reactive, ischemic, infectious, and neoplastic conditions. Radiographs, CT, scintigraphy, and MRI each have a role imaging these disorders.

### Trauma

Most fractures are visible radiographically. A lipohemarthrosis indicates an intraarticular fracture, which may be radiographically occult, if it is nondisplaced [33]. The amount of depression and the congruence of the articular surface(s) determine the treatment and prognosis of tibial plateau fractures. The images need to accurately depict the amount of depression, as well as the presence, location, and size of any areas of articular surface step-off, gap, or die-punch depression. CT is better than radiography for this indication, with the use of multiplanar sagittal and coronal reconstructed images [34]. At some institutions, MR has supplanted CT. The MR examination not only shows the number and position of fracture planes, but also demonstrates associated soft-tissue lesions such as meniscus and ligament tears – that may affect surgical planning [35].

Other common fractures about the knee include patellar fractures, intercondylar eminence fractures, and avulsions. Patellar fractures with a horizontal component require internal fixation when they become distracted due to retraction of the proximal fragment by the pull of the quadriceps. Fractures of the intercondylar eminence and spines of the tibia may affect the attachment points of the cruciate ligaments. Elevation of a fracture fragment may occur due to the attachment of one of the cruciate ligaments. Avulsion fractures may look innocuous, but they can signal serious ligament disruptions. For example, a fracture of the lateral tibial rim (Segond fracture) is a strong predictor of anterior cruciate ligament disruption, while an avulsion of the medial head of the fibula (arcuate fracture) indicates disruption of at least a portion of the posterolateral corner [36, 37].

Bone scintigraphy, CT, or MRI are more sensitive than radiographs for nondisplaced fractures. A positive bone scan after trauma indicates a fracture, as long as there are no other reasons (osteoarthritis, Paget disease, etc.) evident radiographically. However, an abnormal bone scan still does not show the number and position of fracture lines, which impacts treatment. For this reason, and because of the low specificity of bone scintigraphy, CT and MRI have largely replaced it for this indication. MRI probably has an advantage over CT: when there is no fracture present, MRI can show soft-tissue injuries that may clinically mimic an occult fracture. On MRI examination, non-fat-suppressed T1-weighted images best demonstrate fractures, where they appear as very-lowsignal intensity linear or stellate lines surrounded by marrow edema, which has lower signal intensity than marrow fat, but is approximately isointense compared to muscle. On gradient-recalled, proton-density-weighted, and nonfat-suppressed T2-weighted images, fractures lines and marrow edema are often not visible. Marrow edema is most conspicuous on fat-suppressed T2-weighted or short-inversion time recovery (STIR) images, but the amount of edema may obscure underlying fracture lines.

Injuries to the articular surfaces often produce changes in the underlying subcortical bone. In children, these injuries are usually osteochondral, while in adults they may be purely chondral. The osteochondral infractions are visible radiographically, most often involving the lateral aspect of the medial femoral condyle. MRI is the study of choice to stage these lesions. On T2-weighted images, a thin line of fluid-intensity signal surrounding the base of the lesion indicates that the fragment is unstable. Similarly, the presence of small cysts in the base of the crater, or of an empty crater, indicates lesion instability, usually necessitating operative fixation or removal of the osteochondral fragment [38]. Lack of any high signal at the junction between a fragment and its parent bone indicates that the lesion has healed. The most difficult cases are those in which there is a broad area of high signal intensity that is less intense than fluid at the interface. In these instances, the high signal intensity may represent loose connective tissue of an unstable lesion or granulation tissue in a healing lesion. MR arthrography following the direct injection of gadolinium is helpful in this event: Gadolinium tracking around the base of the lesion indicates a loose, in-situ fragment [39].

In the knee, chondral injuries mimic meniscal tears clinically, but are radiographically occult. Arthroscopy, MRI with or without arthrography, or CT arthrography demonstrate these injuries. Arthrographic images show contrast filling a defect in the articular cartilage. Most of the traumatic cartilage injuries are full-thickness and have sharp, vertically oriented walls (unlike degenerative cartilage lesions, which may be partial-thickness, or fullthickness with sloped walls). To visualize small defects, non-arthrographic MR images need high contrast resolution between joint fluid and hyaline cartilage [40]. Useful sequences include T2-weighted spin-echo or fast spinecho ones, in which articular cartilage is dark and fluid is bright, or spoiled gradient-echo images, in which normal cartilage is bright and fluid is dark. A frequent associated finding is focal subchondral edema overlying the defect on fat-suppressed T2-weighted images. Often the subchondral abnormality will be more conspicuous than the chondral defect [41].

Stress fractures - whether of the fatigue or insufficiency type – occur about the knee. Once healing begins, radiographs show a band of sclerosis perpendicular to the long axis of the main trabeculae, with or without focal periosteal reaction. Rarely, a cortical fracture line is visible. Initially, however, stress fractures are radiographically occult. At this stage, either bone scintigraphy or MR examinations are more sensitive [42]. The imaging appearance is similar to that of traumatic fractures. Bone scans show a nonspecific, often linear, focus of intense uptake, with associated increased blood flow (on threephase studies). The MR appearance is a low-signal-intensity fracture line surrounded by a larger region of marrow edema. The proximal tibia is a common location for insufficiency fractures, especially in elderly, osteoporotic patients.

Magnetic resonance examination is also sensitive to lesser degrees of bone trauma. Marrow edema without a fracture line in a patient with a history of chronic repetitive injury represents a "stress reaction." If the offending activity continues without giving the bone time to heal, these injuries may progress to true stress fractures and macroscopic fractures. The term "bone bruise" or "bone contusion" describes trabecular microfracture due to impaction of the bone. Impaction can be due to blunt force from an object outside the body, or more commonly, from two bones striking each other after ligament injuries, subluxations, or dislocation-reduction injuries. Bone bruises appear as reticulated, ill-defined regions in the marrow that are isointense to muscle on T1-weighted images and hyperintense on fat-suppressed T2-weighted or STIR images [43, 44]. This pattern of signal abnormality is commonly referred to as the "bone-marrow edema pattern", even though granulation tissue and fibrosis dominate the histologic appearance [45]. The configuration of bone bruises is an important clue to the mechanism of injury, and it can account for elements of the patient's pain and may predict eventual cartilage degeneration [46, 47, 48]. However, the radiologist should avoid the temptation to label any area of marrow edema as a "bone bruise." This term is reserved for cases in which there is documented direct trauma, and may have medicolegal implications. The focal bone-marrow edema pattern is nonspecific, and is seen in a variety of other conditions – from ischemic, to reactive (subjacent to areas of degenerative chondrosis), to neoplastic and infectious.

### Ischemia and Infarction

Marrow infarction and avascular necrosis (AVN) result from a variety of insults, including endogenous and exogenous steroids, collagen vascular diseases, alcoholism, and hemoglobinopathies. An idiopathic form also occurs in the femoral condyles [49], sometimes precipitated by a meniscal tear or meniscectomy. Radiographically, AVN appears as sclerosis of the subchondral trabeculae, eventually leading to formation of a subchondral crescent and articular surface collapse. In the diaphyses, established infarcts have a serpiginous, sclerotic margin. Evolving infarcts may not show any radiographic findings. At this stage, bone scintigraphy will be positive (albeit nonspecifically) in the reactive margin surrounding the infarcted bone. On occasion, the actual area of infarction may show decreased tracer activity. On MR images, infarctions appear as geographic areas of abnormal marrow signal, either in the medullary shaft of a long bone or in the subchondral marrow (AVN). The signal intensity of the subchondral fragment and of the reactive surrounding bone vary based on the age of the lesion and other factors. As the infarction evolves, a typical serpiginous reactive margin becomes visible, often with a pathognomonic double-line sign on T2-weighted images: a peripheral low signal intensity line of demarcation surrounded by a parallel high-signal-intensity line representing the reactive margin [50].

### Replacement

Normal bone marrow around the knee is composed of a mixture of hematopoietic (red) and fatty (yellow) marrow. Processes that alter marrow composition are typically occult on all imaging modalities, except for specific nuclear marrow scans (using labeled sulfur colloid, for example) and on MR images. Normally, areas of yellow marrow are approximately isointense to subcutaneous fat on all pulse sequences, while red marrow is approximately isointense compared to muscle. In adults, the apophyseal and epiphyseal equivalents should contain fatty marrow. The most common marrow alteration encountered around the knee is hyperplastic red marrow. This can be seen physiologically associated with anemia, obesity, and cigarette smoking, as well as in athletes and persons living at high-altitudes [51, 52]. Unlike the case for pathologic marrow replacement, the signal intensity of red marrow expansion is isointense to muscle, islands of red marrow are separated by areas of residual yellow marrow, and the epiphyses are spared. However, in extreme cases – such as due to hemolytic anemia – the hyperplastic marrow can partly or completely replace the epiphyseal marrow [53].

Other alterations in marrow composition are less common, but relatively characteristic in their MR appearances. Irradiated and aplastic marrow is typically fatty [54]. Fibrotic marrow is low in signal intensity on all pulse sequences, and marrow in patients with hemosiderosis shows nearly a complete absence of signal [55].

### Destruction

Tumors and infections destroy trabecular and/or cortical bone. Subacute and chronic osteomyelitis produce predictable radiographic changes: cortical destruction, periosteal new bone formation, reactive medullary sclerosis, and, eventually, cloacae and sinus tracts. In these cases, the primary role of cross-sectional imaging is staging the infection. For example, CT is useful for surgical planning to identify a sequestrum or foreign body [56]. MRI can also help determine treatment in chronic osteomyelitis [57], by demonstrating non-drained abscesses, and by assessing the viability of the infected bone (by the presence or absence of enhancement after intravenous contrast administration). In patients with known chronic osteomyelitis, uptake by an inflammation-sensitive nuclear medicine agent (like gallium or labeled white blood cells), or focal high signal intensity of the marrow on T2weighted images, suggests superimposed active infection, although neither study is sufficiently specific enough to preclude biopsy, especially in cases in which the causative agent is uncertain.

Bones with acute osetomyelitis may be radiographically normal for the first 2 weeks of infection [58]. While CT scanning can show cortical destruction and marrow edema earlier than radiographs, MRI and nuclear medicine studies are typically the first-line studies. MR images show the marrow edema pattern, but to increase the specificity, osetomyelitis should only be diagnosed when there is also cortical destruction or an adjacent soft-tissue abscess, sinus tract or ulcer, at least in adults, in whom direct inoculation is much more common than hematogenous seeding [59].

Both benign and malignant bone tumors occur commonly around the knee. Radiographs should be the initial study in these patients, and are essential for predicting the biologic behavior of the tumor (by analysis of the zone of transition and the pattern of periostitis) as well as for identification of calcified matrix. The intraosseous extent of tumor and the presence and type of matrix are easiest to determine with CT examination. For staging beyond the bone (to the surrounding soft tissues, skip lesions in other parts of the same bone, and regional nodes), MR or CT are approximately equally effective [60]. In the future, PET scanning may be used to stage some bone tumors as well. Additionally, MRI is at least as sensitive as bone scintigraphy for detecting metastases, and at least as sensitive as radiography in patients with multiple myeloma, although MR is better suited to targeted regions rather than whole body screening in these conditions [61].

### Degeneration

Chondrosis refers to degeneration of articular cartilage. With progressive cartilage erosion, radiographs show the typical findings of osteoarthritis, namely, nonuniform joint-space narrowing and osteophyte formation. Before these findings are apparent, bone scintigraphy may show increased uptake in the subchondral bone adjacent to arthritic cartilage. The activity represents increased bone turnover associated with cartilage turnover. Direct visualization of the cartilage requires a technique that can visualize the contour of the articular surface. On standard CT examination, there is inadequate contrast between articular cartilage and joint fluid to visualize surface defects, while CT arthrography using dilute contrast can show even small areas of degeneration [62]. However, MRI is the most commonly used imaging modality to examine degenerated articular cartilage.

On MR images, internal signal-intensity changes do not reliably correlate with cartilage degeneration [63, 64]. Instead, the diagnosis of chondrosis is based on visualization of joint fluid (or injected contrast) within chondral defects at the joint surface [65]. The accuracy of MRI imaging increase for deeper and wider defects. Many different pulse sequences provide enough tissue contrast between fluid and articular cartilage. The most commonly used ones are T2-weighted fast spin-echo and fat-suppressed spoiled gradient recalled-echo sequences. T1-weighted spin-echo sequences are used in knees that have undergone arthrography with a dilute gadolinium mixture [66, 67, 68]. However, fat-suppressed T2-weighted images have the added advantage of showing reactive marrow edema in the subjacent bone (analogous to the subchondral uptake seen on bone scans), which is often a clue to the presence of small chondral defects in the overlying joint surface [69].

### Soft Tissues

Magnetic resonance imaging, with or without intraarticular or intravenous contrast, is the imaging study of choice for most soft-tissue conditions in and around the knee. Ultrasound can also be used in selected circumstances for relatively superficial structures.

### Fibrocartilage

The fibrocartilagenous menisci distribute the load of the femur on the tibia, and function as shock absorbers. There are two criteria for meniscal tears on MR images. The first is intrameniscal signal on a short-TE (T1-weighted, proton-density-weighted, or gradient-recalled) image that unequivocally contacts an articular surface of the meniscus. Intrameniscal signal that only possibly touches the meniscal surface is no more likely torn than

a meniscus containing no internal signal [70, 71]. The second criterion is abnormal meniscal shape [24]. In cross-section, the normal meniscus is triangular or bowtie shaped, with a sharp inner margin. Any variation from the normal shape – other than a discoid meniscus or one that has undergone partial meniscectomy – represents a meniscal tear.

In addition to diagnosing meniscal tears, the radiologist should describe the features of each meniscal tear that may affect treatment. These properties include the location of the tear (medial or lateral, horns or body, periphery or inner margin), the shape of the tear (longitudinal, horizontal, radial, or complex), the approximate length of the tear, the completeness of the tear (whether it extends partly or completely through the meniscus), and the presence or absence of an associated meniscal cyst. The radiologist should also note the presence of displaced meniscal fragments, which typically occur in the intercondylar notch or peripheral recesses [24].

A meniscal tear that heals spontaneously or following repair will often still contain intrameniscal signal on short-TE images that contacts the meniscal surface. When the abnormality is also present on a T2-weighted image, when there is a displaced fragment, or when a tear occurs in a new location, the radiologist can confidently diagnose a recurrent or residual meniscal tear [72]. If none of these features is present, MRI or CT examination after direct arthrography is useful. On an arthrographic examination, the presence of injected contrast within the substance of a repaired meniscus is diagnostic of a meniscal tear [73, 74]. The problem is compounded after a partial meniscectomy; in these cases both the meniscal shape and internal signal are unreliable signs of recurrent meniscal tear. Again, MR arthrography is the most useful noninvasive test for recurrent meniscal tears following partial meniscectomy [75].

### Ligaments

T2-weighted images demonstrate ruptures of the cruciate, collateral, and patellar ligaments. Both long-axis and cross-sectional images are important to examine. The direct sign of a ligament tear is partial or complete disruption of the ligament fibers [76]. While edema surrounding a ligament is typically seen in acute tears, edema surrounding an intact ligament is a nonspecific finding, which can be seen in bursitis or other soft tissue injuries, in addition to ligament tears [77]. Chronic ligament tears have a more varied appearance. Non-visualization of all ligament fibers or abnormal morphology of the scarred ligament fibers may be the only MR signs [78]. Secondary findings of ligament tears, such as bone contusions or subluxations, are useful when present, but do not supplant the primary findings, and do not reliably distinguish acute from chronic injuries, nor partial from complete tears [79].

Mucoid degeneration within ligaments sometimes occurs with aging. In the knee, the anterior cruciate ligament is most often affected. On MR images, the appearance is that of high-signal intensity amorphous material between the intact ligament fibers on T2-weighted images [80]. The ligament may appear enlarged in crosssection, and often there are associated intraosseous cysts formed near the ligament attachment points. It is important to distinguish degenerated from torn ligaments because degenerated ligaments are stable and do not require surgical intervention [81].

### **Muscles and Tendons**

The muscles around the knee are susceptible to direct and indirect injuries. Blunt trauma to a muscle results in a contusion. On T2-weighted or STIR MR images, contusions appear as high-signal-intensity edema spreading out from the point of contact in the muscle belly. Eccentric (stretching) injuries result in muscle strains. On MRI, these appear as regions of edema centered at the myotendonous junction, with partial or complete disruption of the tendon from the muscle in more severe cases [82]. Around the knee, muscle trauma affects the distal hamstrings, distal quadriceps, proximal gastrocnemius, soleus, popliteus, and plantaris muscles.

Chronic overuse of tendons results in degeneration or "tendonopathy". Tendonopathy can be painful or asymptomatic; but most importantly, tendonopathy weakens tendons, placing them at risk of rupture. The patellar, quadriceps, and semimembranosus tendons are most frequently involved around the knee. Ultrasound can also be used to evaluate these tendons. Sonographically, a degenerated tendon appears enlarged, with loss of the normal parallel fiber architecture, and often with focal hypoechoic or hyperechoic regions. A gap between the tendon fibers indicates that the process has progressed to partial or complete tear. Similarly, on MR images, focal or diffuse enlargement of a tendon with loss of its sharp margins indicates tendonopathy [83]. In those cases in which T2-weighted images show a focus of high signal intensity, surgical excision of the abnormal focus can hasten healing in refractory cases [84]. Partial or complete disruption of tendon fibers represents a tendon tear on MRI [85]. When macroscopic tearing is present, the radiologist should also examine the corresponding muscle belly for fatty atrophy (which indicates chronicity) or edema (suggesting a more acute rupture). If the tear is complete, the retracted stump should be located on the images as well. These last two tasks may require repositioning of the MR coil.

#### Synovium

While radiographs can show medium and large knee effusions, other modalities better demonstrate specific synovial processes. Fluid distention of a synovial structure has water attenuation on CT images, signal isointense to fluid on MR images, and is hypo- or anechoic with enhanced through-transmission on ultrasound images. A
popliteal or Baker's cyst represents distention of the posteromedial semimembranosus-gastrocnemius recess of the knee, and is easily seen with all cross-sectional modalities. At least 11 other named bursae occur around the knee. The most commonly diseased ones are probably the prepatellar, superficial infrapatellar, medial collateral ligament, and semimembranosus-tibial collateral ligament bursae.

Synovitis due to infection, trauma, inflammatory arthritis, or crystal disease is readily identifiable in the knee on both ultrasound and MR images. Power Doppler ultrasound or the use of ultrasound contrast agent may increase sensitivity for active synovitis [86]. On MRI examination, thickening of the usually imperceptibly thin synovial membrane and enhancement of the synovium following intravenous contrast administration indicates active synovitis [87].

Synovial metaplasia and neoplasia are uncommon. In the knee, primary synovial osteochondromatosis appears as multiple cartilaginous bodies within the joint on MR images, also visible on radiographs or CT if the bodies are calcified [88]. The signal intensities of the bodies vary depending on their composition. Diffuse pigmented villonodular synovitis and focal nodular synovitis demonstrate nodular, thickened synovium, which enhances following contrast administration. Hemosiderin deposition in the synovium – which is very low in signal intensity on all MR pulse sequences, with blooming on gradientecho images – is an important, though inconstant, clue to the diagnosis [89].

#### **Biomechanical Approach to Knee Trauma**

Knee trauma often produces predictable groupings of ligamentous and meniscal injuries [90]. Structures that perform related kinematic functions are damaged by the same traumatic mechanisms. When one supporting structure is disrupted, synergistic structures are jeopardized. Locations of meniscal tear, capsulo-ligamentous sprain, and osseous injury all provide clues about the mechanism of injury. By understanding the most common patterns of knee injury, a biomechanical approach can be used in the interpretation of MR images. The identification of abnormality in one structure should lead to a directed search for subtle abnormalities involving anatomically or functionally related structures, thereby improving diagnostic confidence.

In the biomechanical approach to knee trauma, MR images are interpreted with an understanding that structures with strong functional or anatomical relationships are often injured together. By deducing the traumatic mechanism, it is possible to improve diagnostic accuracy by taking a directed search for subtle, surgically relevant abnormalities that might otherwise go undetected. It may also be possible to communicate more knowledgeably with sports orthopedists, enjoy the interpretive process more thoroughly, and read scans faster. This following sections addresses the role of MRI following knee trauma, focusing on the most common traumatic mechanisms and associated injuries to stabilizing structures. Emphasis will be placed on the detection of clinically suspected or occult soft-tissue and bone abnormalities that could be exacerbated by repeat trauma or could lead to chronic instability and joint degeneration unless treated.

## **Biomechanical Principles**

Kinematic laws dictate normal joint motion and the biomechanics of injury [91]. Although the knee moves primarily as a hinge joint in the sagittal plane, it is also designed for internal-external rotation and abduction-adduction. Multidirectional mobility is gained at the expense of stability.

Throughout the normal range of knee motion, the menisci improve joint congruence and load distribution while the femorotibial contact points are shifting anteriorly and posteriorly. This movement of the joint is physiological, but the menisci must shift with the contact points to avoid entrapment and crush injury by the femoral condyles. Paired cruciate and collateral ligaments function collectively with the menisci to maintain joint congruence. The stress endured by each individual ligament depends on the position of the knee as well as the direction and magnitude of mechanical load. In external rotation, for example, the cruciate ligaments are lax whereas the collaterals become tense, resisting varus or valgus rocking. Conversely, in internal rotation, the collateral ligaments are lax whereas the cruciates become twisted around each other, pulling the joint surfaces together and resisting varus or valgus rocking. Within the physiological range of motion, the knee ligaments perform extremely complex, interdependent stabilizing functions.

Knee trauma is the most frequent cause of sports-related disability. In both contact and non-contact sports, knees are subjected to huge external forces that overpower stabilizing structures. Valgus force is directed at the lateral aspect of the joint, and varus force is directed at the medial aspect. During valgus force, tensile stress distracts the medial compartment of the knee and can tear the medial collateral ligament. The lateral compartment is distracted during varus stress, tearing the lateral collateral ligament. In the weight-bearing knee, valgus force also creates compressive load across the lateral compartment, which can cause impaction injury to the lateral femoral condyle and tibia. The medial compartment is compressed during varus stress, leading to impaction of the medial femoral condyle against the tibia. In the knee, the most common traumatic mechanisms combine valgus force with axial load. Therefore, compression with impaction injury usually occurs in the lateral compartment, whereas tension with distraction injury occurs in the medial compartment.

Sudden, violent tension will snap a ligament without elongating its fibers. If tension develops relatively slow-

ly, a ligament is more likely to stretch before tearing. Acute ligamentous injuries are graded clinically into three degrees of severity. In mild sprain (stretch injury), the ligament is continuous but lax. The ligament can return to normal function with appropriate conservative treatment. At operation, the fibers appear swollen and ecchymotic. MR images show an intact ligament that is thickened with variable surrounding edema or hemorrhage. In moderate sprain (partial tear), some but not all fibers are discontinuous. Remaining intact fibers may not be sufficient to stabilize the joint. At operation, torn fiber bundles hang loosely, and intact fibers are overstretched with marked edematous swelling and ecchymosis. MR images demonstrate prominent thickening and indistinct contour of the ligament combined with surrounding edema or hemorrhage. In severe sprain (rupture), the ligament is incompetent. At operation, torn fiber bundles hang loosely and can be moved easily. MR images show discontinuity of the ligament, retracted ligamentous margins and intervening hematoma.

# **Meniscal Injury**

Why are most trauma-related medial meniscal tears peripheral in location and longitudinally orientated, whereas lateral meniscal tears involve the free margin and are transverse in orientation?

Traumatic mechanism determines location and configuration of meniscal tear. When a distractive force separates the femorotibial joint, tensile stress is transmitted across the joint capsule to the meniscocapsular junction, creating traction and causing peripheral tear. Compressive force entraps, splays and splits the free margin of meniscus due to axial load across the joint compartment. Since the most common traumatic mechanisms in the knee involve valgus rather than varus load, the medial femorotibial compartment is distracted whereas the lateral compartment is compressed. Medial distraction means that the medial meniscus is at risk for peripheral avulsion injury at the capsular attachment site. Lateral compression means that the lateral meniscus is at risk for entrapment and tear along the free margin.

In the musculoskeletal system, structures are torn or avulsed at sites where they are fixed, but can escape injury in regions where they are mobile. Compared to the lateral meniscus, the medial meniscus is more firmly attached to the capsule along its peripheral border, and is far less mobile. Normal knee motion involves greater translation of the femorotibial contact point in the lateral compartment. In order to shift with the condyle and avoid injury, the lateral meniscus requires a looser capsular attachment than the medial meniscus.

The firm attachment of medial meniscus is a critical factor in its propensity for trauma-related injury. Since the medial meniscus is tightly secured by meniscofemoral and meniscotibial ligaments along the joint line, it is subjected to greater tensile stress with lesser degrees of distraction, translocation or rotation. Trauma-related medial meniscal tears tend to be located at the posteromedial corner (posterior to the medial collateral ligament) because the capsule is more organized and thickened in this location, and its meniscal attachment is tightest. Anatomists and orthopedists have long recognized the pathophysiological importance of this capsular anchor, which is called the posterior oblique ligament [91]. Although the posterior oblique ligament can be dissected free in most cadaver knees, it is only rarely identified on MR images. Degenerative (attrition) tears of the medial meniscus also predominate posteromedially, but they involve the thinner inner margin of the meniscus rather than the thicker periphery.

The trauma-related medial meniscal tear demonstrates a vertical orientation that can extend across the full thickness of the meniscus (from superior to inferior surface), involve a peripheral corner of the meniscus, or redirect itself obliquely towards the free margin of the meniscus. Once established, this vertical tear can propagate over time following the normal fiber architecture of the meniscus. Propagation to the free margin creates a flap, or parrot-beak, configuration. If the tear propagates longitudinally into the anterior and posterior meniscal thirds, the unstable inner fragment can become displaced into the intercondylar notch (bucket handle tear). The degree of longitudinal extension should be specified in the MRI report because the greater the length of torn meniscus, the greater the eventuality of displaced fragment. Orthopedists recognize an association between longitudinal tears and mechanical symptoms, and may decide to repair or resect the inner meniscal fragment before it becomes displaced and causes locking or a decreased range of motion. If an unstable fragment detaches anteriorly or posteriorly, it can pivot around the remaining attachment site and rotate into an intraarticular recess or the weight-bearing compartment. The identification and localization of a displaced meniscal fragment can be important in the preoperative planning of arthroscopic surgery.

During valgus force and medial joint distraction, tensile stress can avulse the capsule away from the meniscus (meniscocapsular separation), with or without a small corner piece of the meniscus, rather than tear the full thickness of the meniscus. Meniscocapsular injury may be an important cause of disability that can be treated surgically by primary reattachment of the capsule. Since the capsule stabilizes the medial meniscus, meniscocapsular separation or peripheral meniscal avulsion can cause persistent pain and lead to posteromedial instability with eventual degenerative change. On MR images, meniscocapsular injury is more difficult to identify than meniscal tear. Localized edema and focal fluid collection or hematoma may be present in the acute and subacute time periods, but eventually resolve with scarring and apparent reattachment of the capsule to meniscus. Similarly, small avulsed corners of meniscus may be difficult to identify unless a directed search is made for them.

The same valgus force that distracts the medial compartment also compresses the lateral compartment. Since the lateral meniscus is loosely applied to the joint capsule, it moves freely with the condyle and usually escapes entrapment. During axial load across the lateral compartment, the meniscus is sometimes crushed, which splays and splits the free margin, creating a radial (transverse) tear. Radial tears of the lateral meniscus usually originate at the junction of anterior and middle meniscal thirds. They are most difficult to identify on coronal images since they are vertically orientated in the coronal plane. Thin-slice, high-resolution sagittal images optimize the visualization of small radial tears. Sometimes, a fortuitous axial slice through the lateral meniscus is the only image that demonstrates the tear and allows diagnostic confidence. Over time, a radial tear propagates peripherally, transecting the lateral meniscus. If the tear extends all the way to the joint capsule, fluid may leak into the extraarticular space along the lateral joint line, resulting in meniscal cyst formation just posterior to the iliotibial band.

# Anatomical and Functional Synergism of Structures

Supporting structures function synergistically to stabilize the knee. Synergistic structures perform complementary kinematic roles in maintaining joint congruence. They are stressed by the same joint position or mechanical load, and therefore are at risk for combined injuries when that joint position or mechanical load exceeds physiological limits. When one stabilizing structure is disrupted, synergistic structures are jeopardized.

A group of structures that stabilize the knee and exhibit synergism in one position often relinquish that stabilizing function to a different group of structures when the knee position changes [91]. During internal rotation of the knee, the anterior and posterior cruciate ligaments develop functional synergism by coiling around each other, becoming taut, pulling the articular surfaces together and checking excessive internal rotation. During external rotation, the cruciates become lax and lose their stabilizing inter-relationship, but the medial and lateral collateral ligaments develop functional synergism as they both become tightened, pressing the articular surfaces together and checking external rotation beyond physiological limits.

The anterior cruciate and medial collateral ligaments are parallel, functionally related structures that course posteroanteriorly from femur to tibia and together maintain joint congruence when knee flexion and valgus force are combined with external rotation. The posterior cruciate and lateral collateral ligaments are also parallel structures that course anteroposteriorly from femur to tibia and together maintain joint isometry during internal rotation of the knee combined with flexion and varus force. Therefore, depending on knee position and the direction of mechanical load, different structures are functioning synergistically to stabilize the joint.

# Medial Collateral Ligament and Medial Meniscus

The medial collateral ligament complex comprises superficial and deep capsular fibers. The superficial component, also called tibial collateral ligament, resists both valgus force and external rotation. The tibial collateral ligament is the primary restraint to valgus force in the knee, providing 60-80% of the resistance, depending on the degree of knee flexion (greatest stabilizing role occurs at 25-30° of flexion). The deep fibers of medial collateral ligament form the joint capsule, which includes femorotibial fibers that pass directly from bone to bone, as well as meniscofemoral and meniscotibial fibers. These deep fibers provide minimal resistance to valgus force.

The medial collateral ligament and medial meniscus are anatomically related through the deep capsular fibers, which attach to the meniscus at the meniscocapsular junction. These deep meniscocapsular and superficial ligamentous fibers simultaneously develop tension during valgus force, and therefore are often injured together during excessive valgus force. Besides this anatomical synergism, the medial collateral ligament and medial meniscus are functionally related through the posterior oblique ligament at the posteromedial corner of the knee. These structures are both stressed by external rotation, with or without valgus force. During sports-related trauma, which factors determine whether the medial collateral ligament or medial meniscus suffers greatest injury? In large part, it depends on the degree of external rotation compared to medial joint distraction. Pure valgus force is more likely to injure the medial collateral ligament and subjacent medial meniscus; pure external rotation is more likely to injure the posterior oblique ligament (meniscocapsular junction) or medial meniscus posterior to medial collateral ligament. In combined valgus-external rotation, both of these medial structures are injured.

Magnetic resonance imaging is clinically relevant in the assessment of medial collateral ligament injury because findings on physical examination may be subtle, even in complete rupture. Orthopedists often request MRI in order to differentiate medial collateral ligament tear from medial meniscal tear, since these injuries have overlapping clinical symptoms. Although high-grade tears of the tibial collateral ligament are best characterized on coronal MR images, low-grade tears are better demonstrated on axial images. The anterior fibers of tibial collateral ligament develop greatest tension during external rotation and, therefore, are the first to tear. The axial plane is ideal for showing focal abnormalities limited to these anterior fibers, such as thickening or attenuation, displacement from bone, and surrounding edema or hemorrhage. In mild sprain of the medial collateral ligament, coronal MR images will show the normal posterior fibers, leading to false-negative diagnosis.

If MR images demonstrate sprain of the tibial collateral ligament, a knee-jerk reflex (pun intended) should next occur: focus attention on the meniscocapsular junction. First on coronal images, follow the peripheral border of meniscus posteriorly from the level of tibial collateral ligament to the posteromedial corner, searching for contour abnormalities and soft-tissue edema or hemorrhage along the joint line. Then, on sagittal images, follow the medial meniscus and meniscocapsular junction medially from the posterior thirds to the posteromedial corner. Depending on the knee position during imaging, either the coronal or sagittal images may better demonstrate peripheral meniscal tear or avulsion at the posteromedial corner.

# Anterior Cruciate Ligament and Medial Meniscus

The anterior cruciate ligament is made up of two bundles. The anterolateral bundle is tighter in knee flexion and the posterolateral bundle is tighter in extension. The anterior cruciate ligament is the primary restraint to anterior tibial displacement, providing 75-85% of resistance depending on the degree of knee flexion. Tension is least at 40-50° of flexion, and greatest at either 30° or 90° of flexion [92,93]. Quadriceps contraction pulls the tibia forward and creates greatest stress on the anterior cruciate ligament at 30° of knee flexion. Because of this quadriceps effect, the tibia is more likely to translocate anteriorly if the anterior cruciate ligament is torn when the knee is flexed. The posterior oblique ligament is the major secondary restraint to anterior tibial translocation.

Tears of the anterior cruciate ligament are extremely common in many different sports, such as football, basketball, and skiing. A classic mechanism for ligament injury is the pivot shift, when valgus stress and axial load are combined with forceful twisting of the knee as the athlete plants his or her foot and quickly turns direction. Rupture of the anterior cruciate ligament is more common than partial tear, since fiber failure usually occurs simultaneously rather than sequentially. In this way, the anterior cruciate ligament is different from the tibial collateral ligament, which tears sequentially from anterior to posterior.

The anterior cruciate and posterior oblique ligaments are functionally synergistic as primary and secondary restraints of anterior tibial displacement [91]. When one of these stabilizing structures is disrupted, the other is jeopardized. At the moment of anterior cruciate rupture, for example, residual energy causes the tibia to shift anteriorly. The femoral condyle is a physical barrier that prevents the posterior thirds of the medial meniscus from moving freely with the tibia. As the medial tibial plateau slides forward, tension builds in the meniscotibial fibers of the posterior oblique ligament and is transmitted to the meniscocapsular junction. Excessive traction tears the capsule or meniscus. Conversely, the posterior oblique ligament or medial meniscus may tear before the anterior cruciate ligament. Whether this tear results from external rotation or valgus force or both, the anterior cruciate ligament becomes the last remaining check against anterior tibial translocation, markedly increasing its risk for rupture.

Rupture of the anterior cruciate ligament is often obvious or strongly suspected based on history and physical examination. An orthopedist requests MRI not to confirm ligamentous rupture, but rather to identify other intraarticular lesions that might further destabilize the knee. The absence or presence of such a lesion may determine whether the orthopedist decides to prescribe conservative treatment, or repair the lesion at the same time as the anterior cruciate ligament. For example, if MR images show a destabilizing meniscocapsular injury at the posteromedial corner, primary repair might be performed (rather than subtotal meniscectomy) in conjunction with anterior cruciate reconstruction.

High-grade tears of the anterior cruciate ligament are easily identified on sagittal MR images. In the acute setting, mass-like hematoma occupies the expected location of the ligament, which may be completely invisible. After several days or weeks, the torn ligamentous margins become organized and better defined as thickened stumps separated from each other by a variable distance. Axial images are superb for confirming a normal ligament that is indistinct in the sagittal plane due to volume averaging. If the anterior cruciate ligament can be followed from femur to tibia on sequential axial images, its appearance in other planes is irrelevant. Partial tear is unusual, but may be characterized by edema or hemorrhage surrounding and separating the cruciate bundles, which appear indistinct but continuous. Once identified, anterior cruciate tear, same as for medial collateral tear, should lead automatically to a directed search for traumatic injury at the meniscocapsular junction.

Lateral osseous injury is commonly associated with anterior cruciate rupture [94]. The bone abnormalities may not be evident on radiographs, but are easily recognized as kissing contusions or minimally depressed fractures involving the weight-bearing femoral condyle, and the posterior rim of tibial plateau. Since the osseous lesions are not directly opposite each other in the lateral compartment, they provide conclusive and graphic evidence of tibial translocation at the time of injury. In the adult, this extent of translocation is not considered possible without rupture of the anterior cruciate ligament. Valgus force and axial load often cause impaction injury in the lateral osseous compartment, but the pattern of bone marrow abnormality depends on whether the anterior cruciate ruptures or remains intact.

# **Medial Unhappy Triad**

The majority of combination injuries occur when stress limits are exceeded in one of two extreme positions. In flexion, full motion of the knee ranges from valgus-external rotation to varus-internal rotation. Within this range, the joint can be actively exercised without danger of injury. Combination injury occurs when an additional valgus force acts on the knee that already is in extreme valgus-external rotation, or when an additional varus force acts on the knee that is in extreme varus-internal rotation. Based on early orthopedic literature, 80% of these combination injuries take the form of medial or lateral "unhappy triads" [91].

Medial combined injuries are 10-20 times more frequent than lateral combined injuries. In the medial triad (O'Donaghue's triad), excessive valgus stress in the externally rotated knee injures the tibial collateral ligament, anterior cruciate ligament and medial meniscus (meniscocapsular junction). In the era of arthroscopy and MRI, lateral meniscal tear is now recognized as a common associated finding. Therefore, the medial triad is sometimes an even unhappier medial tetrad. In the lateral triad, excessive varus stress in the internally rotated knee injures the fibular collateral ligament, posterior cruciate ligament and lateral meniscus (popliteus muscle or tendon).

Biomechanical principles can be applied to more than just image interpretation. Picture yourself playing basketball and leaping high off the ground to grab a rebound, only to land on another player's foot and twist your knee; running for a touchdown, but getting tackled from the side as you plant your foot to sidestep your opponent; circling the goal in a lacrosse game, then turning quickly towards the net to split the defense while pushing forcefully but awkwardly off your foot; enjoying the scenery along a ski trail, but catching your ski tip on a protruding tree root while losing control on an ice patch. As you are falling to the ground in the agony of medial triad injury, it is possible to recognize and construct mentally the sequence of traumatic events occurring in your knee.

In any of these scenarios, the most likely knee position is valgus-external rotation with some flexion and abduction. Valgus stress tightens the medial collateral ligament, external rotation tenses the posterior oblique ligament at the meniscocapsular junction, and combined valgus-external rotation stretches the anterior cruciate ligament over the lateral femoral condyle. The stabilizing structure that first gives way depends on complex factors, such as the degree of knee flexion and abduction. Excessive valgus force may first tear the deep fibers of the medial collateral ligament, followed by the stronger superficial fibers. As the medial compartment begins to distract, axial load across the lateral compartment entraps and crushes the free margin of the lateral meniscus. At the same time, traction on the posterior oblique ligament avulses the periphery of medial meniscus or tears the meniscocapsular junction. Since the posteromedial corner is now destabilized, the anterior cruciate ligament becomes the primary check against further external rotation. As the tibia continues to externally rotate and slide anteriorly in the medial compartment, all tension is transferred to the anterior cruciate ligament, which is snapped over the lateral femoral condyle. There is no longer passive restraint to anterior translocation, so the entire tibia can shift forward, pulled by the extensor mechanism and quadriceps contraction. Before the joint can reduce itself, continued valgus force and axial load cause impaction across the lateral compartment, with fracture or contusion involving the lateral femoral condyle and the posterior rim of tibial plateau.

# Patterns of Osseous Injury on Magnetic Resonance Images

Osseous injury is an expected finding following knee trauma. MRI is able to demonstrate non-displaced fractures that are not visible on plain radiographs, as well as trabecular contusions. The locations of these osseous abnormalities and their patterns of bone-marrow edema provide additional clues about the traumatic mechanism [95].

Traumatic mechanisms in the knee combine impaction and distraction. Impaction is most closely associated with depressed fracture or osseous contusion, although crushrelated meniscal or cartilage tear may also occur. Distraction rather than avulsion fracture is far more likely to cause ligament injury. Due to the function of paired cruciate and collateral ligaments, compressive load on one side of the knee occurs simultaneously with contralateral tensile stress. During anteromedial impaction of the knee, for example, kissing contusions of the femoral condyle and tibial plateau are associated with lateral collateral sprain or avulsion fracture of the fibular head [96].

On MR images, impaction and distraction fractures show differences that can be explained by their biomechanical etiologies. Since impaction injury results from compressive load, force is transmitted across subchondral bone and dissipated throughout trabecular bone. The fracture line represents compacted trabecular bone or, in the subacute setting, intramedullary callus formation. Surrounding bone-marrow edema or hemorrhage is most prominent closest to the fracture line, and then decreases with distance from the fracture, reflecting the dissipation of compressive forces at the time of injury. Although fatsuppressed T2-weighted images are more sensitive in the detection of marrow edema or hemorrhage, T1-weighted images better demonstrate the fracture line. Trabecular contusion, or microfracture, is diagnosed if no discrete fracture line is visible on T1-weighted images.

Avulsion fracture results from sudden, violent tension that is transmitted to cortical bone by the tendon, ligament or joint capsule. Whereas an impaction fracture fragment shows depression and prominent surrounding bone-marrow edema on MR images, a distraction fracture fragment shows diastasis from its donor site and minimal surrounding marrow edema. Decreased or absent osseous edema reflects the direction of mechanical force away from bone. Since there is no energy deposited in trabecular bone, there is no contusion.

Small avulsion fracture fragments that are obvious on plain radiographs may be difficult or impossible to visualize on MR images. Poor visualization reflects both the absence of marrow fat within the distracted fragment as well as the absence of sentinel bone-marrow edema surrounding the donor site. Larger avulsed fragments contain trabecular bone and marrow fat, which have high signal intensity on T1-weighted images and are conspicuous against the surrounding lower signal intensity of soft-tissue edema and hemorrhage. If the avulsed fragment contains no trabecular bone, the only indication of fracture may be cortical discontinuity at its donor site.

On MR images, the likelihood of identifying small avulsed cortical fragments is improved by inspecting the usual locations of avulsion based on the suspected mechanism of injury. There are seven locations for osseous avulsion in the knee: the medial femoral condyle at the attachment of the medial collateral ligament; the intercondylar eminence at attachments of both cruciate ligaments: the anterior part of the intercondular eminence at attachment of the anterior cruciate ligament; the posterior part of the intercondylar eminence at the attachment of the posterior cruciate ligament; the lateral tibial rim at the attachment of the lateral capsule (Segond fracture); fibular head at attachment of the fibular collateral ligament conjoined with the biceps femoris tendon; and the medial aspect of the patella at the attachment of the retinaculum.

In the knee, the avulsion fracture fragments that are most difficult to identify involve the lateral tibial rim and fibular head. These locations should be inspected whenever there is evidence for distraction injury involving the lateral compartment of the knee, or impaction injury involving the medial compartment [97]. Evidence of lateral distraction injury includes sprain of the fibular collateral ligament and strain of the iliotibial band or popliteus tendon. Anteromedial kissing contusions are closely associated with posterolateral avulsion injury. If lateral distraction fracture is suspected based on MRI findings, it is reasonable to recommend plain radiography to exclude small cortical avulsions.

#### Summary

Multiple traumatic, degenerative, inflammatory, infectious, and neoplastic conditions occur in and around the knee joint. Radiographs, ultrasound, CT, MR, and arthrography each play a role in the imaging evaluation of these conditions. Imaging is important not only to detect or exclude disease, but also to stage, guide therapy, follow up, and prognosticate knee disorders.

Knee injury is the most frequent cause of sports-related disability. Because common traumatic mechanisms

#### References

 Lee JH, Weissman BN, Nikpoor N et al (1989) Lipohemarthrosis of the knee: a review of recent experiences. Radiology 173:189-191

thus improving diagnostic confidence.

- 2. Gray SD, Kaplan PA, Dussault RG et al (1997) Acute knee trauma: how many plain film views are necessary for the initial examination? Skeletal Radiol 26:298-302
- 3. Rosenberg TD, Paulos LE, Parker RD et al (1988) The fortyfive-degree posteroanterior flexion weight-bearing radiograph of the knee. J Bone Joint Surg [Am] 70:1479-1483
- 4. Jones AC, Ledingham J, McAlindon T et al (1993) Radiographic assessment of patellofemoral osteoarthritis. Ann Rheum Dis 52:655-658
- Smith SL, Wastie ML, Forster I (2001) Radionuclide bone scintigraphy in the detection of significant complications after total knee joint replacement. Clin Radiol 56:221-224
- Pelosi E, Baiocco C, Pennone M et al (2004) <sup>99m</sup>Tc-HMPAOleukocyte scintigraphy in patients with symptomatic total hip or knee arthroplasty: improved diagnostic accuracy by means of semiquantitative evaluation. J Nucl Med 45:438-444
- 7. Bouffard JA, Dhanju J (1998) Ultrasonography of the knee. Semin Musculoskelet Radiol 2:245-270
- Khan KM, Bonar F, Desmond PM et al (1996) Patellar tendinosis (jumper's knee) : findings at histopathologic examination, US, and MR imaging. Victorian Institute of Sport Tendon Study Group. Radiology 200:821-827
- Ward EE, Jacobson JA, Fessell DP et al (2001) Sonographic detection of Baker's cysts: comparison with MR imaging. Am J Roentgoenol 176:373-380
- Wicky S, Blaser PF, Blanc CH et al (2000) Comparison between standard radiography and spiral CT with 3D reconstruction in the evaluation, classification and management of tibial plateau fractures. Eur Radiol 10:1227-1232
- Buckwalter KA, Farber JM (2004) Application of multidetector CT in skeletal trauma. Semin Musculoskelet Radiol 8:147-156
- 12. Mutschler C, Vande Berg BC, Lecouvet FE et al (2003) Postoperative meniscus: assessment at dual-detector row spiral CT arthrography of the knee. Radiology 228:635-641
- Vande Berg BC, Lecouvet FE, Poilvache P et al (2002) Assessment of knee cartilage in cadavers with dual-detector spiral CT arthrography and MR imaging. Radiology 222:430-436
- 14. Brossmann J, Preidler KW, Daenen B et al (1996) Imaging of osseous and cartilaginous intraarticular bodies in the knee: comparison of MR imaging and MR arthrography with CT and CT arthrography in cadavers. Radiology 200:509-517
- 15. Sciulli RL, Boutin RD, Brown RR et al (1999) Evaluation of the postoperative meniscus of the knee: a study comparing conventional arthrography, conventional MR imaging, MR arthrography with iodinated contrast material, and MR arthrography with gadolinium-based contrast material. Skeletal Radiol 28:508-514
- Magee T, Shapiro M, Rodriguez J, Williams D (2003) MR arthrography of postoperative knee: for which patients is it useful? Radiology 229:159-163

- Barnett MJ (1993) MR diagnosis of internal derangements of the knee: effect of field strength on efficacy. Am J Roentgoenol 161:115-118
- Franklin PD, Lemon RA, Barden HS (1997) Accuracy of imaging the menisci on an in-office, dedicated, magnetic resonance imaging extremity system. Am J Sports Med 25:382-388
- Rubin DA, Kneeland JB (1994) MR imaging of the musculoskeletal system: technical considerations for enhancing image quality and diagnostic yield. Am J Roentgoenol 163:1155-1163
- 20. Buckwalter KA, Pennes DR (1990) Anterior cruciate ligament: oblique sagittal MR imaging. Radiology 175:276-277
- Yu JS, Salonen DC, Hodler J et al (1996) Posterolateral aspect of the knee: improved MR imaging with a coronal oblique technique. Radiology 198:199-204
- 22. Vande Berg BC, Malghem J, Lecouvet FE et al (1998) Classification and detection of bone marrow lesions with magnetic resonance imaging. Skeletal Radiol 27:529-545
- 23. Bush CH (2000) The magnetic resonance imaging of musculoskeletal hemorrhage. Skeletal Radiol 29:1-9
- Rubin DA, Paletta GA Jr (2000) Current concepts and controversies in meniscal imaging. Magn Reson Imaging Clin North Am 8:243-270
- 25. Ha TPT, Li KC, Beaulieu CF et al (1998) Anterior cruciate ligament injury: fast spin-echo MR imaging with arthroscopic correlation in 217 examinations. Am J Roentgoenol 170:1215-1219
- 26. Sonin AH, Pensy RA, Mulligan ME et al (2002) Grading articular cartilage of the knee using fast spin-echo proton density-weighted MR imaging without fat suppression. Am J Roentgoenol 179:1159-1166
- Kapelov SR, Teresi LM, Bradley WG et al (1993) Bone contusions of the knee: increased lesion detection with fast spineecho MR imaging with spectroscopic fat saturation. Radiology 189:901-904
- Weinberger E, Shaw DW, White KS et al (1995) Nontraumatic pediatric musculoskeletal MR imaging: comparison of conventional and fast-spin-echo short inversion time inversion-recovery technique. Radiology 194:721-726
- 29. Recht MP, Piraino DW, Paletta GA et al (1996) Accuracy of fat-suppressed three-dimensional spoiled gradient-echo FLASH MR imaging in the detection of patellofemoral articular cartilage abnormalities. Radiology 198:209-212
- Disler DG, McCauley TR, Kelman CG et al (1996) Fat-suppressed three-dimensional spoiled gradient-echo MR imaging of hyaline cartilage defects in the knee: comparison with standard MR imaging and arthroscopy. Am J Roentgoenol 167:127-132
- Woertler K, Strothmann M, Tombach B et al (2000) Detection of articular cartilage lesions: experimental evaluation of lowand high-field-strength MR imaging at 0.18 and 1.0 T. J Magn Reson Imaging 11:678-685
- Kladny B, Gluckert K, Swoboda B et al (1995) Comparison of low-field (0.2 Tesla) and high-field (1.5 Tesla) magnetic resonance imaging of the knee joint. Arch Orthop Trauma Surg 114:281-286
- 33. Lee JH, Weissman BN, Nikpoor N et al (1989) Lipohemarthrosis of the knee: a review of recent experiences. Radiology 173:189-191
- 34. Wicky S, Blaser PF, Blanc CH et al (2000) Comparison between standard radiography and spiral CT with 3D reconstruction in the evaluation, classification and management of tibial plateau fractures. Eur Radiol 10:1227-1232
- 35. Kode L, Lieberman JM, Motta AO et al (1994) Evaluation of tibial plateau fractures: efficacy of MR imaging compared with CT. Am J Roentgoenol 163:141-147
- Campos JC, Chung CB, Lektrakul N et al (2001) Pathogenesis of the Segond fracture: anatomic and MR imaging evidence of an iliotibial tract or anterior oblique band avulsion. Radiology 219:381-386

- 37. Huang GS, Yu JS, Munshi M et al (2003) Avulsion fracture of the head of the fibula (the "arcuate" sign) : MR imaging findings predictive of injuries to the posterolateral ligaments and posterior cruciate ligament. Am J Roentgoenol 180:381-387
- De Smet AA, Ilahi OA, Graf BK (1996) Reassessment of the MR criteria for stability of osteochondritis dissecans in the knee and ankle. Skeletal Radiol 25:159-163
- Kramer J, Stiglbauer R, Engel A et al (1992) MR contrast arthrography (MRA) in osteochondrosis dissecans. J Comput Assist Tomogr 16:254-260
- Speer KP, Spritzer CE, Goldner JL et al (1991) Magnetic resonance imaging of traumatic knee articular cartilage injuries. Am J Sports Med 19:396-402
- Rubin DA, Harner CD, Costello JM (2000) Treatable chondral injuries in the knee: frequency of associated focal subchondral edema. Am J Roentgoenol 174:1099-1106
- 42. Spitz DJ, Newberg AH (2002) Imaging of stress fractures in the athlete. Radiol Clin North Am 40:313-331
- 43. Kapelov SR, Teresi LM, Bradley WG et al (1993) Bone contusions of the knee: increased lesion detection with fast spinecho MR imaging with spectroscopic fat saturation. Radiology 189:901-904
- 44. Arndt WF 3rd, Truax AL, Barnett FM et al (1996) MR diagnosis of bone contusions of the knee: comparison of coronal T2-weighted fast spin-echo with fat saturation and fast spinecho STIR images with conventional STIR images. Am J Roentgoenol 166:119-124
- 45. Zanetti M, Bruder E, Romero J, Hodler J (2000) Bone marrow edema pattern in osteoarthritic knees: correlation between MR imaging and histologic findings. Radiology 215:835-840
- Sanders TG, Medynski MA, Feller JF, Lawhorn KW (2000) Bone contusion patterns of the knee at MR imaging: footprint of the mechanism of injury. Radiographics 20 Spec No:S135-151
- 47. Wright RW, Phaneuf MA, Limbird TJ, Spindler KP (2000) Clinical outcome of isolated subcortical trabecular fractures (bone bruise) detected on magnetic resonance imaging in knees. Am J Sports Med 28:663-667
- Costa-Paz M, Muscolo DL, Ayerza M et al (2001) Magnetic resonance imaging follow-up study of bone bruises associated with anterior cruciate ligament ruptures. Arthroscopy 17:445-449
- Björkengren AG, AlRowaih A, Lindstrand A et al (1990) Spontaneous osteonecrosis of the knee: value of MR imaging in determining prognosis. Am J Roentgoenol 154:331-336
- Mitchell DG, Rao VM, Dalinka MK et al (1987) Femoral head avascular necrosis: correlation of MR imaging, radiographic staging, radionuclide imaging, and clinical findings. Radiology 162:709-715
- Deutsch AL, Mink JH, Rosenfelt FP et al (1989) Incidental detection of hematopoietic hyperplasia on routine knee MR imaging. Am J Roentgoenol 152:333-336
- 52. Shellock FG, Morris E, Deutsch AL et al (1992) Hematopoietic bone marrow hyperplasia: high prevalence on MR images of the knee in asymptomatic marathon runners. Am J Roentgoenol 158:335-338
- 53. Rao VM, Mitchell DG, Rifkin MD et al (1989) Marrow infarction in sickle cell anemia: correlation with marrow type and distribution by MRI. Magn Reson Imaging 7:39-44
- Remedios PA, Colletti PM, Raval JK et al (1988) Magnetic resonance imaging of bone after radiation. Magn Reson Imaging 6:301-304
- 55. Lanir A, Aghai E, Simon JS et al (1986) MR imaging in myelofibrosis. J Comput Assist Tomogr 10:634-636
- Hernandez RJ (1985) Visualization of small sequestra by computerized tomography. Report of 6 cases. Pediatr Radiol 15:238-241
- 57. Mason MD, Zlatkin MB, Esterhai JL et al (1989) Chronic complicated osteomyelitis of the lower extremity: evaluation with MR imaging. Radiology 173:355-359
- Capitano MA, Kirkpatrick JA (1970) Early roentgen observations in acute osteomyelitis. Am J Roentgoenol 108:488-490

- 59. Erdman WA, Tamburro F, Jayson HT et al (1991) Osteomyelitis: characteristics and pitfalls of diagnosis with MR imaging. Radiology 180:533-539
- 60. Panicek DM, Gatsonis C, Rosenthal DI et al (1997) CT and MR imaging in the local staging of primary malignant musculoskeletal neoplasms: Report of the Radiology Diagnostic Oncology Group. Radiology 202:237-246
- Daffner RH, Lupetin AR, Dash N et al (1986) MRI in the detection of malignant infiltration of bone marrow. Am J Roentgoenol 146:353-358
- Vande Berg BC, Lecouvet FE, Poilvache P et al (2002) Assessment of knee cartilage in cadavers with dual-detector spiral CT arthrography and MR imaging. Radiology 222:430-436
- Brown TR, Quinn SF (1993) Evaluation of chondromalacia of the patellofemoral compartment with axial magnetic resonance imaging. Skeletal Radiol 22: 325-328
- 64. Disler DG, McCauley TR, Wirth CR et al (1995) Detection of knee hyaline cartilage defects using fat-suppressed three-dimensional spoiled gradient-echo MR imaging: comparison with standard MR imaging and correlation with arthroscopy. Am J Roentgoenol 165:377-382
- 65. Gagliardi JA, Chung EM, Chandnani VP et al (1994) Detection and staging of chondromalacia patellae: relative efficacies of conventional MR imaging, MR arthrography, and CT arthrography. Am J Roentgoenol 163:629-636
- Recht MP, Kramer J, Marcelis S et al (1993) Abnormalities of articular cartilage in the knee: analysis of available MR techniques. Radiology 187:473-478
- 67. Sonin AH, Pensy RA, Mulligan ME et al (2002) Grading articular cartilage of the knee using fast spin-echo proton density-weighted MR imaging without fat suppression. Am J Roentgoenol 179:1159-1166
- 68. Kramer J, Recht MP, Imhof H et al (1994) Postcontrast MR arthrography in assessment of cartilage lesions. J Comput Assist Tomogr 18:218-224
- 69. Turner DA (2000) Subchondral bone marrow edema in degenerative chondrosis [Letter]. Am J Roentgoenol 175:1749-1750
- 70. De Smet AA, Norris MA, Yandow DR et al (1993) MR diagnosis of meniscal tears of the knee: importance of high signal in the meniscus that extends to the surface. Am J Roentgoenol 161:101-107
- 71. Kaplan PA, Nelson NL, Garvin KL et al (1991) MR of the knee: the significance of high signal in the meniscus that does not clearly extend to the surface. Am J Roentgoenol 156:333-336
- 72. Lim PS, Schweitzer ME, Bhatia M et al (1999) Repeat tear of postoperative meniscus: potential MR imaging signs. Radiology 210:183-188
- 73. Farley TÉ, Howell SM, Love KF et al (1991) Meniscal tears: MR and arthrographic findings after arthroscopic repair. Radiology 180:517-522
- 74. Sciulli RL, Boutin RD, Brown RR et al (1999) Evaluation of the postoperative meniscus of the knee: a study comparing conventional arthrography, conventional MR imaging, MR arthrography with iodinated contrast material, and MR arthrography with gadolinium-based contrast material. Skeletal Radiol 28:508-514
- 75. Applegate GR, Flannigan BD, Tolin BS et al (1993) MR diagnosis of recurrent tears in the knee: value of intraarticular contrast material. Am J Roentgoenol 161:821-825
- 76. Tung GA, Davis LM, Wiggins ME et al (1993) Tears of the anterior cruciate ligament: primary and secondary signs at MR imaging. Radiology 188:661-667
- 77. Schweitzer ME, Tran D, Deely DM, Hume EL (1995) Medial collateral ligament injuries: evaluation of multiple signs,

prevalence and location of associated bone bruises, and assessment with MR imaging. Radiology 194:825-829

- Vahey TN, Broome DR, Kayes KJ et al (1991) Acute and chronic tears of the anterior cruciate ligament: differential features at MR imaging. Radiology 181:251-253
- Brandser EA, Riley MA, Berbaum KS et al (1996) MR imaging of anterior cruciate ligament injury: independent value of primary and secondary signs. Am J Roentgoenol 167:121-126
- McIntyre J, Moelleken S, Tirman P (2001) Mucoid degeneration of the anterior cruciate ligament mistaken for ligamentous tears. Skeletal Radiol 30:312-315
- Bergin D, Morrison WB, Carrino JA et al (2004) Anterior cruciate ligament ganglia and mucoid degeneration: coexistence and clinical correlation. Am J Roentgoenol 182:1283-1287
- Nguyen B, Brandser E, Rubin DA (2000) Pains, strains, and fasciculations: lower extremity muscle disorders. Magn Reson Imaging Clin N Am 8:391-408
- Khan KM, Bonar F, Desmond PM et al (1996) Patellar tendinosis (jumper's knee) : findings at histopathologic examination, US, and MR imaging. Radiology 200:821-827
- Shalaby M, Almekinders LC (1999) Patellar tendinitis: the significance of magnetic resonance imaging findings. Am J Sports Med 27:345-349
- Zeiss J, Saddemi SR, Ebraheim NA (1992) MR imaging of the quadriceps tendon: normal layered configuration and its importance in cases of tendon rupture. Am J Roentgoenol 159:1031-1034
- 86. Carotti M, Salaffi F, Manganelli P et al (2002) Power Doppler sonography in the assessment of synovial tissue of the knee joint in rheumatoid arthritis: a preliminary experience. Ann Rheum Dis 61:877-882
- 87. Adam G, Dammer M, Bohndorf K et al (1991) Rheumatoid arthritis of the knee: value of gadopentetate dimeglumine-enhanced MR imaging. Am J Roentgoenol 156:125-129
- Crotty JM, Monu JU, Pope TL Jr (1996) Synovial osteochondromatosis. Radiol Clin North Am 34:327-342
- Lin J, Jacobson JA, Jamadar DA et al (1999) Pigmented villonodular synovitis and related lesions: the spectrum of imaging findings. Am J Roentgoenol 172:191-197
- Hayes CW, Brigido MKI, Jamadar DA, Propeck T (2000) Mechanism-based pattern approach to classification of complex injuries of the knee depicted at MR imaging. Radio Graphics 20:S121-S134
- 91. MuÎler W (1983) The knee. Springer-Verlag, New York
- Amis AA (1991) Functional anatomy of the anterior cruciate ligament: fiber bundle actions related to ligament replacements and injuries. JBJS 73-B:260-267
- 93. Wascher DC, Markolf KL, Shapiro MS, Finerman GA (1993) Direct in vitro measurement of forces in the cruciate ligaments. Part I: the effect of multiplane loading in the intact knee. JBJS 75-A:377-386
- 94. Kaplan PA, Walker CW, Kilcoyne RF et al (1992) Occult fracture patterns of the knee associated with anterior cruciate ligament tears: assessment with MR imaging. Radiology 183:835-838
- Palmer WE, Levine SM, Dupuy DE (1997) Knee and shoulder fractures: association of fracture detection and marrow edema on MR images with mechanism of injury. Radiology 204:395-401
- Recondo JA, Salvador E, Villanus et al (2000) Lateral stabilizing structures of the knee: functional anatomy and injuries assessed with MR imaging. RadioGraphics 200:S91-S102
- Weber WN, Neumann CH, Barakos JA (1991) Lateral tibial rim (Segond) fractures: MR imaging characteristics. Radiology 180:731-734

# Imaging of the Foot and Ankle

Z.S. Rosenberg<sup>1</sup>, M. Zanetti<sup>2</sup>

<sup>1</sup> Department of Radiology, School of Medicine, New York, NY, USA

<sup>2</sup> Department of Radiology, Balgrist University Hospital, Zurich, Switzerland

# Introduction

Foot and ankle pain is a common clinical problem with a wide differential diagnosis. Soft-tissue and osseous pathologic conditions are recognized as significant causes.

Conventional radiography, usually the first imaging technique performed, allows assessment of any potential bone abnormality. In recent years computed tomography (CT) and isotope bone scanning have been superseded by magnetic resonance (MR) imaging for demonstrating most osseous and soft-tissue abnormalities. CT and MR arthrographic techniques produce additional information in the assessment of capsular recesses and cartilage. Ultrasound (US) can be alternatively used for softtissue assessment. The focus of our presentation will be on MR imaging of the foot and ankle, with references, when appropriate, to other imaging modalities, such as CT and US.

# **Traumatic Osseous Abnormalities**

# Occult Fractures, Stress Fractures, Bone Bruises and Stress Reaction

Conventional radiography remains the primary diagnostic method for evaluating bony lesions. However, MR imaging, because of its ability to demonstrate bone-marrow edema, has become a reliable technique for diagnosing occult fractures that are not seen on plain radiographs. Such fractures are also detectable with bone scintigraphy; however, this modality is nonspecific, especially when dealing with the small bones of the feet, and fails to demonstrate anatomic detail. Occult fractures of the foot and ankle occur most frequently in the talus, calcaneus and midfoot bones, and are best assessed on both T1 and T2 or other water-sensitive sequences. When present, a fracture line appears on T1-weighted images as a linear line of low signal intensity traversing the bony trabeculae and extending to a cortical margin. Acute fractures often present with increased signal intensity adjacent to the fracture lines on T2-weighted images, representing bone-marrow edema and hemorrhage.

Fatigue-type stress fractures result from repetitive overloading on normal bone and present more often after several weeks of unaccustomed strenuous training. They are particularly common in athletic individuals. The metatarsals, calcaneus, talus, navicular, medial malleolus and fibula can all be involved. Stress fractures should be distinguished from insufficiency fractures, which occur due to normal stress on weakened bone. The latter are not infrequently noted in the talus and may be confused with transient osteoporosis of the talus. MR images depict a hypointense fracture line or lines that may extend to the cortex and the accompanying bone-marrow edema. Occasionally, early periosteal callous formation can also be identified.

Not infrequently, however, a fracture line will not be easily detected and a fracture may be difficult to distinguish from a bone bruise (microtrabecular disruption without cortical break) or stress reaction (early bone response to stress or abnormal biomechanics). The lack of periosteal reaction in fractures of the hindfoot and tarsal bones makes the diagnosis even more difficult. In those instances, the extent of T1 signal alterations is a useful guideline in differentiating the above three entities. In bone bruises and stress reaction, the T1 signal alterations are subtle or non-existent, while fractures depict more significant signal alterations on both T1 and fluid-sensitive sequences. Metatarsal stress fracture can also be distinguished from stress reaction by the presence of a periosteal reaction, which is not usually seen in stress reaction. Stress reaction particularly related to abnormal biomechanics may be asymptomatic and may involve multiple bones. Isolated periosteal or adjacent soft-tissues edema without T1-weighted changes are other clues to the presence of stress reaction.

Bone bruises are usually associated with acute ankle sprains. They are most commonly seen on the contralateral side of the ankle, in the medial talus, tibia and calcaneus, and are related to an impaction injury. Sprains may also be associated with bone bruises in the talar neck, talar head and navicular, possibly related to talar rotational forces and secondary impaction on the navicular. When the bone bruises are noted in the ipsilateral aspect of the ankle, such as at the origins of the collateral and syndesmotic ligaments, they are most likely related to micro-avulsion injuries. The ipsilateral bone bruises tend to be subtler and smaller in size than the contralateral ones.

Bone bruises usually resolve within 8-12 weeks but may be seen up to a year following the injury. The possibility of chronic instability with repetitive impaction injuries should be raised when the bruises do not resolve quickly. It is recommended that resumption of any sports activity should be delayed for 4-6 weeks due to the potential development of a complete fracture; however, the treatment of bone bruises in the ankle has not yet been fully elucidated.

#### **Osteochondral Talar Lesions**

Osteochondral lesions of the talar dome, formerly called osteochondritis dissecans, osteochondral fractures or talar transchondral fractures, occur in 6.5% of ankle sprains. They develop secondary to impaction injury with laceration of the articular cartilage and fracture of the underlying subchondral microtrabeculae. Talar osteochondral fractures commonly occur in the medial or lateral corners of the dome, although central lesions have been also sporadically described. Medial dome lesions are more common than lateral lesions in most series. The location of the lesion is related to the mechanism of trauma. Medial osteochondral fracture frequently results from inversion injuries with plantar flexion of the foot, which cause impaction of the tibia against the posterior third of the talar dome. Medial cup-shaped lesions are often deeper than lateral wafer-like lesions. Medial osteochondral fragments usually remain in situ, causing fewer symptoms. Lateral osteochondral fractures are related to inversion injuries with dorsiflexion of the foot, causing impaction of the fibula against the middle third of the talar dome. Lateral collateral ligament injury may be associated with both types of fractures, more commonly with the lateral one.

Osteochondral lesions have been classified using arthroscopy and MR imaging into four grades, depending on the integrity of the articular cartilage and the degree of attachment and displacement of the subchondral fragment. Stage I lesions present as intact articular cartilage with signal changes of the subchondral bone. Stage II represents partial detachment of the cartilage and subchondral fragment. Stage III implies a completely detached non-displaced fragment. Stage IV is a detached and displaced osteochondral fragment located away from the crater site. Treatment and prognosis depend to a great extent on the accurate evaluation of the overlying articular cartilage and the stability of the osteochondral fragment. Stages I and II and medial stage III lesions are treated conservatively, while surgical intervention is indicated for lateral stages III and medial and lateral stage IV. Curettage of the lesion and drilling to promote healing as well as cartilage allografts have been utilized in the management of this condition.

Early recognition of osteochondral lesions is of paramount importance, especially in athletic individuals, since premature secondary degenerative arthritis is more prevalent in untreated cases than in the general population. Plain films may depict the osteochondral lesions but are less sensitive than CT and MRI, especially for detecting grade I lesions. Plain films also cannot easily differentiate the grades because of the inability to visualize the overlying articular cartilage. CT is less sensitive than MRI for detecting early lesions and for assessing the articular cartilage. The subchondral bone, however, may be better assessed with CT than with MR imaging.

MR imaging is the optimal modality to assess the presence, size and exact location of the lesion as well as the integrity of the overlying cartilage, the degree of attachment, displacement and viability of the osteochondral fragment, and the location of loose bodies in the joint space. MR diagnosis of fragment stability has relied on the use of water-sensitive sequences but is most reliable with MR arthrography. A low signal intensity rim at the interface between the normal bone and the osteochondral fragment is consistent with healing and stability, while a high signal intensity interface on fat-suppressed post-intra-articular contrast T1-weighted images indicates a completely detached fragment. Partial detachment is inferred when the rim of fluid is not continuous. The presence of cyst underneath an osteochondral lesion indicates instability whether the rim appears complete or not. Perilesional edema is usually seen in the early stages of the disease or may be noted with secondary articular collapse or fracture.

Inaccurate staging of type II-IV osteochondral lesions has been reported to be as high as 50% using conventional MR imaging. This low diagnostic yield is probably secondary to the inability to discriminate between fluid and granulation tissue on T2-weighted images. In these cases, the use of MR arthrography greatly increases the accuracy of MRI. Since osteochondral lesions may be complicated by osteonecrosis, assessment of the fragment viability is also of significance in the MR imaging analysis. Hyperintense signal on T2-weighted or STIR images reflects viable bone marrow, while low signal intensity on all pulse sequences suggests necrosis. Additional evaluation may be obtained using post-intravenous gadolinium fat-suppressed T1-weighted images, which demonstrate enhancement of the marrow in viable bone and the lack of enhancement in necrotic fragments.

# **Sesamoid Dysfunction**

The sesamoid bones of the first metatarsal can undergo a number of pathologic processes, including osteoarthritis, fracture, osteonecrosis and sesamoiditis. Acute fractures and osteonecrosis of the sesamoids are common in athletes and ballet dancers. Osteonecrosis is thought to be traumatic but the exact etiology is not fully known. Sesamoiditis is a painful inflammatory condition secondary to repetitive injury to the plantar soft tissues of the first metatarsal. Conservative treatment of sesamoid disorders with non-weight-bearing and rest is initially attempted. Surgical removal of the sesamoids may be considered in refractory cases.

Disorders of the sesamoids are difficult to diagnose on routine plain radiographs due to overlap of the sesamoids on the first metatarsal head and on each other on the AP view and lateral views, respectively. CT is a useful modality for appreciating fragmentation of the sesamoids but, unlike MRI, is inadequate for assessing marrow abnormalities and soft-tissue disease around the sesamoids.

MR imaging is utilized when the plain films are inconclusive, and can often point to the correct diagnosis. Involvement of two sesamoids is usually reflective of either osteoarthritis or sesamoiditis while a fracture and osteonecrosis are usually noted in a single sesamoid. In general, the medial (tibial) sesamoid is most frequently affected by fractures while the lateral (fibular) sesamoid is more likely to undergo avascular necrosis. Osteoarthritis manifests as subchondral cysts and osteophytosis of the first metatarsal head and both sesamoids without marrow signal alterations. Marrow edema and a fracture line, best noted on sagittal views, are required for establishing the diagnosis of a fracture. Bipartite sesamoid can usually be differentiated from a fracture since in the former entity the marrow signal is normal and the fragments are usually round and have smooth margins (unlike the irregular margins of fracture fragments). Osteonecrosis of a sesamoid is diagnosed when fragmentation and low signal on all pulse sequences are appreciated. However, since the signal characteristics of osteonecrosis can be variable on T2 weighted images and since the osteonecrosis is often advanced by the time it is imaged, it may be difficult to differentiate it from osteoarthritis. Sesamoiditis may depict signal alterations in the sesamoids on fluid-sensitive sequences but the hallmark of the entity is the presence of extensive perisesamoid soft-tissue abnormalities and synovitis.

#### **Tendon Abnormalities**

Tendon abnormalities are important causes of chronic pain in the foot and ankle. They can be grouped into tendinosis, peritendinosis, tenosynovitis, partial tear, rupture, and dislocation. These conditions often coexist and overlap in their clinical, gross, and histologic manifestations and thus can be indistinguishable at imaging. Therefore, the term tendinopathy is used alternatively.

On MR images, tendon assessment is based on changes in morphology, signal intensities and associated findings. Tendinosis is characterized by caliber changes (circumscribed thickening or thinning) and increased signal intensity within the tendon on MR images with short echo time (T1-weighted or proton-density-weighted images). Increased signal abnormality on T2-weighted images are noted when significant intrasubstance degeneration is present. Similar MR findings may also be found in partial tendon tears.

The assessment of signal intensities in ankle tendons is complicated by the magic-angle artifact, which affects tissues with well-ordered collagen fibers, such as tendons, ligaments or hyaline cartilage. The signal increase on MR images with short TEs (under 37ms) depends on the orientation of the fibers and on the main magnetic field  $(B_0)$ . The maximum signal increase is observed at a magic angle of 55° relatively to the orientation of  $B_0$ . In the standard supine body position with neutral position of the foot, a high prevalence (up to 100%) of magic-angle artifact is seen in all ankle tendons, except for the anterior tibial tendon(20%). Magic-angle artifacts in the ankle tendons are almost absent when the patient is scanned in the prone body position with plantar flexion of the foot. This position should be considered for assessment of the ankle tendons

Based on anatomical considerations the ankle tendons can be subdivided into three different groups, each with its own clinical features. The anterior group consists of the anterior tibial tendon, extensor hallucis longus tendon and extensor digitorum longus tendon. The medial group includes the posterior tibial tendon, flexor digitorum longus tendon and flexor hallucis longus tendon. The peroneus brevis and peroneus longus tendons form the lateral group.

#### **Anterior Tibial Tendon**

The anterior tibial tendon is usually exposed only to minor mechanical stress due to its relatively straight course. Consequently, abnormalities are less common than in other tendons. Nevertheless, hypoxic degenerative tendinosis or mucoid degeneration occur and may lead to a partial or complete tear of the anterior tibial tendon. Most tears occur without a trauma. The patients often present with slight foot drop preceded by a long history of swelling and pain at the dorsomedial aspect of the midfoot. Discontinuity of the anterior tibial tendon and occasionally a mass at the anterior ankle are palpable. Whereas a complete tear can usually be easily recognized, the clinical diagnosis of tendinosis or partial tear can be challenging. Characteristic findings of anterior tibial tendinopathy include tendon thickening ( $\geq$ 5mm) and diffuse or posterior signal abnormalities of the tendon within 3 cm from the distal insertion. The significant association of bony irregularities of the underlying tarsal bones with anterior tibial tendon lesions may indicate a mechanical irritation as a pathogenic factor.

#### **Posterior Tibial Tendon**

The posterior tibial tendon is commonly injured in middle-aged women. Failure of the tendon leads to an acquired flatfoot deformity and places an increased load on other structures responsible for maintaining the normal arch of the foot. Therefore, advanced posterior tibial tendon injury has a high association with spring ligament and sinus tarsi abnormalities on MR imaging.

Chronic posterior tibial tendon rupture typically develops in women during the fifth and sixth decades of life. The tear is commonly noted behind the medial malleolus, where the tendon is subjected to a significant amount of friction. Posterior tibial tendon ruptures can be classified into three types. A type I partial tear consists of an incomplete tear with circumscribed enlargement and intrasubstance degeneration. On axial MR images, the diameter of the tendon may be five to ten times that of the adjacent flexor digitorum longus tendon. The normal diameter of the posterior tibial tendon is twice that of the adjacent flexor digitorum longus tendon. Further stretching and elongation of the tendon leads to a type II partial tear of the posterior tibial tendon. On axial images, a decrease in the diameter of the tendon is diagnostic for this pathologic condition. The caliber of the tendon may be equal to or less than that of the adjacent flexor digitorum longus tendon. Complete disruption of the tendon fibers is seen in type III posterior tibial tendon tears. These are quite rare and appear at MR imaging as tendon discontinuity. The gap may be filled with fluid or granulation tissue.

#### **Peroneal Tendons**

Tears of the peroneal tendons are most commonly seen along the lateral malleolus, along the lateral calcaneal wall, and underneath the cuboid tunnel. At the lateral malleolus, tears may be associated with either superior peroneal retinacular tear or laxity secondary to a previous inversion injury.

MR imaging characteristics of peritendinosis and tenosynovitis include scarring around the tendons and fluid within the common tendon sheath, respectively. Care should be taken to differentiate physiologic fluid accumulation within the tendon sheath and tenosynovitis from fluid within the common peroneal sheath secondary to a tear of the calcaneofibular ligament. Care should also be taken as well to differentiate between tendinosis and magic-angle artifact.

Longitudinal intrasubstance tears of the peroneus brevis tendon have a distinct appearance on axial MR images. The tendon assumes a C-shaped or boomerang configuration that partially envelops the peroneus longus tendon.

Isolated tears of the peroneus longus tendon are more frequently seen at the level of the peroneal tubercle or cuboid tunnel. The imaging diagnosis of a tear of the peroneus longus tendon at this location is clinically important, particularly because the patients tend to have nonspecific findings.

An abnormal position of an os peroneum on conventional radiographs may be a clue for possible tear of the peroneus longus tendon. Hypertrophy of the peroneal tubercle has also been implicated as a cause for tear of the peroneus longus tendon at the midfoot. The concept that friction at the tubercle can predispose the peroneus longus tendon to tear is comparable to the pathogenic mechanism in anterior tibial tendon tears (see above).

Clinically acute dislocation of the peroneal tendons is often misdiagnosed as an ankle sprain. A flake-like fracture of the distal fibular metaphysis may be present on conventional radiographs, indicating an avulsed or stripped peroneal retinaculum.

Dislocation is best demonstrated on axial MR images, which show the tendons to be dislocated anterior and lateral to the distal fibula. The tendons are often found within a "pouch" formed by a stripped-off superior peroneal retinaculum. Associated MR imaging findings include tenosynovitis or tears of the peroneal tendons, convex fibular groove, avulsion fracture of the distal fibula, and tear of the lateral collateral ligament. US is advantageous, compared to MRI, in depicting dynamic dislocation of the peroneal tendons.

#### **Achilles Tendon**

Achilles tendon injuries may be classified into non-insertional or insertional categories. The former group includes acute, diffuse and chronic peritendinosis, tendinosis, and a rupture 2-6 cm above the insertion of the tendon on the calcaneus. Insertional Achilles disease includes insertional tendinosis, which may be associated with a Haglund deformity of the calcaneus and insertional tears.

At MR imaging, partial Achilles tendon tears demonstrate heterogeneous signal intensity and thickening of the tendon without complete interruption. Differentiation between partial tear and severe chronic Achilles tendinosis may be difficult even on MR images. Signal changes tend to be along the posterior aspect of the tendon in partial tears and more central in tendinosis. Partial tears, especially when acute, often depict edema and hemorrhage within Kager's fat pad. Complete Achilles tendon rupture manifests as discontinuity with fraying and retraction of the torn edges of the tendon.

US is comparable to MR imaging for assessing Achilles tendon injuries. In addition, US demonstrates neovascularization in painful Achilles tendons. The latter, while strongly associated with pain, is not predictable of an unfavorable outcome. Conversely, tendon inhomogeneity appears to be more relevant with regard to the outcome. Ultrasound may miss Achilles tendon lesions at the muscle- tendon junction, while MR imaging at this site is associated with muscle edema, retraction of muscle fibers and hematoma.

Post-operative MR imaging assessment includes evaluation of the extent of tendinous union and healing. On most follow-up MR imaging studies, intratendinous signal intensity will decrease as the tendon heals. However, the tendon may remain thickened, simulating chronic tendinosis, even after normal signal intensity has been regained.

# Ligaments

#### Lateral Collateral Ligaments

Ankle sprains are the most common musculoskeletal cause for hospital emergency rooms and private practice visits. MR imaging is valuable in the diagnosis of acute injuries of ankle ligaments. The anterior tibiofibular ligament is almost always involved after ankle sprain., and tears are easily diagnosed on axial images at the tip of the fibula. The posterior tibiofibular ligament, also visible on axial images at the tip of the fibula, is rarely torn.

Early investigations have indicated that the calcaneofibular ligament runs obliquely from the fibular tip posteriorly to the lateral surface of the calcaneus. Therefore, oblique images have been considered to be superior in the assessment of the ligament. In our experience, however, this plane is not clearly superior to coronal images in delineating the calcaneofibular ligament. This may be explained by the highly variable course of the ligament (13 - 74°), demonstrated in an anatomic investigation.

Bone bruises are commonly seen, particularly on the contralateral aspect of the talus following inversion injury. This is discussed more fully under the category of osseous injuries.

#### **Tibiofibular Syndesmosis**

The tibiofibular syndesmosis is an important stabilizer of the distal tibiofibular joint. It consists of the anteroinferior tibiofibular ligament, the posteroinferior tibiofibular ligament, the transverse tibiofibular ligament, and the interosseous tibiofibular ligament. Although there have been several reports on the MR diagnosis of distal tibiofibular syndesmosis injuries – with accuracies of up to 100% – the exact criteria and clinical value of diagnosing disruption of the ligaments have not yet been established. Disruption or irregularities of the ligaments, degenerative changes at the distal tibiofibular joint, and proximal extension of fluid into the lateral gutter (greater than 1 cm) aid in making the diagnosis.

#### **Medial Collateral Ligament**

The medial collateral ligament plays an important role in medial ankle instability. Marked inter-individual differences are found for the main components (tibionavicular, tibiospring, tibiocalcaneal, deep posterior and anterior tibiotalar and superficial posterior tibiotalar bands). The tibionavicular ligament is a thickened fibrous layer of the ankle capsule. The tibiocalcaneal and tibiospring ligaments are the longest, while the tibiocalcaneal and posterior deep tibiotalar ligaments are the thickest.

#### Spring Ligament

The spring ligament plays an important role in the stabilization of the longitudinal arch in the midfoot. Tear of the ligament will allow the talar head to collapse resulting in an acquired flat foot deformity. There is a high association between rupture of the spring ligament and dysfunction of the posterior tibial tendon.

The spring ligament is composed of the inferior longitudinal calcaneonavicular and superomedial calcaneonavicular ligaments. Recently, a third component (medial oblique) of the spring ligament has been demonstrated. This component runs from the notch between the anterior and middle calcaneal facets to the tubercle of the navicular in the lower layer of the spring ligament complex, lying beneath the cartilaginous surface of the complex. Each of the three components can be visualized on MR images.

#### **Impingement Syndromes**

The role of impingement syndromes in producing chronic ankle pain has been better appreciated in recent years. Impingement syndromes are usually related to intra-articular osseous or soft-tissue proliferation, which, depending on location, produces pain and limitation of ankle motion.

Among the four most common impingement syndromes in the ankle (anterolateral, anterior, anteromedial, and posterior impingement), anterolateral impingement syndrome has received the most attention in the radiology literature. Intra-articular synovial hypertrophy and fibrosis may occur in the lateral gutter secondary to capsular or ligamentous tears associated with inversion injuries. This condition is optimally assessed with MR arthrography, although positive experience with this approach is so far based only on a few cases.

#### **Entrapment Neuropathies**

The most common entrapment neuropathy in the foot and ankle, excluding Morton's neuroma, is tarsal tunnel syndrome. The tarsal tunnel is a fibro-osseous space formed by the talar surface, the sustentaculum tali, and the calcaneal wall laterally and by the the flexor retinaculum medially. It is traversed by the posterior tibial, flexor digitorum longus, and flexor hallucis longus tendons, the tibial nerve and its branches and accompanying vessels. In about 50% of cases, tarsal tunnel syndrome is idiopathic, whereas in the other 50% a specific cause is identified, such as space occupying lesions including ganglion, varicosities, lipoma, accessory muscles, and nervesheath tumors, as well as pronation or hindfoot valgus deformity, and fracture of the medial malleolus and calcaneus. Patients present with a positive Tinel's sign and pain, burning and numbness along the plantar surface of the foot, usually in the toes or beneath the metatarsal heads. These symptoms sometimes extend to the heel. Entrapment neuropathies of branches of the tibial nerve include entrapment of the first branch of the lateral plantar nerve (Baxter's nerve), which presents with heel pain and is most commonly seen in runners. This entrapment is often associated with hypertrophy of the abductor hallucis muscle but is also produced by inflammation associated with plantar fasciitis and heel spur. This entity should be distinguished from plantar fasciitis. Another cause of heel pain, often seen in joggers and long-distance runners is jogger's foot, which is produced by entrapment of the medial plantar nerve secondary to valgus heel deformity.

Treatment of the entrapment neuropathies is initially conservative but may require surgical release of the nerve and removal of the offending structure. US and MR imaging are the imaging modalities of choice for diagnosing extrinsic mechanical compression of the tibial nerve and its branches. MR imaging has the advantage of depicting associated denervation muscle edema and atrophy.

# Morton's Neuroma

Morton's neuroma is a benign non-neoplastic abnormality characterized by neural degeneration and perineural fibrosis. The plantar interdigital nerves of the second and third intermetatarsal spaces are most commonly involved. The clinical localization of a Morton's neuroma may be equivocal.

MR imaging has shown high sensitivity (87%) and specificity (100%) for the demonstration of Morton's neuroma and is therefore considered to be useful for narrowing the differential diagnosis of forefoot pain. US has demonstrated comparably good results. Morton's neuroma is of low signal intensity on T2-weighted MR images due to the high content of fibrous tissue. The use of intravenous gadolinium contrast is not recommended because only 50% of these lesions enhance. MR imaging permits the exact location of Morton's neuroma, allowing for precise pre-surgical planning. The impact of MR imaging on diagnostic considerations and therapeutic decisions by orthopedic surgeons treating patients with clinically suspected Morton's neuroma is substantial. An MR effectiveness study has demonstrated that the clinical diagnosis of Morton's neuroma is altered in more than one fourth of cases following MR imaging, and a change in location or number occurs in one-third of the remaining cases. These changes in diagnosis and location prompt a change in the treatment plan in more than 50% of the feet.

Large Morton's neuromas (> 5 mm-diameter) are more commonly symptomatic than smaller ones. Post-operative outcome depends on the size as measured by MR imaging. Morton's neuroma larger than 5 mm has a better post-surgical prognosis than a smaller one.

The transverse diameter of a Morton's neuroma on transverse MR images is dependent on the patients body position during imaging. When the patient is in the prone position and the foot is plantar-flexed, Morton's neuroma increases in size and appears 2 mm wider than with patient in the supine position with the foot dorsiflexed. This decrease in diameter is most likely caused by a change in the location of the neuroma, which, as it becomes dislocated more dorsally, is squeezed between the metatarsals.

#### Sinus Tarsi Syndrome

Sinus tarsi syndrome most commonly develops after an inversion injury (70%) and is often associated with tears of the lateral collateral ligaments. It is also noted in patients with rheumatologic disorders or abnormal biomechanics, such as pes planes deformity and posterior tibial tendon dysfunction.

The sinus tarsi is a lateral space located between the talus and the calcaneus. It contains the cervical and the roots of the inferior extensor retinaculum, neurovascular structures, and fat. More medially, the sinus tarsi is continuous with the tarsal canal in which the interosseous talocalcaneal ligaments traverse. Although the latter are not truly lateral structures, they are important in the overall function of the lateral ankle and hind-foot complex.

Patients with sinus tarsi syndrome present with hindfoot instability and pain along the lateral aspect of the foot. Obliteration of the fat in the sinus tarsi space with fluid replacement or fibrous scarring, synovial proliferation and disruption of the interosseous ligaments are MR findings in sinus tarsi syndrome. Associated MR manifestations include edema, sclerosis and cystic changes in the roof of the sinus tarsi, cystic changes at the critical angle of Gissane, lateral collateral ligament tears, and, occasionally, posterior tibial tendon tear. Osteoarthritis of the subtalar joint and subchondral cysts may be present in advanced cases.

Fluid in the sinus tarsi should be differentiated from the normal extension of fluid from posterior subtalar joint recesses.

# **Plantar Fasciitis**

Plantar fasciitis is one of the more common overuse injuries in running sports. The plantar fascia is a multilayered fibrous aponeurosis that extends from the posteromedial calcaneal tuberosity to the plantar plates of the metatarsophalangeal joints, the flexor tendon sheaths, and the bases of the proximal phalanges of the digits. When the metatarsophalangeal joints are dorsiflexed during gait, the windlass mechanism of the plantar fascia tightens and causes repetitive traction on the calcaneal tuberosity. Over time, with repetitive stress, microtears can occur at the origin of the plantar fascia, which result in a secondary reparative inflammatory response. A similar process occurs at the attachment of the flexor digitorum brevis and abductor digiti minimi muscles directly beneath the plantar fascia, which account, at least in part, for the calcaneal spurs often seen at or close to the origin of the plantar fascia. Progression of the inflammatory

process leads to periostitis or even fatigue fractures of the medial calcaneal tuberosity and/or calcaneal spur.

Plantar fasciitis is a clinical diagnosis that does not require imaging studies. However, MR imaging and US are helpful in cases that are refractory to treatment and when fascial rupture is suspected. On sagittal and coronal MR images, the normal plantar fascia is identified as a thin hypointense structure extending anteriorly from the medial calcaneal tuberosity. The diagnostic features of plantar fasciitis include superficial or deep perifascial edema, heterogeneity and fusiform thickening of the fascia at its insertion, and calcaneal marrow edema. Discontinuity of the fibers of the plantar fascia represents rupture.

Rupture of the plantar fascia is often seen secondary to corticosteroid injections for plantar fasciitis. The rupture may develop a considerable time after the injection and usually occurs distal to the calcaneal origin. Acute traumatic rupture has been noted in athletes, particularly runners, and often relates to jamming the foot in a pot hole.

# **Plantar Plate and Turf Toe**

The plantar plate is a strong, fibrocartilaginous structure which, along with the plantar fascia, accessory collateral ligaments, phalangeal collateral ligaments, and flexor and extensor tendons, reinforces the capsule and provides stability to the metatarsophalangeal joint. The plate originates from the plantar surface of the metatarsal head and inserts onto the plantar base of the proximal phalanx. Further support at the first metatarsal joint is provided by the tendons of the abductor hallucis and adductor hallucis muscles. Plantar plate rupture can be acute or chronic. Progressive degeneration and rupture of the plantar plate of the lesser metatarsals are most frequent in the second metatarsophalangeal joint. These processes are common in women, most likely related to the increased weight bearing and hyperextension forces produced by high-heeled, pointed shoes. Progressive degeneration is associated with metatarsalgia, joint instability, and hammer toe, claw toe and crossover toe deformities.

Turf toe refers to a more acute plantar plate injury at the first metatarsophalangeal joint. Injuries to the other capsuloligamentous structures of the first metatarsophalangeal joints are also included in the definition of turf toe. The entity commonly occurs in football players who sustain a hyperextension injury when playing on hard artificial surfaces. Stress injuries in varus, valgus and hyperflexion (least common) are other etiologies for turf toe, which produces significant functional disability and impaired push-off. Grade I injury is a strain of the plantar plate, while grade II injury reflects an avulsion of the plate of the bone. In grade III injury, an impaction to the dorsal surface of the metatarsal head with or without an avulsion or chip fracture is present.

Tear of the plantar plate often occurs at the stronger distal insertion of the plate and is frequently associated with chronic metatarsophalangeal synovitis. The tears can be found laterally in association with a phalangeal collateral ligament tear but medial and proximal tears can also occur.

Optimal MR imaging of the plantar plate is performed utilizing a small field of view in the axial (long axis views, parallel to the plantar surface of the foot), coronal (short axis, perpendicular to the plantar surface of the foot) and sagittal planes. 3D image acquisition technique and small receiver coils are also useful for optimal visualization of the plate. The plantar plate is low in signal and may be difficult to distinguish from the more superficial flexor tendon. Cartilage undercutting at the distal attachment of the plate, best seen on sagittal images, should not be misinterpreted as a tear. Degeneration and rupture of the plate manifest as heterogeneity and indistinctness of the plate. Hyperextension at the joint, capsular distension, synovitis, intermetatarsal bursitis, and plantar soft-tissue edema are frequently associated with MR evidence of plate injury.

Plantar plate injury should be considered when imaging patients with metatarsalgia. One should keep in mind, however, that a long differential diagnosis of metatarsalgia exists, including entities such as bone bruise, stress fracture, degenerative and inflammatory arthritis, Morton's neuroma and Freiberg's infarction.

# Collateral Ligaments of the First Metatarsophalangeal Joint

Stability of the first metatarsophalangeal joint is crucial for proper gait and normal weight-bearing of the foot. The great toe supports more than twice the load of the lesser toes. This load can rise up to almost eight times body weight during strenuous athletic activities. Stability of the joint is provided by the plantar plate, capsule, sesamoids, medial and lateral collateral ligaments, tendons of the abductor and adductor hallucis, and short and long extensor and flexor tendons.

Tears of the medial collateral ligament are related to repetitive valgus and external rotation of the hallux relative to the first metatarsal. With the progression of hallux valgus, there is increased stress on the medial stabilizing structures of the joint, loss of the medial lever arm, and progressive insufficiency of the abductor hallucis longus tendon. Ligamentous degeneration and rupture can then occur. Tears of the medial collateral ligament may be associated with medial capsule and sesamoid ligament injuries.

The medial and lateral collateral ligaments have a narrow origin on the medial or lateral borders of the metatarsal head and fan out to insert on the border of the proximal phalanx and plantar plate. On MR imaging, these ligaments are depicted as low-signal-intensity linear structures best seen on axial images of the forefoot. Discontinuity of the ligaments is appreciated in the acute phase of the injury while thickening and irregularity may be noted when healing is initiated.

# **Suggested Reading**

- Abreu MR, Chung CB, Mendes L, Mohana-Borges A, Trudell D, Resnick D (2003) Plantar calcaneal enthesophytes: new observations regarding sites of origin based on radiographic, MR imaging, anatomic, and paleopathologic analysis. Skeletal Radiol 32(1):13-21
- Ashman CJ, Klecker RJ, Yu JS (2001) Forefoot pain involving the metatarsal region: differential diagnosis with MR imaging. Radiographics 21(6):1425-1440
- Balen PF, Helms CA (2001) Association of posterior tibial tendon injury with spring ligament injury, sinus tarsi abnormality, and plantar fasciitis on MR imaging. Am J Roentgenol 176:1137-1143

Beltran J (1994) Sinus tarsi syndrome. MRI Clin North Am 2:59-65

- Bencardino, J MD, Rosenberg, ZS MD, Delfault, E (1999) MR imaging in sports injuries of the foot and ankle. MR Clin North Am 7(1):131-148
- Berkowitz JF, Kier R, Rudicel S (1991) Plantar fasciitis: MR imaging. Radiology 179:665-667
- Biasca N, Zanetti M, Zollinger H (1999) Outcomes after partial neurectomy of Morton's neuroma related to preoperative case histories, clinical findings, and findings on magnetic resonance imaging scans. Foot Ankle Int 20:568-575
- Boss AP, Hintermann B (2002) Anatomical study of the medial ankle ligament complex. Foot Ankle Int 23:547-553
- Breitenseher MJ, Jaller J, Kukla C et al (1997) MRI of the sinus tarsi in acute ankle sprain injuries. J Comput Assist Tomogr 21:274-279
- Brown KW, Morrison WB, Schweitzer ME, Parellada JA, Nothnagel H (2004) MRI findings associated with distal tibiofibular syndesmosis injury. Am J Roentgenol 182(1):131-136
- Bui-Mansfiedel LT, Kline M, Chew FS, Rogers F, Lenchik L (2000) Osteochondritis dissecans of the tibial plafond: imaging characteristics and a review of the literature. Am J Roentgenol 175:1305-1308
- Chandnani VP, Harper MT, Ficke JR, Gagliardi JA, Rolling L et al (1994) Chronic ankleinstability: Evaluation with MR Arthrography. MR imaging and stress radiography. Radiology 192:189-194
- Cheung YY, Rosenberg ZS, Ramsinghani R, Beltran J, Jahss MH (1997) Peroneus quartus muscle: MR imaging features. Radiology 202:745-750
- Chowchuen P, Resnick D (1998) Stress fracture of the metatarsal heads. Skeletal Radiol 27:22-25
- De Smet AA, Fisher DR, Burnstein MI, Graf BK, Lange RH (1990) Value of MR imaging in staging osteochondral lesions of the talus (osteochondritis dissecans): Results in 14 patients. Am J Roentgenol 154:555-58
- Dunfee WR, Dalinka MK, Kneeland JB (2002) Imaging of athletic injuries to the ankle and foot. Radiol Clin N Am 40:289-312
- Fiorella D, Helms CA, Nunley JA (1999) The MR imaging features of the posteriorintermalleolar ligament in patients with posterior impingement syndrome in the ankle. Skeletal Radiol 28:573-576
- Frey C, Feder KS, DiGiovanni C (1999) Arthroscopic evaluation of the subtalar joint: does sinus tarsi syndrome exist?. Foot Ankle Int 20:185-191
- Guhl JF, Stone JW (1993) Osteochondritis dissecans. In: Guhl JF (ed): Foot and ankle arthroscopy. 2nd edn. Thorafare, Slack, pp 107-110
- Higashiyama I, Kumai T, Takakura Y, Tamail S (2000) Follow-up study of MRI for osteochondral lesion of the talus. Foot Ankle Intl 21(2):127-133
- Ho VW, Peterfy C, Helms CA (1993) Tarsal tunnel syndrome caused by strain of an anomalous muscle: an MRI-specific diagnosis. J Comput Assist Tomogr 17(5):822-823
- Kaminsky S, Griffin L, Milsap J, Page D (1997) Is ultrasonography a reliable way to confirm the diagnosis of Morton's neuroma? Orthopedics 20:37-39

- Karasick D, Schweitzer ME (1998) Disorders of the hallux sesamoid complex: MR features. Skeletal Radiol 27:411-418
- Kier R (1994) Magnetic resonance imaging of plantar fasciitis and other causes of heel pain. MRI Clin North Am 2:97-107
- Kinoshita M, Okuda R, Morikawa J, Abe M (2003) Tarsal tunnel syndrome associated with an accessory muscle. Foot Ankle Intl 24(2):132-136
- Kim DH, Ryu S, Tiel RL, Kline DG (2003) Surgical management and results of 135 tibial nerve lesions at the Louisiana State University Health Sciences Center. Neurosurgery. 53(5):1114-1124 (Discussion 1124-1125)
- Kolker D, Murray M, Wilson M (2004) Osteochondral defects of the talus treated with autologous bone grafting. J Bone Joint Surg 86(4):521-526
- Kramer J, Stiglbauer R, Engel A, Prayer L, Imhof H (1992) MR contrast arthrography (MRA) in osteochondrosis dissecans. J Comput Assist Tomogr 16:254-260
- Lee JK, Yao L (1988) Stress fractures: MR imaging. Radiology 169:217-220
- Lektrakul N, Chung CB, Lai Ym, Theodorou DJ, Yu J, Haghighi P, Trudell D, Resnick D (2001) Tarsal sinus: arthrographic, MR imaging, MR arthrographic, and pathologic findings in cadavers and retrospective study data in patients with sinus tarsi syndrome. Radiology 219:802-810
- Magee TH, Hinson GW (1998) Usefulness of MR imaging in the detection of talar dome injuries. Am J Roentgenol 170:1227-1230
- Taylor JA, Sartoris DJ Juang GS et al (1993) Painful conditions affecting the first metatarsal sesamoid bones. RadioGraphics 13:817-830
- Miller TT, Staron RB, Feldman F et al (1995) The symptomatic accessory tarsal navicular bone: assessment with MR imaging. Radiology 195:849-853
- Mohana-Borges AV, Theumann NH, Pfirrmann CW, Chung CB, Resnick DL, Trudell DJ (2003) Lesser metatarsophalangeal joints: standard MR imaging, MR arthrography, and MR bursography – initial results in 48 cadaveric joints. Radiology. 227(1):175-182
- Nishimura G, Yamato M, Togawa M (1996) Trabecular trauma of the talus and medial malleolus concurrent with lateral collateral ligamentous injuries of the ankle: evaluation with MR imaging. Skeletal Radiol 25:49-45
- Oae K, Takao M, Naito K, Uchio Y, Kono T, Ishida J, Ochi M (2003) Injury of the tibiofibular syndesmosis: value of MR imaging for diagnosis. Radiology 227:155-161
- Pinar H, Akseki D, Kovanlikaya I et al (1997)) Bone bruises detected by magnetic resonance imaging following lateral ankle sprains. Knee Surg Sports Traumatol Arthr 5:113-117
- Quinn TJ, Jacobson JA, Craig JG, van Holsbeeck MT (2000) Sonography of Morton's neuromas. Am J Roentgenol 174:1723-1728
- Rask MR (1978) Medial plantar neurapraxia (jogger's foot): report of 3 cases. Clin Orthop Jul-Aug(134):193-195
- Redd RA, Peters VJ, Emery AF, Branch HM, Rifkin MD (1989) Morton neuroma: sonographic evaluation. Radiology 171:415-417
- Robinson P, White LM, Salonen DC, Daniels TR, Ogilvie-Harris D (2001) Anterolateral ankle impingement: MR arthrographic assessment of the anterolateral recess. Radiology 221:186-190
- Rosenberg ZS, Cheung Y, Jahss MH, Noto AM, Norman A, Leeds NE (1988) Rupture of the posterior tibial tendon: CT and MR imaging with surgical correlation. Radiology 69:229-235
- Rosenberg ZS (1997) MR imaging of longitudinal splits of the peroneus brevis tendon. Am J Roentgenol 168:141-147
- Rosenberg ZS, Beltran J, Bencardino JT (2000) MR imaging of the ankle and foot. Radiographics 20:S153-179
- Schmidt HM, Grunwald E (1981) [Ligament systems of talocrural and intertarsal joints in man]. Gegenbaurs Morphol Jahrb 127:792-831

- Schneck CD, Mesgarzadeh M, Bonakdarpour A (1992) MR imaging of the most commonly injured ankle ligaments. Part II. Ligament injuries. Radiology 184:507-512
- Schuman L, Struijs PA, van Dijk CN (2002) Arthroscopic treatment for osteochondral defects of the talus. Results at followup at 2 to 11 years. J Bone Joint Surg 84(3):364-368
- Schweitzer ME, White LM (1996) Does altered biomechanics cause marrow edema? Radiology 198:851-853

Schweitzer ME. Haims AH (2001) Morrison WB. MR imaging of ankle marrow. Foot Ankle Clin 5(1):63-82, 2000219(3):802-10

- Schweitzer ME, van Leersum M, Ehrlich SS, Wapner K (1994) Fluid in normal and abnormal ankle joints: amount and distribution as seen on MR images. Am J Roentgenol 162:111-114
- Spitz DJ. Newberg AH (2002) Imaging of stress fractures in the athlete. Radiol Clin N Am 40(2):313-331
- Stroud CC. Marks RM (2000) Imaging of osteochondral lesions of the talus. Foot Ankle Clin 5(1):119-133
- Taniguchi A, Tanaka Y, Takakura Y, Kadono K, Maeda M, Yamamoto H (2003) Anatomy of the spring ligament. J Bone Joint Surg Am 85-A:2174-2178
- Taylor JA, Sartoris DJ Juang GS et al (1993) Painful conditions affecting the first sesamoid bone. Radiographics 13:817-830
- Theodorou DJ, Theodorou SJ, Farooki S et al (2001) Disorders of the plantar aponeurosis: a spectrum of MR imaging findings. Am J Roentgenol 176:97-104
- Theodorou DJ, Theodorou SJ, Kakitsubata Y, Lektrakul N, Gold GE, Roger B, Resnick D (2000) Plantar fasciitis and fascial rupture: MR imaging findings in 26 patients supplemented with anatomic data in cadavers. Multicenter Study. Validation Studies] Radiographics. 20:S181-S197
- Theodorou DJ, Theodorou SJ, Farooki S, Kakitsubata Y, Resnick D (2001) Disorders of the plantar aponeurosis: a spectrum of MR imaging findings. Am J Roentgenol 176(1):97-104
- Theumann NH, Pfirrmann CW, Mohana Borges AV, Trudell DJ, Resnick D (2002) Metatarsophalangeal joint of the great toe: normal MR, MR arthrographic, and MR bursographic findings in cadavers. J Comput Assist Tomogr 26(5):829-838
- Umans HR, Elsinger E (2001) The plantar plate of the lesser metatarsophalangeal joints: potential for injury and role of MR imaging. MRI Clin North Am 9(3):659-669

- Weishaupt D, Treiber K, Kundert HP, Zollinger H, Vienne P, Hodler J, Willmann JK, Marincek B, Zanetti M (2003) Morton neuroma: MR imaging in prone, supine, and upright weight-bearing body positions. Radiology 226:849-856
- Yao L, Johnson C, Gentili A et al (1998) Stress injuries of bone: analysis of MR imaging staging criteria. Acad Radiol 5:34-40
- Yao L, Cracchiolo A, Farahani K, Seeger LL (1996) Magnetic resonance imaging of plantar plate rupture. Foot Ankle Intl 17(1):33-36
- Yu JS (2000) Pathologic and post-operative conditions of the plantar fascia: review of MR imaging appearances. Skeletal Radiol 29(9):491-501
- Yu JS, Smith G, Ashman C, Kaeding C (1999) The plantar fasciotomy: MR imaging findings in asymptomatic volunteers. Skeletal Radiol 28(8):447-452
- Zanetti M, De Simoni C, Wetz H, Zollinger H, Hodler J (1997) Magnetic resonance imaging of injuries to the ankle joint: Can it predict clinical outcome? Skeletal Radiol 26:82-88
- Zanetti M, Ledermann T, Zollinger H, Hodler J (1997) Efficacy of MR imaging in patients suspected of having Morton's neuroma. Am J Roentgenol 168:529-532
- Zanetti M, Strehle JK, Kundert H-P, Zollinger H, Hodler J (1999) MR imaging for suspected Morton's neuroma: effect on diagnostic thinking and therapeutic decisions. Radiology 213:583-588
- Zanetti M, Metzdorf A, Kundert HP, Zollinger H, Vienne P, Seifert B, Hodler J (2003) Achilles tendons: clinical relevance of neovascularization diagnosed with power Doppler US. Radiology 227:556-560
- Zeiss J, Fenton P, Ebraheim N, Coombs RJ (1991) Magnetic resonance imaging for ineffectual tarsal tunnel surgical treatment. Clin Orthop 264-266
- Zeiss J, Fenton P, Ebraheim N, Coombs RJ (1991) Magnetic resonance imaging for ineffectual tarsal tunnel surgical treatment. Clin Orthop Rel Res (264):264-266
- Zeiss J, Fenton P, Ebraheim N, Coombs RJ (1990) Normal magnetic resonance anatomy of the tarsal tunnel. Foot Ankle 10(4):214-218
- Zeiss J, Ebraheim N, Rusin J (1990) Magnetic resonance imaging in the diagnosis of tarsal tunnel syndrome. Case report. Clin Imag 14(2):123-126

# **Magnetic Resonance Imaging of Muscle**

M.N. Pathria<sup>1</sup>, R.D. Boutin<sup>2</sup>

<sup>1</sup> UCSD Medical Center, San Diego, CA, USA

<sup>2</sup> Med-Tel International, Davis, CA, USA

# **Learning Objectives**

At the end of this article, readers should:

- Be able to Identify the normal imaging features of skeletal muscle on magnetic resonance (MR) imaging.
- Recognize that some muscle abnormalities do not produce signal alterations.
- Recognize the common patterns of inflammation of muscle.
- Know the common classification system used for muscle injury.
- Understand the evolution of hemorrhage in the muscle tissues.

# **Normal Muscle**

The MR appearance of normal skeletal muscle is the result of an organized admixture of muscle fibers and

fat. Normal skeletal muscle shows a "striated" and "feathery" appearance, produced by high-signal-intensity fat interlaced within and between the major muscle bundles. Normal muscle has low signal intensity on all sequences, and decreases in signal intensity with T2-weighting (Fig. 1). The exterior surfaces of the muscle is smooth and typically shows a mild convexity. An important anatomic region of the muscle is its myotendinous junction, where the muscle fibers interdigitate with the tendon. The myotendinous junction is located at a variable distance from the site of tendon insertion.

Accessory muscles are congenital abnormalities in which an anomalous muscle is present. The accessory muscle may be asymptomatic, or it may present as a palpable mass or may affect adjacent structures, particularly if there is nerve compression. The best-known accessory muscle is the accessory soleus, seen in the pre-Achilles



Fig. 1. Normal muscle. A coronal T1weighted (a) and T2weighted fat-suppressed (b) image of the right hip demonstrates the appearance of normal muscle. This young male patient has minimal intramuscular fat. Note the high-signalintensity fat seen in the intermuscular planes. The outer surfaces of the muscle are smooth and slightly convex. The signal of normal muscle decreases on the T2-weighted image



**Fig. 2.** Accessory soleus. This middle-aged man palpated a mass above his ankle. A sagittal T1-weighted magnetic resonance (MR) image of the ankle shows an anomalous low-lying soleus muscle anterior to the Achilles tendon and filling the pre-Achilles fat pad

fat pad (Fig. 2). This muscle can be very large and is usually felt by the patient. Other common accessory muscles include the peroneus quartus muscle, behind the fibula, the accessory abductor digiti minimi, in the wrist, and the anomalous lumbrical muscle, seen in carpal tunnel.

# **Congenital Muscular Disorders**

There are numerous forms of muscular dystrophy, with Duchenne and Becker muscular dystrophy being the most common. These typically present with progressive proximal muscle weakness in childhood or adolescence. The congenital myopathies all involve multiple muscle groups, typically in a symmetrical fashion. In the acute phase of muscle damage, symmetrical mild hyperintensity of the muscles can be seen (Fig. 3). Unlike inflammatory myopathies, the subcutaneous tissues remain normal. More advanced disease typically shows pseudohypertrophy of lower extremity muscles, particularly the calf musculature, due to excessive fatty infiltration. Ultimately, fatty atrophy of the muscle develops.

# Denervation

Acutely denervated muscle shows a paucity of findings on MR imaging. MR signal alterations are usually seen several weeks following loss of neural innervation. In subacute denervation, the denervated muscle have high signal intensity on T2-weighted and inversion recovery sequences. Typically, the size of the muscle remains normal or is slightly diminished due to concomitant atrophy (Fig. 4). In chronic denervation, the muscle edema resolves, and the involved muscles undergo volume loss



**Fig. 4.** Peroneal nerve denervation. An axial fat-suppressed proton-density (PD)-weighted image of the proximal calf shows a ganglion adjacent to the proximal tibiofibular joint. Due to compression on the peroneal nerve, denervation changes are apparent in the tibialis anterior muscle



**Fig. 3.** Duchenne muscular dystropy. An axial STIR MR image of the thighs shows symmetrical hyperintensity of the quadriceps muscles

and fatty atrophy. The presence of fatty change implies an irreversible lesion. Clinical history and the distribution of the muscle abnormalities, which correspond to a specific nerve distribution, allow accurate diagnosis of muscle denervation.

# Atrophy

Atrophy of muscle is the result or end stage of many muscle abnormalities, so it is only a finding, not a diagnosis. Chronic disuse, denervation, and myopathies are the most common causes seen in clinical practice. Muscle atrophy manifests as decreased size of the involved muscle. Atrophy is associated with fatty infiltration in most cases.

# **Autoimmune Myopathies**

Polymyositis is the classic autoimmune inflammatory myopathy. When associated with skin changes, the same syndrome is called dermatomyositis. Numerous other forms of autoimmune muscle inflammation are recognized. The autoimmune muscle disorders produce symmetrical muscle weakness that primarily involves the proximal muscles. On MR, widespread symmetrical muscle inflammation is the most prominent finding. The inflamed muscles are edematous but the normal muscle architecture is preserved (Fig. 5).

Autoimmune myositis can also be seen in association with collagen vascular diseases, such as rheumatoid arthritis and systemic lupus erythematosus. Myositis is more often unilateral or asymmetrical in association with collagen vascular disease than it is with idiopathic polymyositis. Nodular forms are also recognized.

# **Pyomyositis**

Deep cellulitis can be associated with septic fasciitis or pyomyositis, an infiltrative deep infection of muscle. Pyomyositis used to be seen most frequently in children and in patients from the tropics. More recently, pyomyositis has been recognized with increasing frequency in adult patients with AIDS. While pain is a constant symptom finding, muscle weakness is unusual.

The most common causative organism is *Staphylococcus aureus*, which is responsible for 90% of cases. Pyomyositis is typically limited to one muscle group. Involvement of the lower extremity, particularly the thigh, predominates. The muscle is typically edematous and partly necrotic. It can be well evaluated with MR imaging. Adjacent soft tissue inflammation may be present, but subcutaneous inflammatory changes are minimal compared to those seen in patients with cellulitis or fasciitis, and are disproportionately less prominent than the muscular abnormalities. The underlying bony cortex and bone marrow are typically not involved.

On T1-weighted MR, findings are minimal except for subcutaneous edema and mild enlargement of the affected muscles due to the increased volume of interstitial fluid and fluid collections. The highly proteinaceous material in the center of an abscess may show intermediate signal intensity on T1-weighted images, either diffusely or peripherally. High signal intensity is seen within the muscle on MR on T2-weighted images. Adjacent soft-tissue inflammation is typically present. Areas of necrosis and abscess formation are typically present and can best be seen following the administration of Gd-DTPA (Fig. 6).



**Fig. 5.** Polymyositis. An axial T2-weighted fat-suppressed image of the calf shows widespread feathery muscle edema and mild subcutaneous edema. Note the preservation of the normal muscle architecture



**Fig. 6.** Pyomyositis. This middle-aged HIV-positive male presented with a swollen painful thigh and fever. The Gd-enhanced MR shows subcutaneous and patchy muscle enhancement, as well as a small focal abscess in the vastus lateralis

# **Muscle Injury**

#### **Fascial Herniation**

Herniation of muscle through an overlying fascial tear is an uncommon injury that presents as a painful mass. The mass can be seen to enlarge or contract with muscle contraction, allowing accurate diagnosis. Muscle herniation through fascial tear is very difficult to see with MR; we prefer to use US for this diagnosis, because the mass can be examined during dynamic muscle contraction. On MR, nonspecific contour irregularity of the muscle surface is the only finding.

#### DOMS

Exercise can be followed by pain, muscle soreness, and muscle swelling, particularly in the deconditioned individual. Muscle pain developing hours or days following exercise has been termed delayed onset muscle soreness (DOMS). Unlike the acute onset of symptoms with muscle strain, the symptoms of DOMS develop gradually 1-2 days following exercise, peak 2-3 days following the activity, and then resolve after approximately 1 week. On T1-weighted images, mild enlargement of the muscle may be present. Increased signal is seen on T2-weighted and STIR images. The muscle architecture remains preserved as the edema parallels the muscle fascicles. Signal changes and clinical symptoms are maximal in the region of the myotendinous junction. While clinical symptoms resolve quickly, the MR signal changes of DOMS can persist for up to 80 days.

#### Laceration and Contusion

A muscle laceration is typically produced by direct trauma, usually a penetrating wound extending into the muscle. Less commonly, muscle can be lacerated by the sharp bone ends of a fracture. The area of the laceration can be seen on MR as a linear defect in the muscle, filled with blood and fluid, but MR is not frequently used to assess muscle laceration. Muscle injuries related to a single episode of severe trauma are subdivided into muscle strain and muscle contusion, depending on the mechanism of injury. A muscle strain is caused by an indirect injury, whereas a contusion is due to direct concussive trauma with a blunt, nonpenetrating object. The muscle alterations of contusion are identical to those seen high-grade muscle strains but the location of the injury is independent of the myotendinous junction, corresponding instead with the site of impact. Contusions are more likely to be associated with extensive hemorrhage within the muscle.

# **Muscle Strain**

Muscle strains typically involve the myotendinous junction of the muscle. The myotendinous junction is vulner-



Fig. 7. Muscle strain. This man injured both legs during a soccer game and developed medial thigh pain immediately after the injury. A PDweighted fat-suppressed coronal image of the thighs shows high signal intensity localized around the myotendinous junction of the adductor muscles bilaterally, consistent with a low-grade strain

able to injury because it is the structurally weakest region in the myotendinous unit due to its limited capacity for energy absorption. Muscle strains are most common in the long fusiform muscles of the thigh or calf.

Strains are subdivided into three grades by orthopedic surgeons. A grade 1 strain demonstrates normal muscle morphology and only mild abnormalities of muscle signal, particularly in the region of the myotendinous junction. In grade 2 strain, there are signal changes and mild alterations in the muscle morphology. The T2-weighted images show irregularity, thinning, and mild waviness of the tendon fibers. Muscle edema and hemorrhage are more prominent, often collecting in the subfascial regions around the injured muscle (Fig. 7) More significant morphologic alterations are present in grade 3 strain, which represents a complete rupture of the myotendinous junction. Large amounts of hemorrhage may be present, obscuring the anatomy. The diagnosis is obvious if the tendon ends are retracted, producing a gap in the soft tissues at the expected position of the myotendinous junction, and allowing the muscle to bunch up away from the region.

#### **Parenchymal Hemorrhage**

Hemorrhage within muscle has two different appearances, depending on the pattern of bleeding. Hemorrhage dissecting within the muscle stroma, not forming a discrete collection, is known as parenchymal hemorrhage. When blood forms a discrete collection, the mass is referred to as a hematoma. Both parenchymal hemorrhage and hematoma coexist in most cases with extensive bleeding. Parenchymal hemorrhage does not have a brain correlate so its appearance is less well-known to radiologists. Unlike a hematoma, parenchymal hemorrhage has little mass effect and has a lacy, feathery appearance within muscle with preservation of fascial planes. Parenchymal hemorrhage is best seen on inversion recovery or T2-weighted sequences, and is often normal appearing on T1-weighted images. The appearance of a subacute parenchymal bleed is very nonspecific as the blood does not undergo a phase of methemoglobin formation, as is seen in hematomas.

#### Hematoma

Soft-tissue hemorrhage can collect as a discrete hematoma. Hematomas can be seen in the muscle, in the intermuscular fat planes, or within the subcutaneous tissues. The MR appearance of hematomas is highly variable depending upon their age. The MR appearance of muscle hematomas follows the same progression as in the brain but the time course may be longer and less predictable. Acute blood has low signal intensity on both T1- and T2-weighted images due to the presence of intracellular deoxyhemoglobin. Subacute hematomas have a distinctive appearance due to the formation of methemoglobin, particularly at the periphery of the hematoma (Fig. 8). Methemoglobin produces T1 shortening, resulting in high signal intensity within the hematoma on T1-weighted images. Fluid-fluid levels within the hematoma are common, particularly in large hematomas. In chronic hematoma, some of the iron in the methemoglobin is converted to hemosiderin and ferritin, which deposit in the hemorrhage and adjacent tissues. These substances result in signal loss on both T1- and T2weighted images, producing a low-signal halo around the hematoma.

#### M.N. Pathria, R.D. Boutin



Fig. 8. Hematoma. A sagittal T1-weighted MR of the hip shows a large heterogeneous mass anterior to the hip joint caused by a large hematoma. Note the high signal intensity at the anterior periphery of the lesion produced by methemoglobin



**Fig. 9.** Myositis ossificans. An axial T2-weighted MR of the thighs of an adolescent male shows periosteal new bone formation around the anterior femoral shaft, and an irregular inhomogeneous mass in the overlying soft tissues. On excision, the mass was found to beimmature myositis ossificans



**Fig. 10.** Compartment syndrome. This woman developed severe pain 4 days after total knee replacement and MR was ordered to evaluate for infection. The MR shows hyperintensity of the deep posterior compartment muscles, but no osseous abnormalities. Compartment pressures were subsequently obtained and confirmed the diagnosis of compartment syndrome

# **Myositis Ossificans**

Myositis ossificans is a circumscribed mass of calcified and ossified granulation tissue that forms as a response to trauma. The early MR appearance is very nonspecific and can easily be mistaken for a neoplasm. Underlying periostitis is typically present with this lesion. (Fig. 9) With maturation, a low-signal-intensity rim, due to peripheral calcification, becomes apparent. Mature myositis ossificans may show a fat signal centrally due to marrow formation or there may be persistent granulationtype tissue within its central regions.

# **Compartment Syndrome**

Acute compartment syndrome is a surgical emergency requiring compartment decompression, and MR is not indicated in most cases. MR is only obtained when symptoms are confusing or unclear. Compartment syndrome is seen most commonly in the lower extremity, typically below the knee, in patients who have undergone injury. However, any location can be involved, including the thigh, forearm and paraspinal musculature. The MR findings are nonspecific, though changes limited to all the muscles in a signal compartment should suggest the diagnosis. Mild unilateral swelling and a slight increase of muscle intensity on T2-weighted images is present (Fig. 10) Peripheral enhancement of the involved compartment may be present. In chronic compartment syndrome, the muscles are very atrophic and often densely fibrotic. Compartment calcification may be present, particularly in the peroneal compartment. MR can show the anatomic extent of the muscle injury and the degree of muscle loss present. Calcific myonecrosis is an unusual condition in which either compartment syndrome progresses to form a chronic cystic cavity. The cavity presents as a fusiform mass filled with liquefied necrotic muscle that is surrounded by a thin shell of calcification.

## Rhabdomyolysis

Infarction of muscle may be due to massive trauma or prolonged immobilization. Many other etiologies, including vascular abnormalities and unstable diabetes, have been reported to produce the syndrome of rhabdomyolysis. The T1-weighted images show minimal abnormality. The involved muscles show mild infiltration of the intramuscular fat planes but there is a paucity of mass effect. Rhabdomyolysis is most conspicuous on T2-weighted images, which show high signal intensity within the abnormal muscle due to a combination of edema, necrosis and hemorrhage. STIR and gadolinium enhancement further enhance the sensitivity of MR.

## **References and Suggested Readings**

http://www.neuro.wustl.edu/neuromuscular/index.html Neuromuscular Disease Center Washington University School of Medicine, St. Louis, MO http://www.smd.kcl.ac.uk/kcsmd/hist/chap3.htm Histopathology of Muscle

- Boutin RD, Fritz RC, Steinbach LS (2002) Imaging of sports-related muscle injuries. Radiol Clin North Am 40(2):333-62
- Evans GF, Haller RG, Wyrick PS, Parkey RW, Fleckenstein JL (1998) Submaximal delayed-onset muscle soreness: Correlations between MR imaging findings and clinical measures. Radiology 208:815-820
- Fleckenstein JL, Watumull D, Conner KE, Ezaki M, Greenlee RG, Bryan WW, Chason DP, Parkey RW, Peshock RM, Purdy PD (1993) Denervated human skeletal muscle: MR imaging evaluation. Radiology 187:213-218

- Gordon BA, Martinez S, Collins AJ (1995) Pyomyositis: characteristics at CT and MR imaging. Radiology 197:279-286)
- Holobinko JN, Damron TA, Scerpella PR, Hojnowski L. Calcific myonecrosis: keys to early recognition. Skeletal Radiol 32(1):35-40, (2003)
- Liu GC, Jong YJ, Chiang CH, Jaw TS (1993) Duchenne muscular dystrophy: MR grading system with functional correlation. Radiology 186:475-480
- Lovitt S, Marden FA, Gundogdu B, Ostrowski ML (2004) MRI in myopathy. Neurol Clin 22(3):509-38
- Mellado JM, Pérez del Palomar L, Díaz L, Ramos A, Saurí A (2004) Long-standing Morel-Lavallée lesions of the trochanteric region and proximal thigh: MRI features in five patients. AJR 182:1289-1294
- Palmer WE, Kuong SJ, Elmadbouh HM (1999) MR imaging of myotendinous strain. AJR 173:703-709)
- Petersilge CA, Pathria MN, Gentili A, Recht MP, Resnick D (1995) Denervation hypertrophy of muscle: MR features. JCAT 19:596-600
- Restrepo CS, Lemos DF, Gordillo H, Odero R, Varghese T, Tiemann W, Rivas FF, Moncada R, Gimenez CR (2004) Imaging findings in musculoskeletal complications of AIDS. Radiographics 24(4):1029-49
- Sallomi D, Janzen DL, Munk PL, Connell DG, Tirman PF (1998) Muscle denervation patterns in upper limb nerve injuries: MR imaging findings and anatomic basis. Am J Roentgenol 171:779-84)
- Shellock FG, Fukunaga T, Mink JH, Edgerton VR (1991) Exertional muscle injury: Evaluation of concentric versus eccentric actions with serial MR imaging. Radiology 179:659-664
- Soler R, Rodriguez E, Aguilera C, Fernandez R (2000) Magnetic resonance imaging of pyomyositis in 43 cases. Eur J Radiol 35(1):59-64
- Swenson SJ, Keller PL, Berquist TH, McLeod RA, Stephens DH (1985) Magnetic resonance imaging of hemorrhage. AJR 145:921-927
- Verleisdonk EJ, van Gils A, van der Werken C. (2001) The diagnostic value of MRI scans for the diagnosis of chronic exertional compartment syndrome of the lower leg. Skeletal Radiol. Jun;30(6):321-5
- Yu JS, Resnick D (1994) MR imaging of the accessory soleus muscle appearance in six patients and a review of the literature. Skeletal Radiology 23(7):525-528
- Zeiss J, Guilliam-Haidet L (1996) MR demonstration of anomalous muscles about the volar aspect of the wrist and forearm. Clinical Imaging 20(3):219-221

# Soft-Tissue Tumors and Tumor-Like Masses: A Systematic Approach to Diagnosis

M.J. Kransdorf<sup>1,2</sup>, M.D. Murphey<sup>2,3,4</sup>

<sup>1</sup> Department of Radiology, Mayo Clinic, Jacksonville, FL, USA

<sup>2</sup> Department of Radiologic Pathology, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington, DC, USA

<sup>3</sup> Department of Radiology and Nuclear Medicine, Uniformed Services University of the Heath Sciences, Bethesda, MD, USA

<sup>4</sup> Department of Radiology, University of Maryland School of Medicine, Baltimore, MD, USA

# Introduction

Current imaging techniques have markedly improved our ability to evaluate soft-tissue tumors. Despite these improved modalities, the ultimate goal of imaging remains unchanged: detecting the suspected lesion and establishing a diagnosis or, more frequently, formulating an appropriate differential diagnosis, and radiologic staging of a lesion. This review is not intended as a summary of the radiologic manifestations of soft-tissue tumors, but will present a systematic approach to evaluation, emphasizing magnetic resonance (MR) criteria in differentiating benign from malignant soft-tissue lesions.

# Incidence

Soft-tissue sarcomas, unlike benign soft-tissue lesions, are relatively uncommon, and are estimated to represent about 1% of all malignant tumors [1, 2]. Hajdu [1] noted that, in the United States, the incidence is about the same as that of multiple myeloma or carcinoma of the thyroid. Soft-tissue sarcomas are two to three times as common as primary malignant bone tumors [2, 3]. Benign soft-tissue tumors are far more common, although it is difficult to estimate the annual incidence because many lipomas, hemangiomas, and other benign lesions are not biopsied. The annual clinical incidence of benign soft-tissue tumors is estimated at 300 per 100,000, and is about 100 times more common than malignant soft-tissue tumors [3, 4].

# **Preliminary Evaluation**

The initial evaluation of a patient with a suspected soft-tissue sarcoma begins with a thorough clinical history and radiologic evaluation. In many circumstances, this will provide key information, which will allow a specific diagnosis even when imaging is nonspecific. Is there a history of a previous tumor or underlying malignancy? Has there been previous surgery or radiation? Is there a painless mass? A painful mass always requires that an inflammatory process be included in the differential diagnosis. Is there a history of notable trauma or anticoagulants? Has the lesion remained stable over a long period of time, varied in size, or is it growing? A history of continued growth is always suspicious for malignancy. Unlike bone tumors, however, a slowly growing soft-tissue mass is not invariably indicative of a benign process. Variation in lesion size with time or activity would be exceedingly unusual for a malignancy, and suggests a process such as a ganglion or hemangioma.

Is there more than one lesion? Primary soft-tissue sarcomas are typically solitary, and the identification of multiple lesions markedly limits the differential diagnosis. Multiple lipomas are seen in 5-15% of patients presenting with a softtissue mass [5-7]. The diagnosis in these cases can be made confidently on the basis of MR signal intensity. Aggressive fibromatosis is multifocal in 10-15% of patients, and a second soft-tissue mass in a patient with a previously confirmed desmoid tumor should be regarded as a second desmoid tumor until proven otherwise [8-10]. Patients with neurofibromatosis have multiple lesions and, although the diagnosis is often known or suspected, this is not always the case. The diagnosis may be suggested on the basis of imaging findings by the identification of multiple lesions in a major nerve distribution. A dominant or enlarging lesion in a patient with neurofibromatosis is suspect for malignant transformation.

#### Radiographs

Despite dramatic technological advances in the ability to image soft-tissue tumors, the radiologic evaluation of a suspected soft-tissue sarcoma must begin with the radiograph. While frequently unrewarding, it is impossible to predetermine those tumors in which radiographs are critical for diagnosis. Radiographs may be diagnostic of a palpable lesion caused by an underlying skeletal deformity (such as exuberant callus related to prior trauma) or exostosis, which may masquerade as a soft-tissue mass. Radiographs may also reveal soft-tissue calcifications, which can be suggestive, and at times very characteristic, of a specific diagnosis.

Radiographs are also the best initial method of assessing coexistent osseous involvement, such as remodeling, periosteal reaction, or overt osseous invasion and destruction [11]. Computed tomography (CT) may be a useful adjunct in specific circumstances, and is best used for those patients in whom radiographs do not adequately depict the lesion, its pattern of mineralization or its relationship to the adjacent osseous structures. This is typically in areas in which the osseous anatomy is complex, such as the pelvis, shoulder, hands and feet, and paraspinal regions.

# **Magnetic Resonance Imaging**

#### Technique

Magnetic resonance imaging (MRI) has emerged as the preferred modality for evaluating soft-tissue lesions. Lesions should be imaged in at least two orthogonal planes, utilizing conventional T1-weighted and T2-weighted spinecho MR pulse sequences in at least one of these. Standard spin-echo images are most useful in establishing a specific diagnosis, when possible, and is the most reproducible technique, and the one most often referenced in the tumor imaging literature. It is the imaging technique with which radiologists are most familiar for tumor evaluation [12]. The main disadvantage of spin-echo imaging remains the relatively long acquisition times, especially for double-echo T2weighted sequences [12]. Radiologists are most familiar with conventional axial anatomy, and we recommend that axial T1- and T2-weighted spin-echo images be obtained in almost all cases. The choice of additional imaging plane or planes varies with the involved body part, the lesion location, and the relation of the lesion to crucial structures. In these additional planes, it is useful to use a combination of conventional T1-weighted spin-echo images, turbo (fast) spin-echo images, gradient images and short tau-inversion recovery (STIR) imaging, as the case requires.

Fast scanning techniques allow for shorter imaging times, decreased motion artifact, increased patient tolerance, and higher patient throughput [12, 13], although they have not replaced standard spin-echo imaging. Gradientecho imaging may be a useful supplement in demonstrating hemosiderin, due to its greater magnetic susceptibility, and susceptibility artifacts related to metallic material, hemorrhage, and air are usually accentuated with this pulse sequence [13]. STIR imaging produces fat suppression and enhances the identification of abnormal tissue with increased water content, and as such, is useful to confirm subtle areas of soft-tissue abnormality [14]. This technique increases lesion conspicuity [14, 15], but typically has a lower signal-to-noise ratio than does spin-echo imaging; it is also more susceptible to degradation by motion [12, 14]. Lesions are generally well seen on standard imaging, and STIR imaging tends to reduce the variations in signal intensities identified on conventional spin-echo MRI, which is most helpful in tissue characterization.

Field-of-view is dictated by the size and location of the lesion. In general, a small field-of-view is preferred; however, it must be large enough to evaluate the lesion and to allow appropriate staging. When an extremity is being evaluated, it is not usually necessary to obtain the contralateral extremity for comparison, unless no lesion is detected on initial sequences. It is useful to place a marker over the area of clinical concern in order to insure it is appropriately imaged. This becomes important in evaluation of lesions such as subcutaneous lipoma or lipomatosis, in which the lesion may not be appreciated as distinct from the adjacent adipose tissue. When small superficial lesions are being evaluated, care should be taken to insure that the marker or patient position does not compress the mass.

#### **Magnetic Resonance Contrast Enhancement**

While there is general agreement on the value of MR in the detection, diagnosis, and staging of soft-tissue tumors and tumor-like lesions, the use of intravenous contrast in their evaluation remains more controversial. In general, MR contrast agents enhance the signal intensity on T1-weighted spin-echo images of many tumors. In some cases it can enhance the demarcation between tumor and muscle and tumor and edema, as well as provide information on tumor vascularity [16,17]; information that is usually well delineated on fluid-sensitive sequences. Dynamic enhancement may also be useful in differentiating benign and malignant lesions by assessing the time-dependent rate of contrast enhancement [18]; however, results using this technique are often not definitive as there are overlapping patterns for benign and malignant processes.

Information on tumor enhancement is not without a price. The use of intravenous contrast substantially increases the length and cost of the examination. Consequently, gadolinium-enhanced imaging should be reserved for those cases in which the results influence patient management. One specific circumstance in which gadolinium-enhanced imaging is useful is in the evaluation of hematomas. In such cases, contrast-enhanced imaging may reveal a small tumor nodule that may have been inapparent within the hemorrhage on conventional MRI [19, 20]. Caution is required, however, in that the fibrovascular tissue in organizing hematomas may show enhancement [21]. Gadolinium-enhanced imaging is probably most useful to differentiate solid from cystic (or necrotic) lesions, or to identify cystic or necrotic areas within solid tumors, since these necrotic or cystic areas show no enhancement [16]. This distinction may be difficult or impossible to make on conventional T2-weighted images, when both tumor and fluid show high signal intensity, well-defined margins, and homogeneous signal intensity, and is particularly important when guiding biopsy to areas that harbor diagnostic tissue.

#### **Magnetic Resonance Diagnosis**

Despite the superiority of MRI in delineating soft-tissue tumors, it remains limited in its ability to precisely characterize them, with most lesions demonstrating prolonged T1 and T2 relaxation times. The majority of lesions remain nonspecific, with a correct histologic diagnosis reached on the basis of imaging studies alone in only approximately 25-35% of cases [22-24]. There are instances, however, in which a specific diagnosis may be made or strongly suspected on the basis of MRI features (Table 1). This is usually done on the basis of lesion signal intensity, pattern of growth, location and associated "signs" and findings. The MRI appearance of these lesions has been well reported and is not reviewed here. More commonly, MRI may reveal a nonspecific appearance. In such cases, it is often not possible to establish a meaningful differential diagnosis or reliably determine whether a lesion is benign or malignant.

#### **Benign versus Malignant**

While there is general agreement on the diagnostic value of MR in many cases, the issue of whether it can reliably distinguish benign from malignant is much less clear. One

 Table 1. Specific diagnoses that may be made or suspected on the basis of magnetic resonance imaging

Vascular lesions	Hemangioma Hemangiomatosis (angiomatosis) Arteriovenous hemangioma (arteriovenous malformation) Lymphangioma Lymphangiomatosis
Bone and cartilage forming lesions	Myositis ossificans Panniculitis ossificans
Fibrous lesions	Elastofibroma Fibrous hamartoma of infancy Musculoaponeurotic fibromatosis Superficial fibromatosis (plantar fibromatosis/Dupuytren contracture)
Lipomatous lesions	Lipoma Lipomatosis Hibernoma Intramuscular lipoma Neural fibrolipoma Lipoblastoma Lipoblastomatosis Liposarcoma Parosteal lipoma
Peripheral nerve lesions	Neurofibroma and schwannoma MPNST (malignant peripheral nerve sheath tumors)
Synovial lesions	Pigmented villonodular synovitis Giant cell tumor of tendon sheath Synovial chondromatosis Synovial cyst Synovial sarcoma
Tumor-like lesions	Aneurysm Abscess Bursitis Calcific myonecrosis Diabetic muscle infarction Ganglion Hematoma Myxoma Pseudoaneurysm

study has suggested that MR can differentiate benign from malignant masses in greater than 90% of cases based on the morphology of the lesion [23]. Criteria used for benign lesions included smooth, well-defined margins, small size, and homogeneous signal intensity, particularly on T2weighted images. Other studies, however, note that malignant lesions may appear as smoothly marginated, homogeneous masses and MR cannot reliably distinguish benign from malignant processes [16, 22, 24]. This discrepancy likely reflects differences within the studied populations.

When the MR images of a lesion are not sufficiently characteristic to suggest a specific diagnosis, a conservative approach is warranted. Malignancies, by virtue of their very nature and potential for autonomous growth, are generally larger and more likely to outgrow their vascular supply, with subsequent infarction and necrosis, presenting as heterogeneous signal intensity on T2-weighted spin-echo MR image. Consequently, the larger a mass is, and the greater its heterogeneity, the greater is the concern for malignancy. Only 5% of benign soft-tissue tumors exceed 5 cm in diameter [25, 26]. In addition, most malignancies are deep lesions, whereas only about 1% of all benign soft-tissue tumors are deep [25, 26] Although these figures are based on surgical, not imaging, series, these trends likely remain valid for radiologists.

When sarcomas are superficial, they generally have a less aggressive biologic behavior than do deep lesions [27]. As a rule, most malignancies grow as deep space-occupying lesions, enlarging in a centripetal fashion [27], pushing, rather than infiltrating adjacent structures (although clearly there are exceptions to this general rule). As sarcomas enlarge, a pseudocapsule of fibrous connective tissue is formed around them by compression and layering of normal tissue, associated inflammatory reaction, and vascularization [27]. Generally, they respect fascial borders and remain within anatomic compartments until late in their course. It is this pattern of growth which gives most sarcomas relatively well-defined margins, in distinction to the general concepts of margins used in the evaluation of osseous tumors.

Increased signal intensity in the skeletal muscle surrounding a musculoskeletal mass on T2-weighted SE MR images or other fluid-sensitive sequences (i.e., STIR) has also been suggested as a reliable indicator of malignancy [28, 29]. These results are based on studies in which both bone and soft-tissue lesions were evaluated. Although this increased signal intensity may be seen with malignancy, in our experience this finding is quite nonspecific. In fact, prominent high signal intensity surrounding a soft-tissue mass more commonly suggests an inflammatory processes, abscesses, myositis ossificans, local trauma, hemorrhage, biopsy or radiation therapy rather than a primary soft-tissue neoplasm.

DeSchepper et al. [30] performed a multivariate statistical analysis of ten imaging parameters, individually and in combination. These researchers found that malignancy was predicted with the highest sensitivity when lesions had a high signal intensity on T2-weighted images, were larger than 33 mm in diameter, and had an heterogeneous

Ages	Hand and Wrist	No (%)	Upper Extremity	No (%)	Axilla and Shoulder	No (%)	Foot and Ankle	No (%)	Lower Extremity	No (%)
	Fibrosarcoma	5(45) <sup>b</sup>	Fibrosarcoma	9(29)	Fibrosarcoma	9(56)	Fibrosarcoma	5(45)	Fibrosarcoma	24(45)
	Angiosarcoma	1(9)	Rhabdomyosarcoma	7(23)	Rhabdomyosarcoma	4(25)	DFSP	2(18)	Rhabdomyosarcoma	8(15)
	Epithelioid sarcoma	1(9)	Angiomatoid MFH	3(10)	Angiomatoid MFH	1(6)	MPNST	2(18)	Giant cell fibroblastoma	5(9)
0-5	Malig GCT tendon sheath	1(9)	DFSP	2(6)	Chondrosarcoma	1(6)	Rhabdomyosarcoma	2(18)	MPNST	5(9)
	DFSP	1(9)	Giant cell fibroblastoma	2(6)	MPNST	1(6)			Angiomatoid MFH	3(6)
	MPNST <sup>d</sup>	1(9)	MPNST	2(6)					DFSP	3(6)
	Rhabdomyosarcoma	1(9)	MFH	2(6)					Angiosarcoma	2(4)
			Other	4(13)					Other	3(6)
	Epithelioid sarcoma	9(21)	Angiomatoid MFH	30(33)	Angiomatoid MFH	8(21)	Synovial sarcoma	11(21)	Synovial sarcoma	28(22)
	Angiomatoid MFH	7(16)	Synovial sarcoma	14(15)	MFH	5(13)	DFSP	9(17)	Angiomatoid MFH	22(17)
	Synovial sarcoma	5(12)	Fibrosarcoma	8(9)	Ewing sarcoma	4(10)	Rhabdomyosarcoma	5(9)	MFH	13(10)
6-15	MFH	4(9)	MPNST	7(8)	MPNST	4(10)	Angiosarcoma	4(8)	Liposarcoma	11(9)
	Angiosarcoma	3(7)	MFH	7(8)	Rhabdomyosarcoma	4(10)	Clear cell sarcoma	4(8)	MPNST	9(7)
	Rhabdomyosarcoma	3(7)	Rhabdomyosarcoma	7(8)	Fibrosarcoma	3(8)	Fibrosarcoma	4(8)	DFSP	8(6)
	Clear cell sarcoma	2(5)	Epithelioid sarcoma	4(4)	Synovial sarcoma	3(8)	Chondrosarcoma	3(6)	Rhabdomyosarcoma	6(5)
	Other	10(23)	Other	15(16)	Other	8(21)	Other	13(25)	Other	31(24)
	Epithelioid sarcoma	25(29)	Synovial sarcoma	32(23)	Synovial sarcoma	13(18)	Synovial sarcoma	27(30)	Synovial sarcoma	76(22)
	MFH	11(13)	MFH	19(14)	DFSP	12(16)	Clear cell sarcoma	10(11)	Liposarcoma	45(13)
	DFSP	7(8)	MPNST	16(12)	MPNST	11(15)	Fibrosarcoma	7(8)	MPNST	44(13)
16-25	Synovial sarcoma	7(8)	Fibrosarcoma	12(9)	Fibrosarcoma	8(11)	DFSP	7(8)	MFH	36(11)
	Rhabdomyosarcoma	7(8)	Angiomatoid MFH	10(7)	MFH	8(11)	MFH	6(7)	Fibrosarcoma	24(7)
	Angiomatoid MFH	5(6)	Epithelioid sarcoma	9(7)	Rhabdomyosarcoma	4(5)	Hemangioendothelioma	6(7)	DFSP	18(5)
	Hemangioendothelioma	5(6)	Hemangioendothelioma	6(4)	Angiomatoid MFH	3(4)	MPNST	5(6)	Angiomatoid MFH	15(4)
	Other	19(22)	Other	34(25)	Other	15(20)	Other	22(24)	Other	80(24)
	MFH	26(18)	MFH	65(28)	DFSP	55(33)	Synovial sarcoma	50(26)	Liposarcoma	196(28)
	Epitheliod sarcoma	24(16)	MPNST	29(12)	MFH	30(18)	Clear cell sarcoma	25(13)	MFH	151(21)
	Synovial sarcoma	21(14)	Fibrosarcoma	25(11)	Liposarcoma	22(13)	MFH	25(13)	Synovial sarcoma	78(11)
26-45	Fibrosarcoma	17(12)	Synovial sarcoma	23(10)	MPNST	21(12)	Hemangioendothelioma	14(7)	MPNST	70(10)
	Clear cell sarcoma	9(6)	Liposarcoma	20(8)	Fibrosarcoma	10(6)	DFSP	13(7)	DFSP	47(7)
	Liposarcoma	9(6)	DFSP	18(8)	Synovial sarcoma	7(4)	Liposarcoma	13(7)	Leiomyosarcoma	35(5)
	MPNST	7(5)	Epithelioid sarcoma	13(6)	Chondrosarcoma	6(4)	MPNST	11(6)	Fibrosarcoma	33(5)
	Other	33(23)	Other	43(18)	Other	18(11)	Other	38(20)	Other	98(14)
	MFH	16(19)	MFH	133(46)	MFH	66(35)	MFH	39(25)	MFH	399(43)
	Synovial sarcoma	12(14)	Liposarcoma	34(12)	Liposarcoma	39(21)	Synovial sarcoma	27(17)	Liposarcoma	232(25)
	Fibrosarcoma	8(10)	Leiomyosarcoma	22(8)	DFSP	22(12)	Leiomyosarcoma	19(12)	Leiomyosarcoma	63(7)
46-65	Epithelioid sarcoma	7(8)	Fibrosarcoma	18(6)	MPNST	20(11)	Kaposi sarcoma	14(9)	Synovial sarcoma	40(4)
	Liposarcoma	7(8)	MPNST	17(6)	Leiomyosarcoma	14(7)	Liposarcoma	9(6)	MPNST	38(4)
	Chondrosarcoma	7(8)	Synovial sarcoma	16(5)	Fibrosarcoma	8(4)	Fibrosarcoma	8(5)	Chondrosarcoma	37(4)
	Clear cell sarcoma	5(6)	Hemangioendothelioma	9(3)	Synovial sarcoma	4(2)	Clear cell sarcoma	7(5)	Fibrosarcoma	24(3)
	Other	22(26)	Other	43(15)	Other	15(8)	Other	32(21)	Other	87(9)
	MFH	28(35)	MFH	183(60)	MFH	67(50)	Kaposi sarcoma	49(37)	MFH	455(55)
	Leiomyosarcoma	8(10)	Liposarcoma	25(8)	Liposarcoma	30(23)	MFH	26(19)	Liposarcoma	178(22)
	Synovial sarcoma	6(8)	Leiomyosarcoma	23(8)	MPNST	12(9)	Leiomyosarcoma	20(15)	Leiomyosarcoma	86(10)
≥66	Kaposi sarcoma	5(6)	MPNST	20(7)	DFSP	6(5)	Fibrosarcoma	9(7)	Fibrosarcoma	22(3)
	DFSP	4(5)	Kaposi sarcoma	10(3)	Fibrosarcoma	4(3)	Chondrosarcoma	6(4)	Chondrosarcoma	16(2)
	MPNST	4(5)	Fibrosarcoma	8(3)	Leiomyosarcoma	3((2)	MPNST	5(4)	MPNST	15(2)
	Clear cell sarcoma	3(4)	Angiosarcoma	6(2)	Chondrosarcoma	2(2)	Liposarcoma	3(2)	Synovial sarcoma	11(1)
	Other	21(27)	Other	29(10)	Other	9(7)	Other	16(12)	Other	43(5)

Table 2. Distribution of common malignant soft-tissue tumors by anatomic location and patient age: part I. MFH, malignant fibrous histiocytoma; DFSP, dermatofibrosarcoma protuberans; MPNST, malignant peripheral nerve sheath tumor

<sup>a</sup> Based on an analysis of 12,370 cases seen in consultation by the Department of Soft Tissue Pathology, AFIP, over 10 years (modified from AJR 1995;164:129-134) <sup>b</sup> 5(45) indicates there were 5 fibrosarcomas in the hand and wrist of patients 0-5 years, and this represents 45% of all malignant tumors in this location and age group 

 Table 3. Distribution of common malignant soft-tissue tumors by anatomic location and age: part II. MFH, malignant fibrous histiocytoma; DFSP, dermatofibrosarcoma protuberans;

 MPNST, malignant peripheral nerve sheath tumor

Ages	Hip, groin, buttocks	No (%)	Head and neck	No (%)	Trunk	No (%)	Retroperitoneum	No (%)
	Fibrosarcoma	7(32)	Fibrosarcoma	22(37)	Fibrosarcoma	13(26)	Fibrosarcoma	4(20)
	Giant cell fibroblastoma	3(14)	Rhabdomyosarcoma	20(33)	Giant cell fibroblastoma	8(16)	Neuroblastoma	4(20)
	Rhabdomyosarcoma	3(14)	Malig hemangiopericytoma	3(5)	Rhabdomyosarcoma	8(16)	Rhabdomyosarcoma	4(20)
)-5	DFSP	2(9)	Alveolar soft part sarcoma	2(3)	Angiomatoid MFH	6(12)	Ganglioneuroblastoma	3(15)
	MFH	2(9)	DFSP	2(3)	DFSP	4(8)	Angiosarcoma	2(10)
	Leiomyosarcoma	1(5)	MPNST	2(3)	Ewing sarcoma	3(6)	Leiomyosarcoma	2(10)
	Synovial sarcoma	1(5)	Giant cell fibroblastoma	2(3)	Neuroblastoma	3(6)	Alveolar soft part sarcoma	1(5)
	Other	3(14)	Other	7(12)	Other	5(10)		
	Angiomatoid MFH	8(21)	Rhabdomyosarcoma	17(26)	Angiomatoid MFH	14(15)	Rhabdomyosarcoma	9(31)
	Synovial sarcoma	7(19)	Fibrosarcoma	13(20)	Fibrosarcoma	13(14)	MPNST	5(17)
	Rhabdomyosarcoma	6(16)	Synovial sarcoma	7(11)	Ewing sarcoma	12(13)	Neuroblastoma	4(14)
-15	MFH	4(11)	MPNST	6(9)	DFSP	12(13)	Ewing sarcoma	2(7)
	Epithelioid sarcoma	2(5)	MFH	6(9)	MPNST	9(10)	Fibrosarcoma	2(7)
	Fibrosarcoma	2(5)	Angiomatoid MFH	4(6)	Rhabdomyosarcoma	8(9)	MFH	2(7)
	MPNST	2(5)	DFSP	2(3)	MFH	3(3)	Malignant	2(7)
	Other	7(18)	Other	10(15)	Other	20(22)	hemangiopericytoma	3(10)
							Other	
	Synovial sarcoma	15(18)	Fibrosarcoma	15(17)	DFSP	37(23)	MPNST	9(20)
	MPNST	13(16)	DFSP	14(16)	MFH	21(13)	Ewing sarcoma	8(18)
	Liposarcoma	8(10)	MPNST	8(9)	MPNST	19(12)	Leiomyosarcoma	6(14)
-25	DFSP	6(7)	Synovial sarcoma	8(9)	Fibrosarcoma	15(9)	Ganglioneuroblastoma	4(9)
	MFH	6(7)	Rhabdomyosarcoma	8(9)	Synovial sarcoma	13(8)	Neuroblastoma	4(9)
	Rhabdomyosarcoma	5(6)	MFH	7(8)	Ewing sarcoma	12(7)	Rhabdomyosarcoma	3(7)
	Leiomyosarcoma	4(5)	Angiomatoid MFH	6(7)	Angiomatoid MFH	6(4)	Malig hemangiopericytoma	2(5)
	Other	26(31)	Other	23(26)	Other	38(24)	Other	8(18)
	Liposarcoma	45(18)	DFSP	59(30)	DFSP	129(30)	Leiomyosarcoma	57(32)
	DFSP	42(17)	MPNST	27(14)	MFH	77(18)	Liposarcoma	52(29)
	MFH	38(16)	Liposarcoma	18(9)	MPNST	45(10)	MFH	22(12)
-45	Leiomyosarcoma	26(11)	MFH	15(8)	Liposarcoma	41(9)	MPNST	11(6)
	MPNST	15(6)	Fibrosarcoma	14(7)	Fibrosarcoma	36(8)	Fibrosarcoma	7(4)
	Synovial sarcoma	13(5)	Synovial sarcoma	10(5)	Synovial sarcoma	20(5)	Malig hemangiopericytoma	7(4)
	Fibrosarcoma	12(5)	Ángiosarcoma	9(4)	Ángiosarcoma	15(3)	Ewing sarcoma	3(2)
	Other	53(22)	Other	42(22)	Other	70(16)	Other	20(11)
	Liposarcoma	67(24)	MFH	54(28)	MFH	131(31)	Liposarcoma	170(33)
	MFH	66(23)	DFSP	28(15)	Liposarcoma	80(Ì9)	Leiomyosarcoma	154(30)
	Leiomyosarcoma	40(14)	MPNST	23(12)	DFSP	60(14)	MFH	111(22)
-66	DFSP	20(7)	Liposarcoma	22(12)	MPNST	35(8)	MPNST	23(5)
	Fibrosarcoma	16(6)	Angiosarcoma	16(8)	Leiomyosarcoma	27(6)	Malignant mesenchymoma	10(2)
	Synovial sarcoma	14(5)	Atypical fibroxanthoma	12(6)	Fibrosarcoma	24(6)	Fibrosarcoma	9(2)
	Chondrosarcoma	14(5)	Leiomyosarcoma	11(6)	Angiosarcoma	15(4)	Malign hemangiopericytoma	7(1)
	Other	46(16)	Other	24(13)	Other	50(12)	Other	27(5)
	MFH	111(46)	MFH	82(34)	MFH	137(44)	Liposarcoma	164(39)
	Liposarcoma	49(20)	Atypical fibroxanthoma	41(17)	Liposarcoma	56(18)	Leiomyosarcoma	118(28)
	Leiomyosarcoma	24(10)	Angiosarcoma	27(11)	Leiomyosarcoma	23(7)	MFH	93(22)
66	Angiosarcoma	11(5)	Liposarcoma	20(8)	MPNST	20(6)	MPNST	13(3)
	MPNST	11(5)	MPNST	16(7)	DFSP	17(5)	Fibrosarcoma	8(2)
	Fibrosarcoma	10(4)	Leiomyosarcoma	13(5)	Fibrosarcoma	12(4)	Osteosarcoma	6(1)
	Chondrosarcoma	7(3)	Fibrosarcoma	10(4)	Chondrosarcoma	11(4)	Malignant mesenchymoma	5(1)
	Chonteroodi vonna	20(8)	1 1010000000000	10(7)	Other	35(11)	Other	9(2)

<sup>a</sup> Based on an analysis of 12,370 cases seen in consultation by the Department of Soft Tissue Pathology, AFIP, over 10 years (modified from AJR 1995;164:129-134) <sup>b</sup> 5(45) indicates there were 5 fibrosarcomas in the hand and wrist of patients 0-5 years, and this represents 45% of all malignant tumors in this location and age group 58

Ages	Hand and Wrist	No (%)	Upper Extremity	No (%)	Axilla and Shoulder	No (%)	Foot and Ankle	No (%)	Lower Extremity	No (%)
	Hemangioma	15(15)	Fibrous hamartoma infancy	15(16)	Fibrous hamartoma infancy	23(29)	Granuloma annulare	23(30)	Granuloma annulare	42(23)
	Granuloma annulare	14(14)	Granuloma annulare	15(16)	Hemangioma	12(15)	Infantile fibromatosis	11(14)	Hemangioma	26(14)
	Infantile fibromatosis	13(13)	Hemangioma	14(15)	Lipoblastoma	11(14)	Hemangioma	8(11)	Myofibromatosis	16(9)
0-5	Infantile digital fibroma	8(8)	Infantile fibromatosis	12(13)	Fibrous hamartoma	7(9)	Fibromatosis	8(11)	Fibrous histiocytoma	15(8)
	Fibromatosis	8(8)	Fibrous histiocytoma	6(6)	Myofibromatosis	6(8)	Infantile digital fibroma	7(9)	Lipoblastoma	13(7)
	Aponeurotic fibroma	7(7)	Juvenile xanthogranuloma	6(6)	Lymphangioma	5(6)	Lipoblastoma	6(8)	Lymphangioma	10(6)
	Fibrous histiocytoma	5(5)	Myofibromatosis	6(6)	Nodular fasciitis	4(5)	Lipoma	4(5)	Juvenile xanthogranuloma	10(6)
	Other	27(28)	Other	20(21)	Other	12(15)	Other	9(12)	Other	48(27)
	Fibrous histiocytoma	32(14)	Fibrous histiocytoma	41(23)	Fibrous histiocytoma	25(34)	Fibromatosis	35(22)	Hemangioma	47(22)
	Hemangioma	31(13)	Nodular fasciitis	39(21)	Nodular fasciitis	18(25)	Granuloma annulare	21(13)	Fibrous histiocytoma	34(16)
	Aponeurotic fibroma	25(11)	Hemangioma	24(13)	Hemangioma	7(10)	Hemangioma	21(13)	Nodular fasciitis	22(10)
6-15	Fibroma tendon sheath	22(9)	Granuloma annulare	12(7)	Granular cell tumor	4(5)	Fibrous histiocytoma	14(9)	Granuloma annulare	20(9)
	GCT tendon sheath <sup>2</sup>	17(7)	Fibromatosis	11(6)	Neurofibroma	3(4)	GCT tendon sheath	13(8)	Fibromatosis	14(6)
	Fibromatosis	13(6)	Neurofibroma	7(4)	Lymphangioma	2(3)	Chondroma	11(7)	Lipoma	13(6)
	Lipoma	9(4)	Neurothekeoma	6(3)	Myofibromatosis	2(3)	Lipoma	9(6)	Neurofibroma	8(4)
	Other	86(37)	Other	42(23)	Other	12(16)	Other	37(23)	Other	58(27)
	GCT tendon sheath	84(20)	Nodular fasciitis	130(35)	Fibrous histiocytoma	62(36)	Fibromatosis	46(22)	Fibrous histiocytoma	118(24)
	Fibrous histiocytoma	57(14)	Fibrous histiocytoma	87(23)	Nodular fasciitis	35(20)	GCT tendon sheath	29(14)	Nodular fasciitis	61(13)
	Hemangioma	40(10)	Hemangioma	36(10)	Fibromatosis	16(9)	Granuloma annulare	25(11)	Hemangioma	55(11)
6-25	Fibroma tendon sheath	40(10)	Neurofibroma	24(6)	Lipoma	14(8)	Fibrous histiocytoma	24(12)	Neurofibroma	48(10)
0 20	Nodular fasciitis	26(6)	Granuloma annulare	20(5)	Neurofibroma	12(7)	Hemangioma	13(6)	Fibromatosis	38(8)
	Granuloma annulare	21(5)	Granular cell tumor	17(5)	Hemangioma	4(2)	PVNS <sup>3</sup>	12(6)	Lipoma	22(5)
	Ganglion	20(5)	Schwannoma	11(3)	Schwannoma	4(2)	Neurofibroma	11(5)	Schwannoma	20(4)
	Other	132(31)	Other	51(14)	Other	25(15)	Other	45(22)	Other	122(25)
	Fibrous histiocytoma	167(18)	Nodular fasciitis	309(38)	Lipoma	105(28)	Fibromatosis	99(21)	Fibrous histiocytoma	245(25)
	GCT tendon sheath	148(16)	Fibrous histiocytoma	145(18)	Fibrous histiocytoma	92(24)	Fibrous histiocytoma	74(16)	Nodular fasciitis	229(23)
	Fibroma tendon sheath	148(10) 106(11)	Angiolipoma	48(6)	Nodular fasciitis	55(14)	GCT tendon sheath	41(9)	Lipoma	101(10)
6-45	Hemangioma	86(10)	Hemangioma	43(5)	Fibromatosis	29(8)	Hemangioma	36(8)	Neurofibroma	71(7)
0-45	Nodular fasciitis	79(8)	Schwannoma	43(5)	Hemangioma	17(4)	Schwannoma	30(8)	Schwannoma	59(6)
	Fibromatosis	46(5)	Neurofibroma	37(5)	Neurofibroma	13(3)	Neurofibroma	24(5)	Myxoma	53(5)
	Chondroma	40(3)	Lipoma	32(4)	Schwannoma	13(3) 12(3)	Chondroma	23(5)	Hemangioma	52(5)
	Other	269(29)	Other	153(19)	Other	57(15)	Other	135(29)	Other	185(19)
		. ,		· · · ·				. ,		
	GCT tendon sheath	143(23)	Nodular fasciitis	86(20)	Lipoma	189(58)	Fibromatosis	83(25)	Lipoma	157(23)
	Fibrous histiocytoma	63(10)	Lipoma	80(19)	Fibrous histiocytoma	28(9)	Fibrous histiocytoma	43(13)	Myxoma	109(16)
	Hemangioma	61(10)	Fibrous histiocytoma	44(10)	Myxoma	16(5)	Lipoma	35(11)	Fibrous histiocytoma	93(14)
46-65	Lipoma	59(9)	Schwannoma	30(7)	Fibromatosis	14(4)	Schwannoma	25(8)	Nodular fasciitis	40(6)
	Chondroma	52(8)	Neurofibroma	24(6)	Nodular fasciitis	13(4)	GCT tendon sheath	21(6)	Schwannoma	39(6)
	Fibromatosis	43(7)	Myxoma	24(6)	Schwannoma	12(4)	Chondroma	21(6)	Neurofibroma	31(5)
	Fibroma tendon sheath	37(6)	Hemangioma	19(4)	Granular cell tumor	12(4)	Hemangioma	16(5)	Proliferative fasciitis	28(4)
	Other	172(27)	Other	125(29)	Other	44(13)	Other	89(27)	Other	186(27)
	GCT tendon sheath	51(21)	Lipoma	39(22)	Lipoma	83(58)	Fibromatosis	16(14)	Lipoma	68(26)
	Hemangioma	24(10)	Myxoma	19(11)	Myxoma	14(10)	Schwannoma	15(13)	Myxoma	44(17)
	Schwannoma	24(10)	Nodular fasciitis	18(10)	Schwannoma	6(4)	Fibrous histiocytoma	13(11)	Fibrous histiocytoma	33(13)
5 & Up	Chondroma	24(10)	Schwannoma	17(9)	Fibromatosis	5(3)	Chondroma	11(9)	Schwannoma	31(12)
	Neurofibroma	21(9)	Glomus tumor	12(7)	Fibrous histiocytoma	5(3)	Lipoma	10(8)	Hemangiopericytoma	10(4)
	Fibromatosis	14(6)	Neurofibroma	10(6)	Proliferative fasciitis	5(3)	Granuloma annulare	8(7)	Neurofibroma	9(4)
	Lipoma	13(5)	Angiolipoma	10(6)	Hemangioma	4(3)	GCT tendon sheath	6(5)	Hemangioma	8(3)
	Other	71(29)	Other	55(31)	Other	22(15)	Other	39(33)	Other	56(22)

**Table 4.** Distribution of common benign soft-tissue tumors by anatomic location and age: part I

<sup>1</sup> 15(15) indicates there were 15 hemangioma in the hand and wrist of patients 0-5 years, and this represents 15% of all benign tumors in this location and age group
 <sup>2</sup> Giant cell tumor of tendon sheath
 <sup>3</sup> Pigmented villonodular synovitis

Ages	Hip, Groin & Buttocks	No (%)	Head and Neck	No (%)	Trunk	No (%)	Retroperitoneum	No (%)
	Fibrous hamartoma infancy	14(20)	Nodular fasciitis	47(20)	Hemangioma	36(18)	Lipoblastoma	7(37)
	Lipoblastoma	14(20)	Hemangioma	43(18)	Juvenile xanthogranuloma	24(12)	Lymphangioma	5(26)
	Myofibromatosis	8(11)	Myofibromatosis	27(11)	Myofibromatosis	24(12)	Hemangioma	4(21)
0-5	Lymphangioma	7(10)	Fibromatosis	17(7)	Nodular fasciitis	17(8)	Ganglioneuroma	2(11)
	Fibrous histiocytoma	5(7)	Granuloma annulare	14(6)	Lipoblastoma	17(8)	Fibrous hamartoma	1(5)
	Nodular fasciitis	4(6)	Fibrous histiocytoma	13(5)	Infantile fibromatosis	15(7)	infancy	
	Infantile fibromatosis	4(6)	Infantile fibromatosis	13(5)	Fibrous hamartoma infancy	15(7)		
	Other	14(20)	Other	63(27)	Other	55(27)		
	Nodular fasciitis	15(27)	Nodular fasciitis	75(33)	Nodular fasciitis	54(28)	Lymphangioma	7(37)
	Fibroma	7(13)	Fibrous histiocytoma	34(15)	Fibrous histiocytoma	43(22)	Ganglioneuroma	4(21)
	Fibrous histiocytoma	6(11)	Neurofibroma	23(10)	Hemangioma	25(13)	Schwannoma	2(11)
6-15	Fibromatosis	5(9)	Hemangioma	21(9)	Lipoma	9(5)	Fibromatosis	2(11)
	Lipoma	5(9)	Myofibromatosis	14(6)	Neurofibroma	7(4)	Paraganglioma	1(5)
	Lipoblastoma	3(5)	Fibromatosis	12(5)	Fibromatosis	6(3)	Hemangioma	1(5)
	Neurofibroma	3(5)	Lipoma	6(3)	Granular cell tumor	6(3)	Inflammatory pseudotumor	1(5)
	Other	11(20)	Other	43(19)	Other	45(23)	Other	1(5)
	Neurofibroma	20(16)	Nodular fasciitis	61(21)	Nodular fasciitis	112(24)	Fibromatosis	14(20
	Fibromatosis	18(15)	Hemangioma	48(17)	Fibromatosis	72(16)	Schwannoma	10(14
	Fibrous histiocytoma	18(15)	Fibrous histiocytoma	45(16)	Fibrous histiocytoma	71(15)	Neurofibroma	9(13
6-25	Nodular fasciitis	12(10)	Neurofibroma	37(13)	Hemangioma	52(11)	Hemangiopericytoma	8(11
	Hemangioma	9(7)	Schwannoma	19(7)	Neurofibroma	38(8)	Lymphangioma	8(11
	Lipoma	8(7)	Fibromatosis	11(4)	Lipoma	21(5)	Ganglioneuroma	6(8)
	Hemangiopericytoma	8(7)	Lipoma	10(4)	Schwannoma	17(4)	Hemangioma	4(6)
	Other	29(24)	Other	56(19)	Other	79(17)	Other	12(17
	Lipoma	57(17)	Lipoma	168(22)	Lipoma	178(19)	Schwannoma	38(23
	Neurofibroma	38(12)	Nodular fasciitis	145(19)	Nodular fasciitis	150(16)	Fibromatosis	30(18
	Fibrous histiocytoma	37(11)	Fibrous histiocytoma	137(18)	Fibromatosis	148(16)	Hemangiopericytoma	25(15
26-45	Fibromatosis	36(11)	Hemangioma	97(13)	Fibrous histiocytoma	98(10)	Neurofibroma	13(8
	Nodular fasciitis	31(9)	Neurofibroma	57(8)	Hemangioma	78(8)	Angiomyolipoma	10(6
	Hemangiopericytoma	24(7)	Hemangiopericytoma	37(5)	Neurofibroma	65(7)	Hemangioma	9(5)
	Мухота	22(7)	Schwannoma	27(4)	Schwannoma	51(5)	Sclerosing retroperitonitis	7(4)
	Other	83(25)	Other	91(12)	Other	180(19)	Other	34(20
	Lipoma	76(35)	Lipoma	306(46)	Lipoma	290(44)	Schwannoma	33(19
	Myxoma	36(17)	Nodular fasciitis	66(10)	Fibromatosis	63(9)	Fibromatosis	25(14
	Fibrous histiocytoma	17(8)	Hemangioma	55(8)	Nodular fasciitis	44(7)	Sclerosing retroperitonitis	25(14
6-65	Schwannoma	17(8)	Fibrous histiocytoma	42(6)	Hemangioma	31(5)	Hemangiopericytoma	21(12
	Nodular fasciitis	11(5)	Neurofibroma	30(4)	Fibrous histiocytoma	29(4)	Angiomyolipoma	12(7
	Hemangiopericytoma	11(5)	Schwannoma	25(4)	Neurofibroma	28(4)	Lipoma	10(6
	Hemangioma	9(4)	Myxoma	23(3)	Schwannoma	28(4)	Paraganglioma	9(5)
	Other	40(18)	Other	120(18)	Other	151(23)	Other	40(23
	Lipoma	22(21)	Lipoma	158(50)	Lipoma	124(42)	Schwannoma	19(26
	Myxoma	16(15)	Hemangioma	22(7)	Fibromatosis	26(9)	Hemangiopericytoma	14(19
	Neurofibroma	13(12)	Schwannoma	18(6)	Neurofibroma	20(7)	Lipoma	6(8)
≥66	Schwannoma	10(9)	Fibrous histiocytoma	17(5)	Schwannoma	18(6)	Mesothelioma	6(8)
	Hemangiopericytoma	10(9)	Neurofibroma	16(5)	Elastofibroma	17(6)	Sclerosing retroperitonitis	5(7)
	Hemangioma	8(8)	Nodular fasciitis	13(4)	Myxoma	16(5)	Fibromatosis	4(6)
	Nodular fasciitis	4(4)	Myxoma	12(4)	Hemangioma	14(5)	Paraganglioma	4(6)
	Other	23(22)	Other	58(18)	Other	61(21)	Other	14(19

 Table 5. Distribution of common benign soft-tissue tumors by anatomic location and age: part II

<sup>a</sup> Based on an analysis of 18,677 cases seen in consultation by the Department of Soft Tissue Pathology, AFIP, over 10 years n (modified from AJR 1995;164:395-402)

signal intensity on T1-weighted images. Signs that had the greatest specificity for malignancy included tumor necrosis, bone or neurovascular involvement, and a mean diameter of more than 66 mm.

#### **Differential Diagnosis**

When a specific diagnosis is not possible, it is often useful to formulate a suitably ordered differential diagnosis on the basis of imaging features, suspected biological potential, and a knowledge of tumor prevalence based on the patient's age and the anatomic location of the lesion. This can be further refined by considering clinical history and radiologic features, such as pattern of growth, signal intensity and localization (subcutaneous, intramuscular, intermuscular, etc.). The most common malignant and benign lesions, by tumor location and patient age, are listed in Tables 2-5.

#### Summary

In summary, MR is the preferred modality for the evaluation of a soft-tissue mass following radiography. The radiologic appearance of certain soft-tissue tumors or tumorlike processes may be sufficiently unique to allow a strong presumptive radiologic diagnosis. It must be emphasized that MR imaging cannot *reliably* distinguish between benign and malignant lesions. When radiologic evaluation is nonspecific, one is ill advised to suggest that a lesion is benign or malignant solely on its MR appearance. When a specific diagnosis is not possible, knowledge of tumor prevalence by location and patient age, with appropriate clinical history and radiologic features, can be used to establish a suitably ordered differential diagnosis.

#### References

- 1. Hajdu SI (1981) Soft tissue sarcomas: classification and natural history. CA Cancer J Clin 31:271-280
- 2. du Boulay CEH (1985) Immunohistochemistry of soft tissue tumors: a review. J Pathol 146:77-94
- 3. Weiss SW, Goldblum JR (2001) Enzinger and Weiss's Soft Tissue Tumors, 4th ed. St. Louis: Mosby, pp 1-44
- Mettlin C, Priore R, Rao U, Gamble D, Lane W, Murphy GP (1982) Results of the national soft-tissue sarcoma registry. Analysis of survival and prognostic factors. J Surg Oncology 19:224-227
- Osment LS (1968) Cutaneous lipomas and lipomatosis. Surg Gynecol Obstet 127:129-132
- Leffert RD (1972) Lipomas of the upper extremity. J Bone Joint Surg Am 54-A:1262-1266
- Rydholm A, Berg NO (1983) Size, site and clinical incidence of lipoma. Factors in the differential diagnosis of lipoma and sarcoma. Acta Orthop Scand 54:929-934
- Disler DG, Alexander AA, Mankin HJ, O'Connell JX, Rosenberg AE, Rosenthal DI (1993) Multicentric fibromatosis with metaphyseal dysplasia. Radiology 187:489-492
- Rock MG, Pritchard DJ, Reiman HM, Soule EH, Brewster RC (1984) Extra-abdominal desmoid tumors. J Bone Joint Surg Am 66-A:1369-1374

- Sundaram M, Duffrin H, McGuire MH, Vas W (1988) Synchronous multicentric desmoid tumors (aggressive fibromatosis) of the extremities. Skeletal Radiol 17:16-19
- Moser RP, Parrish WM (2001) Radiologic evaluation of soft tissue tumors. In: Weiss SW, Goldblum JR. Enzinger and Weiss's Soft Tissue Tumors, 4th ed. St. Louis: Mosby, pp 45-102
- Rubin DA, Kneeland JB (1994) MR imaging of the musculoskeletal system: technical considerations for enhancing image quality and diagnostic yield. AJR 163:1155-1163
- Mirowitz SA (1993) Fast scanning and fat-supression MR imaging of musculoskeletal disorders. AJR 161:1147-1157
- 14. Shuman WP, Baron RL, Peters MJ, Tazioli PK (1989) Comparison of STIR and spin-echo MR imaging at 1.5T in 90 lesions of the chest, liver and pelvis. AJR 152:853-859
- Dwyer AJ, Frank JA, Sank VJ, Reinig JW, Hickey AM, Doppman JL (1988) Short-Ti inversion-recovery pulse sequence: analysis and initial experience in cancer imaging. Radiology 168:827-836
- Beltran J, Chandnani V, McGhee RA, Kursungoglu-Brahme S (1991) Gadopentetate dimeglumine-enhanced MR imaging of the musculoskeletal system. AJR 156:457-466
- Verstraete KL, De Deene Y, Roels H, Dierick A, Uyttendaele D, Kunnen M (1994) Benign and malignant musculoskeletal lesions: dynamic contrast-enhanced MR imaging-parametric "first pass" images depict tissue vascularization and perfusion. Radiology 192:835-843
- van Rijswijk CSP, Geirnaerdt MJA, Hogendoorn PCW, et al (2004) Soft tissue tumors: value of static and dynamic gadopentetate dimeglumine-enhanced MR imaging in prediction of malignancy. Radiology 233:493-502
- Harkens KL, Moore TE, Yuh WTC, et al (1993) Gadoliniumenhanced MRI of soft-tissue masses. Australas Radiol 37:30-34
- Seeger LL, Widoff BE, Bassett LW, Rosen G, Eckardt JJ (1991) Preoperative evaluation of osteosarcoma: value of gadopentitate dimeglumine-enhanced MR imaging. AJR 157:347-351
- Kransdorf MJ, Murphey MD (1997) The use of gadolinium in the MR evaluation of soft tissue tumors. Semin Ultrasound CT MR 18:251-268
- 22. Kransdorf MJ, Jelinek JS, Moser, et al (1989)Soft-tissue masses: diagnosis using MR imaging. AJR 153:541-547
- Berquist TH, Ehman RL, King BF, Hodgman CG, Ilstrup DM (1990) Value of MR imaging in differentiating benign from malignant soft tissue masses: study of 95 lesions. AJR 155:1251-1255
- Crim JR, Seeger LL, Yao L, Chandnani V, Eckardt JJ (1992) Diagnosis of soft-tissue masses with MR imaging: can benign masses be differentiated from malignant ones? Radiology 185:581-586
- Myhre-Jensen O (1981) A consecutive 7-year series of 1331 benign soft tissue tumors. Clinicopathologic data. Comparison with sarcomas. Acta Orthop Scand 52:287-293
- Rydholm A (1983) Management of patients with soft-tissue tumors. Strategy developed at a regional oncology center. Acta Orthop Scand Suppl 203:13-77
- Peabody TD, Simon MA (1993) Principles of staging of soft-tissue sarcomas. Clin Orthop 289:19-31
- Beltran J, Simon DC, Katz W, Weis LD (1987) Increased MR signal intensity in skeletal muscle adjacent to malignant tumors: pathologic correlation and clinical relevance. Radiology 162:251-255
- 29. Hanna SL, Fletcher BD, Parham DM, Bugg MF (1991) Muscle edema in musculoskeletal tumors: MR imaging characteristics and clinical significance. J Magn Reson Imaging 1:441-449
- DeSchepper A, Ramon F, Degryse H (1992) Statistical analysis of MRI parameters predicting malignancy in 141 soft tissue masses. Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr : 156:587-591

# **Tumors and Tumor-Like Lesions of Bone**

M. Sundaram<sup>1</sup>, D. Vanel<sup>2</sup>

<sup>1</sup> Radiology, Cleveland Clinic Foundation, Cleveland, OH, USA

<sup>2</sup> Institut Gustav Roussy, Villejuif, France

# Introduction

Primary malignant bone tumors are rare. In one large series they constituted 0.2% of all neoplasms [1]. In the year 2000, the National Cancer Institute's Surveillance. Epidemiology and End Results (SEER) program estimated 1,220,000 new cancer cases in the USA [2]. Of these patients, 20-85% may develop bone metastases [3, 4]. During the same period it was estimated there would be 1.400 sarcomas of bone [2]. In the UK, there were 527 new cases of primary bone cancer in 1999, with a rate of eight per million of the female population and 10 per million for the male population [5]. Despite their relative rarity, or perhaps because of it, radiologists need to be familiar with these lesions, which can mimic or be mimicked by benign and non-neo-plastic reactive lesions, so that imaging beyond the radiograph and biopsy is performed on a logical and knowledgeable basis. The spectrum and breadth of primary bone tumors is extensive, as outlined by the 2002 tables of the World Health Organization [6]. (WHO) and the American Joint Committee on Cancer (AJCC) [7]. The relative frequencies of bone sarcomas according to histological type, sex, and race are shown in Table 1 [8]. No classification system, however, is so allencompassing as to include traumatic reactive and infective lesions, all of which have to be considered by the radiologist when faced with a seemingly neoplastic lesion.

Primary bone tumors as a group are bimodal, the first peak occurring during the second decade of life and the second peak in patients older than 60. Osteosarcoma, the most common non-hematological primary malignancy of bone, occurs predominantly in individuals younger than age 20, and in 80% of these patients the tumor is found in a long bone of the extremity. The predilection for the appendicular skeleton tends to decrease with age, and in patients older than 50, osteosarcoma accounts for 50% of extremity lesions [6]. Chondrosarcomas show a gradual increase in incidence rates up to the age of 50. Half of chondrosarcomas occur in the long bones; other major sites are the pelvis and ribs. Ewing's sarcoma is similar to osteosarcoma in its age incidence and affinity for the long bones but, unlike osteosarcoma, it occurs almost exclusively in the white population (Table 1). Other selective primary tumors will be discussed under separate headings.

The modality that has been introduced to the realm of orthopedic oncology since the previous edition of this volume is positron emission tomography (PET). Elevated 18-fluoro-2-deoxyglucose (18-FDG) accumulation has been demonstrated in a variety of malignant tissues, including sarcomas, and this information can be used to augment the information obtained from conventional imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI).

	Total		W	Thite	Black	
Histological type	No.	%	No.	%	No.	%
Osteosarcoma	922	35.1	743	32.6	106	57.9
Chondrosarcoma	677	25.8	615	27.0	35	19.1
Ewing's sarcoma	420	16.0	392	17.3	7	3.8
Chordoma	221	8.4	200	8.8	4	2.2
Malignant fibrous histiocytomoa	149	5.7	125	5.5	13	7.1
Angiosarcoma	36	1.4	35	1.5	1	0.5
Unspecified	32	1.2	27	1.2	3	1.6
Other	170	6.4	139	6.1	14	7.8
Total	2627	100.0	2276	100.0	183	100.0

Table 1. Relative frequencies of bone sarcomas by histological type, sex and race (from [8])

# **Clinical Features**

In general terms, the important features for staging primary bone tumors are local disease extent, the presence or absence of metastatic disease, and histologic grade of the tumor. There are several staging classifications for bone tumors, each with its own criteria for categorization. According to the AJCC staging, the rules for classification of primary bone tumors include clinical staging (which encompasses imaging), pathological staging, and histological grading [7]. Clinical staging includes all relevant data prior to primary definitive therapy, including physical examination, imaging and biopsy. The radiograph is the mainstay in determining whether a lesion of bone requires further staging and is the modality that usually permits reliable prediction of the probably histology of a lesion of bone.

Staging of all potentially malignant tumors in bone is most accurately achieved by MRI, which should be performed prior to biopsy. Axial imaging complemented by either coronal or sagittal imaging planes using T1-weighted spin-echo sequences augmented by fat-suppressed T2weighted spin-echo sequences most often provides accurate depiction of intra- and extraosseous tumors. The maximum dimension of the tumor must be measured prior to any treatment [9]. CT has a limited role in local staging of tumors but is the examination of choice for evaluation of the thorax for metastatic disease. CT is the preferred technique in those situations in which characterization of a lesion by radiography may be incomplete or difficult because of inadequate visualization of the matrix of a lesion (e.g. usually in flat bones such as the pelvis, scapula or posterior elements of the vertebrae). The role of CT in these circumstances is to characterize the lesion and determine whether it is potentially malignant or not, and often the obtained CT images may suffice for local staging [10, 11]. Technetium scintigraphy is the examination of choice for evaluation of the entire skeleton in order to determine whether there are multiple lesions.

PET has little role in staging the local extent of a tumor. Although the spatial resolution of PET imaging is higher than that of other scintigraphic techniques, it is not sufficiently accurate to delineate tumor margins and anatomic boundaries, and information regarding local disease extent is better assessed by CT/MRI.

Preliminary studies also indicate a limited role for PET in the detection of metastatic disease. Although 18-FDG PET has been found to be accurate in the detection of nodal, pulmonary, and osseous metastases in patients with soft-tissue sarcomas [12, 13], the results for osseous tumors are much less promising. Although pulmonary metastases from osteogenic sarcoma can be detected with 18-FDG PET [14], chest CT has been found to be superior for detection and delineation of pulmonary metastases [15], and a negative PET scan does not exclude the presence of metastases. For each bony metastasis, PET has been found to be superior to conventional skeletal scintigraphy for detection of osseous metastases from Ewing's sarcoma, but inferior for detection of osseous metastases from osteosarcoma [16].

In the context of determining tumor grade, however, PET may play a role. Unlike other imaging techniques, imaging with 18-FDG PET relies on metabolic rather than anatomic parameters. As such, it has been shown in multiple tumor types that intense FDG uptake is predictive of high metabolic activity and therefore higher tumor grade. In a study of 89 patients with sarcoma, both bone and soft-tissue types, tumors were assessed with 18-FDG PET and the results compared to both histopathological grading and markers of cellular proliferation and gene expression [17]. The standardized uptake value on PET was found to be associated with tumor grade, tumor cellularity, mitotic activity, proliferation markers and p53 overexpression. The use of standardized uptake value (SUV) allowed for differentiation between low-grade tumors and intermediate-to high-grade tumors, although PET was not able to reliably differentiate intermediate and high-grade tumors. The association between FDG uptake and tumor grade has been supported by the observation that the ratio of tumor to non-tumor FDG activity in patients with untreated osteosarcoma correlates with both overall survival and event-free survival following adjuvant chemotherapy and surgery [18].

# **Pathological Staging**

Pathological staging includes pathological data from examination of a resected specimen sufficient to evaluate the highest T category, histopathological type and grade, regional lymph nodes as appropriate, or distant metastases (Table 2). As a result, the pathological stage group-

Table 2. Definition of TNM

Primary	Primary tumor (T)									
TX	Prir	nary tumor	cannot be a	assessed						
T0			f primary tu							
T1		Tumor ≥8 cm in greatest dimension								
T2			in greatest d							
Т3		Discontinuous tumors in the primary bone site								
Regiona	Regional lymph nodes (N)									
NX	Reg	ional lymp	h nodes can	not be asse	ssed					
N0	No	No distant metastasis								
N1	Regional lymph node metastasis									
Distant	metas	tasis (M)								
MX	Dis	tant metasta	asis cannot	be assessed						
M0	No	distant met	astasis							
M1	Dis	tant metasta	asis							
M1a	Lun	ıg								
M1b	Oth	er distant s	ites							
Stage g	roupir	ıg								
Stage 1		T1	N0	M0	G1, 2 low grade					
Stage 1	В	T2	N0	M0	G1, 2 low grade					
Stage II	A	T1	N0	M0	G3, 4 high grade					
Stage II	Stage IIB T2			M0	G3, 4 high grade					
Stage II	Stage III T3			M0	Any G					
Stage I	VA	Any T	N0	M1a	Any G					
Stage I	VB	Any T	N1	Any M	Any G					
		Any T	Any N	Mĺb	Any G					

 Table 3. Surgical staging system of the Musculoskeletal Tumor

 Society for Bone and Soft-Tissue Tumors

	-				
Stage	Histologic grade	s Sit	te	Met	astasis
IA	Low (grade I)	Intracompa Extracompa		/	e (M0)
IIA	High (grade 2)	Intracompa Extracompa	/	e (M0)	
IIIA	Low (grade I)	Either (T1		ional or ant (M1)	
IIIB	High (grade 2)	Any T	Any N	M1b	Any G

ing includes any of the following combinations: pT pG pN pM, or pT pG cN cM, or cT cN pM. The TNM staging system is not commonly used for sarcomas of bone because of the rarity with which sarcomas metastasize to lymph nodes.

A special staging system, first described by Enneking, has been adopted by the Musculoskeletal Tumor Society for the Staging of Bone Tumors (Table 3) and includes a combination of the local extent (T), the grade (G) and the presence or absence of regional or distant metastases (M).

In this system, neoplasms are divided into two grades, low (G1) and high (G2). The anatomic extent (T) is divided according to whether the lesion is intracompartmental (A) or extracompartmental (B). The presence or absence of metastasis (M) is the final component. The influence of accurate imaging in determining whether a lesion is intra or extracompartmental is critical to the staging system used and subsequent therapy.

# Choice of Diagnostic Imaging Procedure and Image-Guided Percutaneous Procedures

In practice, the radiologist is faced with a wide array of lesions and has to judiciously decide where the matter can be put to rest with the radiograph, which lesions require further imaging or whether surgery should be scheduled immediately, and which lesions demand follow-up staging. This is best achieved by close consultation with the treating orthopedic surgeon. Based on the radiograph, the next step in patient evaluation is largely determined by the radiographic diagnosis or differential diagnosis that is rendered [19]. The lesion may be:

- Ignored, e.g. non-ossifying fibroma, osteochondroma, bone, infarct, Paget's disease, vertebral hemangioma, asymptomatic fibrous dysplasia
- Reassessed with radiographs following a short interval of time, e.g. enchondroma, simple bone cyst, fibrous dysplasia

- Biopsied directly and treated, e.g. chondroblastoma, or clearly intracompartmental benign lesions such as aneurysmal bone, cyst, simple bone cyst, fractured non-ossifying fibroma, or osteochondroma
- Further imaged and staged because a biopsy is required, e.g. eosinophilic granuloma vs. Ewing's sarcoma, acute osteomyelitis vs. Ewing's sarcoma, enchondroma vs. chondrosarcoma, or when the nature of the lesion demands further staging (i.e. all primary malignant tumors of bone and non-malignant lesions that require local staging for surgical planning, such as large giant cell tumors, aneurysmal bone cysts, and vertebral tumors)

Thus, not every lesion of bone requires further imaging, but evaluations of some lesions of bone would be incomplete without it.

While many bone lesions can be diagnosed with confidence based on their radiographic appearance, certain lesions remain indeterminate, with differential diagnoses including both benign and malignant disease. 18-FDG PET has been examined as a possible means of differentiating benign and malignant disease. Most malignant bone tumors, such as osteogenic sarcoma and Ewing's sarcoma, avidly accumulate FDG and therefore appear to be hyperintense on PET imaging [20]. By contrast, many benign bone lesions are not metabolically active and are therefore hypointense on PET imaging. In a descriptive study of 52 benign and malignant bone lesions, Aoki et al. [21]. found a statistically significant difference in the SUV between benign and malignant bone lesions. The average SUV of the benign group was 2.2, compared to 4.3 for the malignant group. Benign lesions, such as osteochondroma, osteoid osteoma, enchondroma, and bone cysts, had low 18-FDG uptake, and malignant tumors such as osteosarcoma. Ewing's sarcoma, and lymphoma all demonstrated elevated FDG uptake.

However, there was overlap between the benign and malignant groups. In the benign group, the lesion with the highest 18-FDG uptake was the giant cell tumor (GCT). There was no statistical difference in SUV between the GCT and osteosarcoma, and the mean SUV for GCT was higher than that of chondrosarcoma. Other benign lesions that demonstrated high metabolic activity included chondroblastoma, sarcoid, Langerhans' cell histiocytosis, and some cases of fibrous dysplasia. The malignancy with the lowest accumulation of FDG was chondrosarcoma, with no statistical difference in SUV between chondrosarcoma and fibrous dysplasia. In a separate study, the degree of 18-FDG uptake in chondrosarcoma was found to correlate with pathological tumor grade, with low-grade tumors demonstrating minimal 18-FDG accumulation and high-grade tumors demonstrating intense uptake [22]. Using current techniques, PET appears better able to demonstrate benignity than a malignancy, as most lesions that are hypointense are truly benign. The most common false-negative tumor is chondrosarcoma, but those that are hypointense also tend to be low-grade tumors. Lesions that are hyperintense on 18FDG PET must still be approached with caution, due to the number of benign lesions that can demonstrate increased 18-FDG uptake. Recent studies have examined the role of quantitative 18-FDG PET imaging in the evaluation of indeterminate bone lesions, including examination of perfusion and volume of distribution parameters [23, 24]. However, these advanced techniques are not widely utilized, and their clinical usefulness is therefore unclear.

Biopsy of the tumor completes the staging process and is a crucial part of staging. It must be done with an appropriate technique and a clear view of the eventual surgical treatment. In addition, the biopsy site must be carefully planned in order to allow for an eventual en-bloc resection of a malignant neoplasm together with the entire biopsy tract.

Over the past 20 years, percutaneous biopsy under imaging guidance has become the standard procedure for obtaining tissue from bone neoplasms for histological diagnosis. The satisfactory result obtained by this technique of biopsy have, in many centers including that of the authors, supplanted the open-incision biopsy as the primary interventional approach [25-30]. The advantage of percutaneous techniques, leading to its widespread acceptance, has resulted in overall cost-effectiveness of percutaneous biopsy compared with that of open biopsy, a lower complication rate, a smaller limited biopsy tract, and the ability to more rapidly begin neoadjuvant chemotherapy and/or radiation therapy. With a definitive diagnosis, neoadjuvant chemotherapy or radiation therapy can be started the day after core-needle biopsy. A surgical approach often results in a delay of 10 days to 3 weeks to allow wound healing [30]. The radiologist should work closely with the orthopedic oncologist and orthopedic pathologist in a team effort that results in patients being well served. The presence of a cytopathologist to assess the quality and adequacy of the fine-needle aspirate, in order to ensure that viable tumor cells are obtained, improves the quality of the tissue samples and accurate histological diagnostic yield [29]. In addition to the cooperative team effort, radiologists performing this procedure need to have a clear understanding of compartmental anatomy [31]. The hazards and ensuing complications of improperly carried out open biopsies have been addressed twice, 14 years apart, by some of the same authors, who surprisingly found that neither errors in the approach to biopsies nor complications had decreased since the first publications [32, 33].

Radiofrequency ablation is a percutaneous treatment technique most extensively used in osteoid osteoma [34]. This approach was extended to treating a small series of patients with chondroblastoma and was used in the treatment of a complicated malignant vascular tumor [35, 36].

Osteosarcoma (excluding parosetal and low-grade intraosseous osteosarcoma) and Ewing's sarcoma are treated with chemotherapy. Depending on location and extent of disease, radiation therapy may be added to the neoadjuvant armamentarium in the treatment of Ewing's sarcoma. Neoadjuvant therapy is not used in the treatment of chondrosarcoma but may be used in the treatment of dedifferentiated forms of this tumor. The goal of surgery is to resect the tumor with a wide margin and reconstruct the limb. The combination of neoadjuvant therapy and limb-salvage surgery has reduced mortality and morbidity compared with patient outcome prior to the introduction of these treatment techniques. Critical to outcome is accurate depiction of local disease by MRI and systemic disease by CT. It is imperative that MRI be performed after neoadjuvant therapy and prior to definitive surgery (re-staging) and ideally in planes and pulse sequences comparable to the initial staging examination. Since the previous edition of this volume, although there have been advances in CT and MRI significant enough to influence prevailing clinical practice during the course of neoadjuvant therapy or in planning limb salvage, these imaging techniques remain critical for recognizing tumor response, tumor necrosis, and evaluating extent. Radiologists in referring institutions, who are likely to be primarily responsible for imaging and interpretation of patient examination following definitive surgery, need to be aware of some of the findings of tumor recurrence, infection, pseudotumor and rickets, which may be encountered [37-39].

Most osseous malignancies are treated with chemotherapy, and in some instances combined with radiotherapy, followed by surgery. The effectiveness of presurgical treatment regimens can be assessed preoperatively by MRI and postoperatively by evaluating histological necrosis within the tumor. The success of anatomical imaging techniques such as CT and MRI is gauged by examining the physical properties of the tumor, such as macroscopic necrosis and reduction in tumor dimension. A good response as seen on MRI would include disappearance of the soft-tissue element of the tumor and encirclement of the bone by a heterogeneous well-defined cuff of tissue [40].

There are no effective MRI criteria for reliable early identification of good responders; however, an increase in tumor volume and increase in signal intensity of the extraosseous tumor both predict a poor response [41]. Dynamic contrast-enhanced MRI enhancement characteristics have also been investigated. Erlemann et al [42]. reported an 86% accuracy for identification of malignancy using this technique, and in another study, responders with less than 3% viable tumor remaining could be distinguished reliably from non-responders [43]. The intensity of 18-FDG uptake by the tumor is a measure of its metabolism and viability. PET therefore provides a non-invasive means of assessing metabolic changes in the tumor prior to surgery.

In patients with both osteosarcoma and Ewing's sarcoma, findings of 18-FDG PET have been shown to be predictive of therapeutic response. In one study of 15 patients with either osteogenic sarcoma or Ewing's sarcoma, PET was performed prior to initiation of therapy and again after completion of therapy [44]. PET scans were scored based on tumor to non-tumor FDG uptake ratios (T/NT), and tumor response to therapy was judged by postsurgical histological evaluation. A decrease in T/NT ratio of >30% on PET was found to be associated with a good histological response, whereas an increase in the T/NT ratio or a decrease of <30% was found to be associated with a poor histological response. In a second study of 33 patients with osteogenic sarcoma or Ewing's sarcoma, the ratio of pretherapy/post-therapy SUVs significantly correlated with histological outcome of neoadjuvant therapy [45]. Ultimately, 18-FDG PET may provide the means to assess effectiveness of therapy early after initiation, and therefore direct changes in treatment regimens in order to achieve optimal pre-surgical necrosis.

# References

- 1. Ries LAG, Kosary CL, Hankey BF et al (1999) SEER Cancer Statistics Review 1973-1996. National Cancer Institute, Bethesda, MD
- Greenlee R T, Murray T, Bolden S, Wingo PA (2000) Cancer statistics. CA Cancer J Clin 50:7-33
- Frassica FJ, Sim FH (1988) Pathologenesis and prognosis. In: Sim FH (ed) Diagnosis and management of metastatic bone disease: a multidisciplinary approach. Raven, New York pp 1-6
- Robbins SG, Lane JM, Healey JH et al (1993) Metastatic bone disease. Epidemiology, biology, diagnosis and treatment. In: Lane JM, Healey JH (eds) Diagnosis and management of pathologic fractures. Raven, New York, pp 83-98
- 5. Cancer Stats, Incidence UK. Cancer Research UK, April 2003
- 6. Dorfman HD, Czerniak B, Rotz R et al (2002) WHO Classification of Tumors of Bone: Introduction. In: Fletcher CDM, Unni KK, Mertens F (eds) World Health Organization classification of tumors. pathology and genetics of tumors of soft tissue and bone. IARC, Lyon, pp 226-232
- 7. Greene FL, Page DL, Fleming I D et al (2002) AJCC staging manual, 6th edn. Springer, New York, pp 187-190
- 8. Dorfman HD, Czerniak B (1995) Bone cancers. Cancer 75:203-210
- Sundaram J, McDonald DJ (1990) Magnetic resonance imaging in the evaluation of the solitary tumor of bone. Curr Opin Radiol (CORA) 2:697-702
- Sundaram M, McDonald DJ (1989) The solitary tumor or tumor-like lesion of bone. Top Magn Reson Imaging 1:17-29
- Sundaram M, Maguire MH (1988) Computed tomography or magnetic resonance for evaluating the solitary tumor or tumorlike lesion of bone? Skeletal Radiol 17:393-401
- Lucas JD, O'Doherty MJ, Wong JC et al (1998) Evaluation of fluorodeoxyglucose positron emission tomography in the management of soft-tissue sarcomas. J Bone Joint Surg 80B:441-447
- Lucas JD, O'Doherty MJ, Cronin BF et al (1999) Prospective evaluation of soft tissue masses and sarcomas using fluorodeoxyglucose positron emission tomography. Br J Surg 86:550-556
- Tse N, Hoh C, Hawkins R, Phelps M, Glaspy J (1994) Positron emission tomography diagnosis of pulmonary metastases in osteogenic sarcoma. Am J Clin Oncol 17:22-25
- 15. Franzius C, Daldrup-Link HE, Sciuk J et al (2001) FDG-PET for detection of pulmonary metastases from malignant primary bone tumors: comparison with spiral CT. Ann Oncol 12:479-486
- 16. Franzius C, Sciuk J, Daldrup-Link H E, Jurgens H, Schober O

(2000) FDG-PET for detection of osseous metastases from malignant primary bone tumors: comparison with bone scintigraphy. Eur J Nucl Med 27:1305-1311

- 17. Folpe AL, Lyles RH, Sprouse JT, Conrad III EU, Eary JF (2000) (F-18) fluorodeoxyglucose positron emission tomography as a predictor of pathologic grade and other prognostic variables in bone and soft tissue sarcoma. Clin Cancer Res 6:1279-1287
- Franzius C, Bielack S, Flege, Sciuk J, Jurgens H, Schober O (2002) Prognostic significance of (18) F-FDG and (99m) Tcmethylene diphosphonate uptake in primary osteosarcoma. J Nucl Med 43:1012-1017
- Wolf RE, Enneking WF (1996) The staging and surgery of musculoskeletal neoplasms. Orthop Clin North Am 27:473-481
- Schulte M, Brecht-Krauss D, Heymer B et al (2000) Grading of tumors and tumorlike lesions of bone: evaluation by FDG PET. J Nucl Med 41:1695-1701
- Aoki J, Watanabe H, Shinozaki T et al (2001) FDG PET of primary benign and malignant bone tumors: standardized uptake value in 52 lesions. Radiology 219:774-777
- Aoki J, Watanabe H, Shinozaki T et al (1999) FDG-PET in differential diagnosis and grading of chondrosarcomas. J Comp Tomograph 23:603-608
- Wu H, Dimitrakopoulou-Strauss A, Heichel T O et al (2001) Quantitative evaluation of skeletal tumors with dynamic FDG PET: SUV in comparison to Patlak analysis. Eur J Nucl Med 28:704-710
- 24. Dimitrakopoulou-Strauss A, Strauss L G, Heichel T et al (2002) The role of quantitative (18) F-FDG PET studies for the differentiation of malignant and benign bone lesions. J Nucl Med 43:510-518
- 25. de Santos LA, Murray JA, Ayala AG (1979) The value of percutaneous needle biopsy in the management of primary bone tumors. Cancer 43:735-744
- Fraser-Hill MA, Renfrew DL (1992) Percutaneous needle biopsy of musculoskeletal lesions. 1. Effective accuracy and diagnostic utility. Am J Roentgenol 158:809-812
- Fraser-Hill MA, Renfrew DL, Hilsenrath PE (1992) Percutaneous needle biopsy of musculoskeletal lesions. 2. Cost-effectiveness. AJR AM J Roentgenol 158:813-818
- Stoker DJ, Cobb JP, Pringle JAS (1991) Needle biopsy of musculoskeletal lesions: A review of 208 procedures. J Bone and Joint Surg (Br) 73:498-500
- Hau MA, Kim JI, Kattapuram S et al (2002) Accuracy of CTguided biopsies in 359 patients with musculoskeletal lesions. Skeletal Radiol 31:349-353
- Jelinek JS, Murphey MD, Welker JA et al (2002) Diagnosis of primary bone tumors with image-guided percutaneous biopsy: Experience with 110 tumors. Radiology 223:731-737
- Anderson MW, Temple HT, Dussault RG, Kaplan PA (1999) Compartmental anatomy: relevance to staging and biopsy of musculoskeletal tumors. Am J Roentgenol 173:1663-1671
- 32. Mankin HJ, Lange TA, Spainer SS (1982) The hazards of biopsy in patients with malignant primary bone and soft tissue tumors. J Bone Joint Surg Am 64:1121-1127
- Mankin HJ, Mankin CJ, Simon MA (1996) The hazards of biopsy, revisited. J Bone Joint Surg Am 78:656-663
- Rosenthal DI, Springfield DS, Gebhardt MC, Rosenberg AE, Mankin HJ (1995) Osteoid osteoma: percutaneous radio-frequency ablation. Radiology 197:451-454
- Erickson JK, Rosenthal DI, Zaleske DJ, Gebhardt MC, Cates JM (2001) Primary treatment of chondroblastoma with percutaneous radio-frequency heat ablation: report of three cases. Radiology 221:463-468
- Rosenthal DI, Treat ME, Mankin HJ, Rosenberg AE, Jennings CL (2001) Treatment of epithelioid hemangioendothelioma of bone using a novel combined approach. Skel Radiol 30:219-222
- Fletcher BD, Wall JE, Hanna SL (1993) Effect of hematopoietic growth factors on MR images of bone marrow in children undergoing chemotherapy. Radiology 189:745-751
- Ryan SP, Weinberger E, White KS et al (1995) MR imaging of bone marrow in children with osteosarcoma: Effect of granulocyte colony-stimulating factor. Am J Roentgenol 165:915-920
- 39. Silberzweig JE, Haller JO, Miller S (1992) Ifosfamide: A new cause of rickets. Am J Roentgenol 158:823-824
- 40. Van der Woude HJ, Bloem JL, Holscher HC et al (1994) Monitoring the effect of chemotherapy in Ewing's sarcoma of bone with MR imaging. Skel Radiol 23:493-500
- 41. Holscher HC, Bloem JL, Van der Woude HJ et al (1995) Can MRI predict the histopathological response in patients with osteosarcoma after the first cycle of chemotherapy? Clin Radiol 50:384-390
- 42. Erlemann R, Reiser MF, Peters PE et al (1989) Musculoskeletal

neoplasms: Static and dynamic Gd-DTPA-enhanced MR imaging. Radiology 171: 767-773

- 43. de Baere T, Vanel D, Shapeero LG et al (1992) Osteosarcoma after chemotherapy: evaluation with contrast material-enhanced subtraction MR imaging. Radiology 185:587-592
- 44. Franzius C, Sciuk J, Brinkschmidt C et al (2000) Evaluation of chemotherapy response in primary bone tumors with F18 FDG positron emission tomography compared with histologically assessed tumor necrosis. Clin Nucl Med 25:874-881
- 45. Hawkins D S, Rajendran J G, Conrad E U et al (2002) Evaluation of chemotherapy response in pediatric bone sarcomas by [F-18]-flourodeoxy-d-glucose positron emission tomography. Cancer 94:3277-3284

# **Imaging of Bone Marrow Disorders**

B. Vande Berg, J. Malghem, F. Lecouvet, B. Maldague

Department of Radiology, Cliniques Universitaires St. Luc UCL, Brussels, Belgium

The objectives of this chapter are three-fold. First, the reader will become familiar with the magnetic resonance (MR) appearance of both the normal bone marrow and its variants. Second, a logical analysis of MR imaging (MRI) patterns of elementary lesions will be offered. Finally, the opportunity to analyze MR images of patients with bone metastases, multiple myeloma or lymphoma, with emphasis on lesion detection, will be provided.

# Normal Adult Bone Marrow: Distribution, Composition and MR Appearance

Yellow marrow contains almost exclusively adipocytes (Table 1) which accounts for its high signal intensity on T1-weighted SE and T2-weighted FSE images (Table 2). Hematopoietic cells and adipocytes are present in red marrow and cause intermediate signal intensity of red marrow on T1- and T2-weighted images. Yellow marrow also differs from red marrow in vasculature and in distribution within the body (Table 1).

At birth, red marrow occupies the entire skeleton. Conversion of red to yellow marrow is an age-dependent process in which red marrow is progressively replaced by yellow marrow in the peripheral skeleton. Meanwhile, the proportion of fat cells within the axial red marrow progressively increases. By the age of 25, red and yellow marrow have reached their final adult distribution (red marrow in skull, axial skeleton, ribs, sternum, pelvis and proximal femurs and humeri). This fundamental process explains the distribution of most marrow lesions in the body.

# Normal Variants in MR Appearance of Bone Marrow

Important variations in red-marrow signal intensity and heterogeneity at MRI are encountered.

High signal intensity areas on T1-weighted SE images in axial marrow reflect focal fatty involution, and are irrelevant (Fig. 1). Common vertebral hemangiomas that

Table 1. Anatomy of red and yellow marrow

	Yellow marrow	Red marrow
Chemical composition	80% lipids, 15% water	40% lipids, 40% water
Cellular composition	Fat cells	Hematopoietic and fat cells
Vasculature	Few capillaries	Permeable sinusoids
Distribution	Appendicular skeleton	Axial skeleton

Table 2.	Magnetic resonance	imaging (MRI)	) characteristics of red and yellow marrow

MRI	Red marrow	Yellow marrow
T1-weighted	Intermediate	High
T2-weighted SE	Intermediate	Intermediate/high
STIR, T2-fat saturated	Moderately high	Low
Gradient-echo	Low	Intermediate
Contrast enhancement	Moderate enhancement	No enhancement



**Fig. 1a, b.** Lesion detection. **a** Sagittal T1-weighted spin-echo magnetic resonance (MR) image of the lumbar spine of a 53-year-old woman with breast cancer shows areas of low signal intensity (marrow replacement, *large arrows*) compatible with metastases and areas of high signal intensity (marrow depletion, *white arrows*). **b** On the corresponding fat-saturated T2-weighted spin-echo image, the areas of focal marrow replacement are not visible (better detection of lesion on T1- than on T2-weighted images). The low-signal-intensity areas on the fat-saturated images (*white arrows*) with high signal intensity on T1-weighted images correspond to areas of fatty marrow and lack clinical significance

shows high signal intensity on both T1- and T2-weighted SE images are also irrelevant. They can be confused with relevant marrow lesions if T2-weighted images only are available. The T1-weighted SE images are mandatory to demonstrate the presence of fat, which confirms the benign nature of these lesions.

Ill-delimited areas of moderately decreased signal intensity on T1-weighted images occur frequently in normal marrow and correspond to areas of more cellular red marrow. Confusion with marrow lesions is possible. T2and fat-saturated intermediate-weighted FSE images as well as enhanced T1-weighted SE images may help to differentiate this normal variant from lesions (Table 3). Hematopoietic marrow hyperplasia frequently occurs in middle-aged women. It is defined by the presence of hypercellular marrow in axial marrow and the expansion of red marrow in the appendicular skeleton. It can be idiopathic or associated with heavy smoking habit, long distance running and obesity. It is most generally incidentally discovered on routine knee MR examination, as it shows low to intermediate signal intensity in the distal femoral metaphyses on T1-weighted images in patients older than 25 years. The marrow signal intensity should remain consistent with that of red marrow on other sequences and the adjacent epiphysis should contain fatty marrow. Significant marrow heterogeneity can be encountered in axial skeleton of patients with red-marrow hyperplasia.

#### **Elementary Lesion Patterns**

MRI enables recognition of the decrease in the amount of marrow fat that is concomitant to the presence of an abnormal marrow component in marrow lesions (Figs. 1-3). In other words, the T1-weighted SE sequence with its exquisite sensitivity to the presence of fat enables assessment of the fat/non-fat marrow balance in the medullary cavity. It must be emphasized that the decrease in the amount of marrow fat that can be detected at MRI completely lacks specificity, and the clinical value of MRI resides in its sensitivity for lesion detection and not in its specificity. The T2weighted sequences detect changes in water content that are not systematically altered in marrow lesions. Therefore, the T1-weighted sequence represents the cornerstone in marrow imaging. Elementary lesion patterns at MRI can be classified according to their signal intensity on T1-weighted SE images, bearing in mind that pattern combinations result in a spectrum of focal or diffuse signal alterations (Table 4). However, it must be kept in mind that a strictly normal appearance of the bone marrow on T1- and T2-weighted MR images does not exclude clinically significant alterations of its content.

	Benign heterogeneity	Marrow lesion
T2-weighted images	Low/intermediate signal intensity	High signal intensity
STIR, fat-saturated, T2-weighted images	Low/intermediate signal intensity	High signal intensity
Contrast-enhanced images	No/discrete enhancement	Enhancement
Distribution in bone	Near cortical bone	Any area
Distribution in body	Symmetrical	Variable
Margins	Generally fuzzy	Generally sharp
Centre on T1-weighted images	High signal intensity	Low signal intensity
Bone scintigraphy	No change	Altered uptake
CT image	Normal trabecular bone	Altered trabeculae
Follow-up	No change	Increase in size and number

Table 3. Suggested guidelines that can be used cautiously to differentiate benign heterogeneities of normal red marrow from marrow lesions

	T1-weighted signal intensity	Fat amount	Clinical significance
Depletion	High	Increased	None
Infiltration	Moderately low	Moderately reduced	Reactional lesion
Replacement	Low	Markedly reduced	Primary lesion
Signal void	Black	Absent	Lack of protons

Table 4. Elementary lesions patterns at MRI

#### **Marrow Depletion**

Red-marrow depletion is a pattern characterized on T1weighted SE images by a marked increase in signal intensity compared to adjacent red marrow. It reflects an increase in fat content and a decrease in the non-fat marrow content.

- 1. *Focal* red-marrow depletion: quiescent or healed lesions, Paget disease, and vertebral hemangioma.
- 2. Regional red-marrow depletion: local radiation therapy.
- 3. *Diffuse* red marrow depletion: steroids, chemotherapy, aplastic anaemia.

#### **Marrow Infiltration**

Marrow infiltration is a pattern characterized by a subtle to moderate decrease in marrow signal intensity on T1weighted SE images. Margins are generally indistinct, with a gradual zone of transition toward normal bone marrow. The term "infiltration" suggests that the abnormal marrow component infiltrates or permeates the normal marrow, with some possible residual adipocytes in the lesion. The term bone marrow "edema" is frequently used to characterize marrow infiltration because of its high signal intensity on T2-weighted SE images, consistent with an increase in the free water content. However, hemorrhage or fibrosis can alter marrow signal intensity in a similar manner, and the term edema is frequently inappropriately used.

- 1. *Focal* marrow infiltration: secondary to adjacent lesions (bone fracture, tumour, infection, disc disease, etc.).
- 2. *Diffuse* marrow infiltration: systemic disorders, including anaemia, chronic infection, AIDS, bone marrow cancers. Marrow infiltration by neoplastic cells, interstitial fibrosis or storage disorders can result in a similar marrow picture at MRI.

#### **Marrow Replacement**

Marrow replacement is a pattern characterized by a marked decrease in signal intensity on T1-weighted SE images (Fig. 1). The term "replacement" suggests that the normal marrow component is completely replaced by another tissue, without residual adipocytes. Margins can be sharp, or indistinct if marrow infiltration is also present. On other sequences, signal intensity and enhancement patterns vary greatly but basically reflect the histopathologic changes of the abnormal marrow component. Differentiating focal marrow "replacement" from "infiltration" is important because marrow infiltration is frequently a reaction to changes in an adjacent lesion. Marrow replacement is non-specific but can be a valuable target for biopsy if necessary.

## **Lesion Detection**

For lesion detection, the T1-weighted SE sequence is frequently sufficient, and lesions show low signal intensity on a background of intermediate signal intensity. Fat-saturated T2- or intermediate-weighted images and STIR are also efficient in lesion detection and will also show high signal intensity on a background of low to intermediate signal intensity. In the setting of highly cellular marrow, including in young women and children, these sequences can become mandatory for lesion detection because on T1-weighted images lesions are swamped in the low signal intensity of hypercellular marrow. T2-weighted gradient-echo images are generally not used for lesion detection except in the work-up of patients with multiple myeloma (purely lytic lesions) (Fig. 2). Contrast-enhanced T1-weighted images can be used to differentiate diffusely infiltrated marrow from abnormally cellular albeit normal marrow, which generally shows only moderate signal intensity enhancement (Table 5).

Investigation of relevant body areas is an important feature for optimal lesion detection. Symptomatic areas must be examined in patients with clinical symptoms. In asymptomatic patients, imaging of the spine (sagittal imaging plane) is mandatory. In order to optimize detection, imaging of the pelvis and proximal femurs (coronal T1-weighted SE images) combined with spine imaging offers additional information because the pelvic girdle contains a large amount of hematopoietic marrow (isolated lesions) and because diffuse marrow infiltration is more easily recognized in the pelvis than in the axial skeleton (detection of epiphyseal marrow infiltration) (Fig. 3). Finally, whole-body imaging by performing coronal fat-STIR and T1-weighted images from the skull to the lower limbs certainly adds more information and is becoming more popular than spinal imaging alone.

For lesion characterization, MRI plays little role because it detects a decrease in fat content and does not Lesion detection

Suspicion of meningeal carcinomatosis Benign versus pathological fracture Suspicion of discal/vertebral infection Rarely for detection of focal lesion (fat-saturated T1-weighted spin-echo images) Used for diffuse marrow changes Abnormal intradural enhancement Return to normal signal intensity on T1-W spin-echo images Abscesses?

<sup>a</sup> No value for characterization of multiple focal lesions and limited value for characterization of unique vertebral lesion.



**Fig. 2a, b.** Lesion patterns. **a** Sagittal T1-weighted spin-echo and **b** T2-weighted gradient echo images of the lumbar spine of a patient with lumbar pain show focal marrow changes consisting of areas of low and high signal intensity on T1- and T2-weighted images, respectively (*arrows*). Diffuse marrow changes are also visible on the T1-weighted image as disseminated spots of low signal intensity. MR findings lack specificity but the triad of focal lesion, diffuse infiltration and vertebral fracture suggests multiple myeloma (confirmed at iliac crest biopsy)

demonstrate changes specific for any abnormal component. Nowadays, lesion characterization is rarely the role of medical imaging because of the accuracy of blood tests and the availability of biopsy procedures. The radiological pattern of bone lysis/sclerosis or the combination of radiological, bone scintigraphy and MRI findings contribute more to a presumptive diagnosis that MRI alone.

#### **Imaging Features of Multiple Myeloma**

Radiographs/CT: punched-out lytic lesion, chronic expansive slow-growing lytic lesion, disseminated lytic areas, rarely sclerotic lesions (POEMS)

Bone scintigraphy: generally, no abnormal uptake except in fractures

MRI: focal marrow replacement, diffuse infiltration, replacement and infiltration, pepper-and-salt appearance; generally, high signal intensity on T2-weighted spin-echo and gradient-echo images

#### **Imaging Features in Lymphoma**

Radiographs/CT: permeative lytic, mixed lytic/sclerotic, sclerotic lesions

Bone scintigraphy: increased uptake

MRI: focal marrow replacement, diffuse infiltration. Adjacent soft tissue infiltration with apparently preserved





Fig. 3. Whole-body imaging. a The coronal T1-weighted image of the femur of a 53year-old man with left knee pain and mixed sclerotic lesion in the distal femur on radiographs shows focal marrow replacement (arrow). Given the age of the patient, a primary bone lesion was unlikely and a MR study of the spine and pelvis was performed rather than more precise analysis of the femur lesion (which would have included gadolinium injection). b The sagittal T1weighted MR image of the lumbar spine demonstrated additional focal lesions (arrows) and prevertebral lymph nodes (white arrows). Lymphoma was demonstrated at biopsy of an iliac crest lesion

cortical bone is suggestive. Look for associated lymph nodes (sagittal images of lumbar spine and coronal images of pelvis).

#### **Imaging Features in Metastases**

Radiographs/CT: from lytic to sclerotic

Bone scintigraphy: generally, increased uptake, but also normal or decreased uptake

MRI: focal or diffuse replacement

## **Suggested Reading**

Normal Bone Marrow

- Vogler JB, Murphy WA (1988) Bone marrow imaging. Radiology 168:679-693
- Kricun ME (1985) Red-yellow marrow conversion: its effect on the location of some solitary bone lesions. Skeletal Radiol 14:10-19
- Vande Berg BC, Malghem J, Lecouvet FE, Maldague BE (1998) Magnetic resonance imaging of the normal bone marrow. Skeletal Radiol 27:471-483
- Ricci C, Cova M, Kang YS, Yang A, Rahmouni A, Scott WW Jr, Zerhouni EA (1990) Normal age-related patterns of cellular and fatty bone marrow distribution in the axial skeleton: MR imaging study Radiology 177:83-88
- Baur A, Stabler A, Bartl R, Lamerz R, Scheidler J, Reiser MF (1997) MRI gadolinium enhancement of bone marrow: age-related changes in normals and in diffuse neoplastic infiltration. Skeletal Radiol 26:414-418
- Caldemeyer KS, Smith RR, Harris A, Williams T, Huang Y, Eckert GJ, Slemenda CW (1996) Hematopoietic bone marrow hyperplasia: correlation of spinal MR findings, hematologic parameters, and bone mineral density in endurance athletes. Radiology 198:503-508
- Dawson KL, Moore SG, Rowland JM (1992) Age-related marrow changes in the pelvis: MR and anatomic findings. Radiology 183:47-51
- Moore SG, Bisset GS, 3d, Siegel MJ, Donaldson JS (1991) Pediatric musculoskeletal MR imaging. Radiology 179:345-360
- Hajek PC, Baker LL, Goobar JE, Sartoris DJ, Hesselink JR, Haghighi P, Resnick D (1987) Focal fat deposition in axial bone marrow: MR characteristics. Radiology 162:245-249

#### Abnormal Bone Marrow

- Steiner RM, Mitchell DG, Rao VM, Murphy S, Rifkin MD, Burk DL Jr., Ballas SK, Vinitski S (1990) Magnetic resonance imaging of bone marrow: diagnostic value in diffuse hematologic disorders. Magn Reson Q 6:17-34
- Steiner RM, Mitchell DG, Rao VM, Schweitzer ME (1993) Magnetic resonance imaging of diffuse bone marrow disease. Radiol Clin North Am 31:383-409
- Vande Berg BC, Malghem J, Lecouvet FE Maldague BE (1998) Classification and detection of bone marrow lesions with magnetic resonance imaging. Skeletal Radiol 27:529-545

- Schweitzer ME, Levine C, Mitchell DG, Gannon FH, Gomella LG (1993) Bull's-eyes and halos: useful MR discriminators of osseous metastases. Radiology 188:249-252
- Vande Berg BC, Lecouvet F, Michaux L, Ferrant A, Maldague B, Malghem J (1998) Magnetic resonance imaging of the bone marrow in hematological malignancies. Eur Radiol 3:1335-1344
- Eustace S, Tello R, DeCarvalho V et al (1997) A comparison of whole-body turbo short tau inversion recovery MR imaging and planar technetium 99m methylene diphosphonate scintigraphy in the evaluation of patients with suspected skeletal metastases. Am J Roentgenol 169:1655-1661
- Eustace S, Tello R, DeCarvalho V et al (1998) Whole-body MR imaging versus isotope bone scanning for metastases. Am J Roentgenol 171:519-520

Multiple Myeloma

- Libshitz HI, Malthouse SR, Cunningham D, MacVicar AD, Husband JE (1992) Multiple myeloma: appearance at MR imaging. Radiology 182:833-837
- Lecouvet FE, Vande Berg BC, Michaux L, Malghem J, Maldague B, Jamart J, Ferrant A Michaux JL (1998) Stage III multiple myeloma: clinical and pronostic value of spinal bone marrow MR Imaging. Radiology 209:653-660
- Moulopoulos LA, Dimopoulos MA, Alexanian R, Leeds NE, Libshitz HI (1994) Multiple myeloma: MR patterns of response to treatment. Radiology 193:441-446
- Moulopoulos LA, Dimopoulos MA, Weber D, Fuller L, Libshitz HI, Alexanian R (1993) Magnetic resonance imaging in the staging of solitary plasmacytoma of bone. J Clin Oncol 11:1311-1315
- Rahmouni A, Divine M, Mathieu D, Golli M, Dao TH, Jazaerli N, Anglade MC, Reyes F, Vasile N (1993) Detection of multiple myeloma involving the spine: efficacy of fat-suppression and contrast-enhanced MR imaging. Am J Roentgenol 160:1049-1052
- Stäbler A, Baur A, Bartl R, Munker R, Lamerz R, Reiser MF (1996) Contrast enhancement and quantitative signal analysis in MR imaging of multiple myeloma: assessment of focal and diffuse growth patterns in marrow correlated with biopsies and survival rates. Am J Roentgenol 167:1029-1036
- Vande Berg BC, Lecouvet FE, Michaux L et al (1996) Stage I multiple myeloma: value of MR imaging of the bone marrow in the determination of prognosis. Radiology 201:243-246

#### Lymphoma

- Parker BR, Marglin S, Castellino RA (1980) Skeletal manifestations of leukemia, Hodgkin's disease and non-Hodgkin lymphoma. Semin Roentgenol, 15:302-321.(Abstract)
- Hoane BR, Shields AF, Porter BA Shulman HM (1991) Detection of lymphomatous bone marrow involvement with magnetic resonance imaging [see comments]. Blood 78:728-738
- Rodriguez M (1998) Computed tomography, magnetic resonance imaging and positron emission tomography in non-Hodgkin's lymphoma. Acta Radiol Suppl 417

# **Bone Marrow Disorders**

## A. Stäbler

Department of Radiology, Orthopaedic Clinic München Harlaching, München, Germany

# Introduction

Bone marrow imaging is part of various muskuloskeletal diagnosic tasks including detection and staging of diseases originating in the bone marrow like multiple myeloma, lymphoma, leukaemia and myeloproliferative disorders, imaging of secondary bone marrow involvement (metastasis) in malignant diseases and reactive bone marrow changes due to stress or trauma of bones and joints. Nonneoplastic reasons for changes of bone marrow cellularity are marrow reconversion, which can be caused by various diseases including haemolytic anemias, chronic infection, smoking, and menstruation. These reactive changes must be differentiated from diffuse malignant bone marrow infiltration. By far the most common signal change of bone marrow is related to pathologic load to bone and joints.

Of all the imaging methods used to image bone marrow, such as projection radiography, computed tomography (CT), magnetic resonance (MR) imaging, bone scintigraphy, and positron emission tomography (PET), only MR imaging is capable of direct visualizing bone marrow cells, the remaining methods use indirect ways to image bone marrow pathologies.

The basic principle of bone marrow imaging in MRI consists of determining cellularity and the amount of intracellular and extracellular water in the bone marrow cavity. Both, celluarity and interposition of water is visualized in demonstration of differencies in the fat-water ratio, as hematopoetic and malignant cells consist mainly of water whereas fat cells contain mainly fat.

## **Bone Marrow Anatomy**

In general, hematopoetic, red bone marrow must be differentiated from fatty, yellow bone marrow. In the adults, hematopoesis (red marrow) is situated in the spine, the ribs, the flat bones of the pelvis, scapula, and skull as well as in the proximal (metaphyseal) parts of the humerus and femur. The bone marrow cavity of the peripheral long bones is occupied more or less exclusively by fatty marrow (Fig. 1).



**Fig. 1.** Adult pattern of red and yellow marrow distribution. Cellular (red) marrow in the adult is confined to the spine, the ribs, proximal femurs and humeri, and the skull (dark). The peripheral skeleton contains fatty (yellow) marrow. From Vande Berg BC et al. (1996) Radiology 201:519-523

The primary biochemical difference between red and yellow marrow is the water content or the fat/water ratio (Table 1). Following the skin, the spongious and compact bone and the muscles, the bone marrow is the fourth biggest organ system of the human body compared to the body weight. The mean weight of the bone marrow is 2.6 kg in females and 3.0 kg in men.

Table	1.

	Red bone marrow	Yellow bone marrow
Water	35%-40%	15%
Fat	40%-45%	80%
Protein	15%	5%

# **Development of the Bone Marrow**

At birth, the whole bone marrow cavity including the tip of the fingers contains hematopoetic marrow. With birth, conversion of the red marrow to yellow marrow starts at the peripheral phalanges of the digits of the hands and feet and progresses centrally (Fig. 2). In general marrow conversion is completed around the age of 25 years. Although complete conversion of red to fatty marrow stops around 25 years of age, the number of hematopoetic cells continues to decrease while the number of fatty cells continues to increase throughout life in the hematopoetic marrow, with a gradual change of the fat/water ratio (Fig. 3).

Red marrow produces the cellular components responsible for the oxygen transport (erythrocytes), the immune system (white blood cells), and hemostasis (thrombocytes). These processes, like all metabolic procedures in the human body, are energy consumptive and dependent on the oxygen supply and on the supply of metabolic products. Therefore, blood supply and perfusion are dependent on the level of cellular activity, which is correlated to cellularity in general. This is supported by a decrease of Gadolinium uptake in red marrow with age.

## Principles of MR Imaging of the Bone Marrow

Hematopoetic, red bone marrow in adults 40-70 years of age is composed of approximately 20-25% bone substance, 40-45% fat, and 30-35% cellular marrow (Fig. 4). Bone mineral itself does not add to the MR signal. Signal intensity is dependent on the water and fat components.

Neoplastic bone marrow infiltration and marrow reconversion increase the amount of water containing hematopoetic cells with replacement of fat cells resulting in a change of the fat/water ratio (Fig. 5). All sequences



**Fig 2.** Marrow conversion progresses from peripheral to central in the skeleton. From Vande Berg BC et al. (1996) Radiology 201:519-523



**Fig. 4.** Normal red bone marrow of a 30 year old man. Between the hematopoetic cells fat cells are depicted. The hematopoetic cells occupy more than 50% of the marrow space (Courtesy of Rainer Bartl, MD, Großhadern Clinic)



**Fig. 3.** Percentage cellularity of red marrow with age. A decrease of cellular marrow with age is also found in the spine. From Vande Berg BC et al. (1996) Radiology 201:519-523



**Fig. 5.** Packed marrow in a patient with chronic myeloic leukemia (CML). The whole marrow space is packed with tumor cells. No normal hematopoetic cells and, most important for MR imaging, no fat cells had remained (Courtesy of Rainer Bartl, MD, Großhadern Clinic)

sensitive to water are able to depict bone marrow pathologies with an increased amount of water. Inversion recovery sequences with a short inversion time (STIR) can suppress fat signal by suppressing short T1-relaxation times. Frequency selective fat suppression techniques for PD- and T2-weighted spin echo sequences also highlight the increased signal of water. Due to the elevated signal intensity of fat on T2-weighted fast spin echo images (FSE), FSE-sequences without fat suppression are not useful for bone marrow imaging. T1-weighted sequences are still necessary for imaging the fat component.

Normal hematopoetic bone marrow with near to equal amounts of fat- and water-containing cells is depicted on sequences with subtraction of the transverse magnetization of fat and water, such as opposed phase long TR GRE sequences, with a very low signal intensity. Spin-dephasing, due to effects of magnetic susceptibility, add to the subtraction effect of the fat- and water-magnetization and result in a signal void of normal hematopoetic marrow on opposed-phase GRE images. A shift in the fat-water ratio results in an increase of signal intensity, and therefore bone marrow infiltrating processes can be detected using opposed-phase sequences. GRE sequences are sensitive to pulsation and moving artifacts which can cause problems when using a spine array coil with a large FoV.

Standard imaging of the marrow space should always include T1-weighted images and a sequence sensitive to water. The dedicated techniques described below are only indicated in special clinical settings.

## Whole Body Imaging

To prove or rule out metastasis of the bone, the preferred imaging modality must be able to image the whole skeleton. Bone scintigraphy is widely used, but is an indirect method, showing indiscriminate changes of bone metabolism. MR imaging can detect tumor infiltration of the bone marrow space before metabolic or structural changes of trabecular or cortical bone are depicted on bone scans.

The most important factor to cut down scanner time to acceptable times for whole body MR imaging protocols are the use of appropriate hardware and sequences. Two principle (manufacturer dependent) techniques use either multiple surface coils and cover the whole body by these coils or use a especially adopted circularly polarized body coil. For the spine, a phased array coil should be used. A tendency not to increase the field-of-view but to shorten the magnet and to move the patient through the center of the magnet while acquisition of the data is recognized in the hard ware development. Screening is done by using water sensitive STIR-sequences and FSE images, specificity is improved by adding T1-weighted fast GRE or conventional FSE or SE images (Fig. 6a-c).

Whole body MR imaging with rolling (moving) table detects more lesions than bone scintigraphy. Although bone scans tend to detect more lesions in the ribs and the skull, MRI detects more lesions in the spine and the



Fig. 6a-c. Patient with bronchial cancer and bone metastasis. On bone scan only the lesion to the rib on the right side is obvious (a, b). The STIR image revealed multiple metastasis in the spine (c) (Courtesy Mark Steinborn, MD, Großhadern Clinic)

pelvis. These locations are clinically more important than the ribs and the skull. Scintigraphy grade lesions, especially in the spine, significantly more frequent as uncertain of origin compared to MRI, underlining the low specificity of bone scans. MR imaging offers morphologic information, which can be important for treatment planning and MRI detects tumor-associated complications and organ metastasis in the lung and liver.

The inferiority of MRI in detecting metastases of the skull and ribs is likely to be related to the small and curved marrow spaces of flat bones and motion artifacts in the thoracic region. The clinical relevance of this is given only in cases where these lesions are solitary findings and make the case metastasis-positive, a situation not found in the patient population of our clinic. Whole body MR imaging is only surpassed by whole body PET/CT-imaging concerning sensitivity and specificity in detection and staging of tumors including the bone marrow.

## Gadolinium-enhanced Bone Marrow Imaging

It was believed that bone marrow in general does not enhance with gadolinium, which is not true for red marrow. The average percentage of gadolinium-related signal intensity increase (percentage enhancement) in hematopoetic bone marrow of the spine following a standard dose application of Gadolinium in humans aged 27-35y is  $26.4\%\pm8.6\%$ , for those older than 35y the average percentage enhancement is  $17.5\%\pm7.9\%$ .

When the upper limit for the normal percentage enhancement value is 2 SD above the normal mean percentage enhancement, cut-off values for abnormal gadolinium uptake in bone marrow is 43.6% for those 35 years old and younger and 33.3% for those older than 35 years. In a study on 44 patients with diffuse multiple myeloma and 86 controls the mean level of contrast enhancement is 18% in the control group, 26% in patients with low-grade infiltration, 49% in patients with intermediate-grade infiltration, and 90% in patients with high-grade infiltration. In addition, fat cell content was found to be inversely correlated with contrast enhancement (chi-square test: p < 0.01). Percentage enhancement is a good and reliable tool to estimate marrow cellularity and neovascularization. Although pathologic enhancement values do not differentiate between reactive increase of marrow cellularity and malignant infiltration, tumorrelated enhancement in general is higher than increased percentage enhancement due to reactive changes. Some authors recommend the use of dynamic contrast-enhanced MR imaging for diagnosis of lymphoproliferative diseases and diffuse bone marrow infiltration.

# (U)SPIO-Imaging of the Bone Marrow

There was early recognition of the potential of ultrasmall superparamagnetic iron oxide (USPIO) particles for spe-

cific imaging the bone marrow. Because of the longer blood half-time of the 5-15 nm small particles these preparations reduce signal intensity of bone marrow on MR images. Bone marrow contains parenchymal macrophages of the reticulo endothelial system (RES) with phagocytic activity against ultrasmall superparamagnetic iron oxide (USPIO) particles dependent on the size of the iron oxide particles. 80nm particles (Endorem<sup>®</sup>) are incorporated to a lesser extend than particles of smaller size (<20 nm). The resulting signal drop is more pronounced using the ultra small particles (USPIO).

The diagnostic mechanism of USPIO-imaging of bone marrow disorders is similar to SPIO-imaging of the liver. Only normal bone marrow with RES cells is capable of exhibiting a pronounced signal loss following the application of USPIO particles. In the case of malignant bone marrow infiltration, no signal drop will be observed on post-USPIO images. USPIO imaging of the bone marrow can also be used for evaluation of the blood-bone marrow barrier e.g following radiation. It was shown, that also SPIO decreases the signal intensity of bone marrow. Superparamagnetic iron oxides are taken up by normal and hypercellular reconverted bone marrow, but not by neoplastic bone marrow lesions, thereby providing significantly different enhancement patterns on T2-weighted MR images; thus, superparamagnetic iron oxides are useful to differentiate normal and neoplastic bone marrow and to increase the bone marrow-to-tumor contrast.

# **Diffusion Imaging of the Bone Marrow**

Diffusion-weighted imaging (DWI) is another method of assessing the functional properties of normal and pathological tissues in the bone marrow. Measurement of intravoxel incoherent motion (IVIM) with MR imaging allows for the assessment of the self-diffusion of water in biological tissues. This parameter could serve as a new type of MR image contrast and tissue type characterization. Signal intensity on diffusion-weighted images (DWI) is dependent on the random motion of water protons including extra-, intra- and transcellular motion of water molecules, as well as microcirculation (perfusion). The extracellular component and perfusion contribute most to the signal loss in DWI. The greater the mean free path lengths of the water molecules are, the greater the signal loss achieved with a diffusion-weighted sequence. A diffusion gradient included in the beginning of a diffusion sequence causes dephasing of spins. Stationary tissue is rephased at the end of the sequence by an opposite gradient, so that stationary tissue reveals a high signal intensity. This is due to the T2-weighted component of diffusion weighted sequences. Diffusion induced molecular movement of water protons is responsible for a signal loss since the protons have moved out of plane at the time of the rephasing opposite gradient. While diffusion weighted sequences are sensitive to molecular diffusion, they are also sensitive to the macroscopic motion of the

examined object. This can cause signal loss and artifacts. Cell membranes and organelles cause restricted diffusion. Therefore, free water, e.g. cerebrospinal fluid in the ventricles, has an extremely high diffusion as compared to solid tissue.

On T1- and T2-weighted spin echo, as well as STIR, images and following contrast-enhancement acute benign (osteoporotic) and tumor-related vertebral compression fractures can show similar signal alterations. Signal intensity of bone marrow edema and tumor infiltration is decreased on unenhanced T1-weighted images and increased on (fat-suppressed) proton density (PD)-, T2weighted and STIR images. Especially in cases of severe edema affecting the whole marrow space of a vertebral body, and in patients with known history of cancer differential diagnosis of acute osteoporotic and tumor-related vertebral fracture can be difficult. The diffusionweighted SSFP sequence allows for a reliable differentiation of acute benign and neoplastic vertebral compression fractures. On the diffusion-weighted SSFP sequence the benign fractures exhibited isointense or low signal intensity as compared to surrounding normal bone marrow, whereas metastatic vertebral compression fractures showed high signal intensity. This probably reflects a higher diffusion of water protons in acute benign fractures with bone marrow edema in comparison to vertebral bodies filled with tumor cells.

## **Reactive Changes of Bone Marrow Cellularity**

A replacement of fat cells by tumor cells or non-neoplastic cells in hemolytic disorders with stimulation of the bone marrow cells, increases the amount of water bound protons. This is accompanied by a diffuse decrease of bone marrow signal intensity (SI) on T1-weighted images and an increased SI on STIR images, which can be found in uncontrolled stem cell proliferation in cases of myelodysplastic syndrome or malignant transformation, and stem cell stimulation in hemolytic anemia. Bone marrow cellularity may also be influenced by smoking, menstruation, hemolytic anemia, various drug therapies, such as hematopoetic growth factor during chemotherapy or enzyme therapy e.g. in Gaucher disease, and endurance activities. Hematopoetic activity induced by growth factors can produce changes in bone marrow SI that may simulate bone marrow involvement by musculoskeletal tumors. Hematopoietic bone marrow hyperplasia or reconversion has also been recognized in endurance athletes and around the knees in smokers. Cellularity may also be increased in patients suffering from chronic bacterial infectious spondylitis (Fig. 7a,b). In these cases, MR imaging signal intensity alterations are probably due to reactive bone marrow stimulation. Fat cells are replaced by non-neoplastic stimulated, bone marrow cells, which are necessary for the production of white blood cells in chronic infection.



Fig. 7a, b. Patient with infectious spondylitis. The signal intensity of uninvolved bone marrow is decreased on unenhanced T1-w image (a). Following Gadolinium application strong enhancement is visible at the level of the spondylitis as well in the not involved bone marrow (b). Percentage enhancement was 43%. This reactive change represents marrow stimulation in chronic infection

# Imaging focal Bone Marrow Abnormalities and Metastasis

In the spine, a large field-of-view (500 mm) can only be achieved using a spine phased array coil (Fig. 8). Again, a combination of T1-weighted sequences with a water sensitive STIR sequence is sufficient for most of the clinical situations. At the spine, axial images are important for treatment planning because they show the exact location in the vertebra and the relationship to the pedicles, spinal canal and surrounding soft tissues.

As tumor nodules on T1-weighted spin-echo images become obscured following Gadolinium application, frequency selective fat-suppressed sequences are necessary to disclose focal lesions, especially when diffuse bone marrow infiltration is also present (Fig. 9a,b).

Signal intensity of GRE sequences is also dependent upon magnetic susceptibility, allowing for differentiating tumor infiltration with and without trabecular destruction. This situation can be found in tumor infiltration of breast cancer and in multiple myeloma. The subtraction of fat and water signal on opposed GRE sequences provides a perfect background with low signal intensity to detect even very small tumor foci.



Fig. 8. Multiple metastasis from bronchial carcinoma. On the STIR-image multiple metastasis are outlined with high signal intensity. The location of the metastasis, which is of risk for a neurologic complication by compressing the spinal cord, is easily recognized

## **Imaging Diffuse Bone Marrow Abnormalities**

When there are diffuse abnormalities of the bone marrow signal in hematologic neoplasias and myeloproliferative diseases but no focal disease is present, a pathologic signal intensity of the bone marrow can be overlooked. In this situation, a homogenous diffuse decrease of signal intensity over all vertebral bodies on T1-weighted spinecho images results from a homogenous replacement of fat cells by cellular marrow or an accumulation of iron in the bone marrow in hemolytic disorders.

In the presence of diffuse neoplastic bone marrow infiltration or bone marrow stimulation, low homogenous SI on T1-weighted images is seen, in addition to increased SI on STIR-images. The percentage enhancement following Gadolinium injection is increased (Fig. 10a,b). After US-PIO administration, a decrease of SI is reduced or absent.



Fig. 10a, b. Diffuse neoplastic bone marrow infitration in a patient with breast cancer. On unenhanced T1-weighted image a diffuse low SI is present in all vertebrae (a). Gadolinium enhancement is heavily increased indicating the diffuse tumor infiltration (b)



**Fig.9a, b.** 60-year-old woman with vertebral metastasis due to breast cancer. SI of the whole vertebral body is decreased on unenhanced T1-weighted image (**a**). After Gadolinium injection, frequency selective fat suppression creates a low intensity background to highlight the enhancing metastasis (**b**)

# **Multiple Myeloma**

Multiple myeloma is characterized by bone marrow infiltration with neoplastic plasma cells. Except for non-secretory and Bence Jones plasmacytoma, these cells produce monoclonal immunglobulins, recognizable in serum electrophoresis. Survival times show great variability. After diagnosis, patients live only few months or survive up to ten years in cases of smoldering myeloma. Diagnosis and staging is established by laboratory parameters, bone marrow biopsies and plain radiographs using the Durie and Salmon classification. MR imaging is now routinely added to the staging process.

When minimal plasma cell infiltration is present, this is usually accompanied by a normal or even increased amount of marrow fat cells. In malignant tumors with diffuse bone marrow infiltration, there is rapid displacement of fat cells by tumor cells. At the beginning of interstitial tumor infiltration in multiple myeloma, monoclonal plasma cells arrange themselves in such a way as to not displace the fat cells. Apparently, these cells produce factors which inhibit normal hematopoesis, thus increasing the relative fat component. Therefore, despite tumor cell infiltration and replacement of hematopoetic cells, bone marrow fat may be normal or even increased without signal alterations on MR imaging. As long as there is no critical shift in the water to fat ratio of the bone marrow, myeloma remains undetected in MR imaging.

In *diffuse* plasma cell infiltration, no contrast to uninvolved bone marrow is present. Patients with a diffuse infiltration pattern in multiple myeloma are generally in stage II or III disease which is prognostically unfavorable.

*Focal* marrow involvement results in replacement of normal bone marrow by neoplastic plasma cells with or without trabecular destruction. Myelomatous foci in general show low signal intensity on T1-weighted spin-echo images, but they can be isointense or hyperintense compared with surrounding bone marrow. On opposed GRE and STIR images, focal plasmocytoma nodules exhibit a high or very high signal intensity with pronounced increase of signal intensity, if Gadolinium is added. Because T2-weighted fast-spin-echo sequences reveal fat with high signal intensity, they are not useful for the assessment of myeloma. However, when fat saturation is added to the fast spin-echo T2-weighted sequence, the high signal intensity of fat is removed and the sequence becomes useful for assessing myeloma.

*Focal* myeloma nodules *within diffuse* infiltration of the bone marrow may be difficult to diagnose on T1-weighted spin-echo sequences with low intensity nodules isointense or slightly hypointense to the diffuse plasma cell infiltration. On opposed phase GRE images, however, myelomatous foci are clearly delineated. As tumor nodules on T1-weighted spin-echo images become obscured following Gadolinium application, frequency selective fat-suppressed sequences are necessary to reveal focal lesions in addition to diffuse bone marrow infiltration. The "salt-and-pepper" pattern is characterized by an irregular bone marrow structure with irregular areas of high and low signal intensity on T1-weighted spin-echo images, similar to tumor infiltration. Hyperintense areas on T1-weighted spin-echo images represent focal fat deposits, whereas hypointense areas correlate with hematopoetic bone marrow with low interstitial plasma cell infiltration. The "salt-and-pepper" pattern correlates with a stage of low activity of the disease, most of the patients with "salt and pepper" pattern are stage I according to the classification of Durie and Salmon and do not need systemic therapy.

Bone marrow biopsy is essential for diagnosis of multiple myeloma and gives direct proof for atypical plasma cells. Because of the small size of the biopsy sample, however, the result is not always representative of the entire bone marrow, especially in cases of nodular involvement, in which the correlation of bone marrow biopsy and MRI is low. Laboratory parameters, such as serumparaprotein,  $\beta_2$ -microglobulin and the labeling index, are indirect criteria, but correlate well with tumor mass and survival times. In cases of non-secretory and Bence Jones plasmacytoma, these parameters may be negative. When "solitary" plasmacytoma is present, MR imaging can detect or exclude additional marrow abnormalities.

# Differentiation of acute osteoporotic and tumor-related vertebral fractures

On T1- and T2-weighted spin echo as well as STIR images and following contrast enhancement, acute benign and neoplastic vertebral compression fractures can show similar signal alterations. Bone marrow edema as well as tumor infiltration exhibit hypointense signal on non-enhanced T1-weighted spin echo-images and increased signal intensity on T2-weighted spin echo or STIR-images. Especially, if the whole vertebral body is affected due to bone marrow edema and if a malignant tumor is known in the patient's medical history, differential diagnosis can be difficult. There exist morphologic criteria for differentiation osteoporotic from malignant vertebra compression fractures and specific imaging findings including the intraosseous vacuum phenomenon on CT, the fluid sign on MRI and signal changes on opposed phase gradient echo imaging and diffusion weighted imaging.

Morphologic imaging findings characteristic for tumor related vertebral fractures are a convex posterior cortex, diffuse low signal-intensity on T1-weighted images due to a loss of fat signal, lesion extension into the pedicle, high or inhomogenous signal-intensity following Gadolinium, and a high signal-intensity of the bone marrow on T2-weighted or STIR images. Findings indicative for osteoporotic fractures are retropulsion of a bone fragment, preservation of some fat signal on T1-weighted images, a return to normal signal-intensity after Gadolinium, a horizontal bandlike signal pattern, and isointense bone marrow signal on T2-weighted or STIR images.

Intravertebral vacuum phenomenons following vertebral fractures are inversely correlated to bone mineral density and are a sign of a benign vertebral fracture. In fractured vertebral bodies, the fluid sign is adjacent to the fractured end plates and exhibits signal intensity isointense to that of cerebrospinal fluid. The fluid sign is linear or focal and is significantly associated with osteoporotic fractures. In fact, the intraosseous vacuum phenomenon on CT in benign osteoporotic vertebral compression fractures is linked to the fluid sign on MRI and reflects the same pathoanatomic phenomenon and mechanism: different to a neoplastic infiltration with neovascularity, an osteoporotic spongious bone with increased amount of marrow fat has only few vessels contributing to nutrition of the bone. Following fracture some of these vessels will disrupt due to the mechanical compression and by lying in a prone position and by the paraspinal muscles in part a distraction of the fractured und compressed spongious bone will take place with the result of a cavity. By distraction a vacuum will develop. In osteoporotic bone this cavity is quickly filled not by tissue but with nitrogen gas responsible for the vacuum phenomenon on the CT scans. With time also fluid will show up in this cavity creating a fluid sign on the MR images.

Using opposed phase GRE-sequence with increased repetion time, tumor-related vertebral fractures show high signal intensity compared to acute osteoporotic fracture, which have intermediate to low signal intensity. On the diffusion-weighted SSFP sequence, the benign fractures exhibited isointense or low signal intensity as compared to surrounding normal bone marrow, whereas metastatic vertebral compression fractures showe a high signal intensity. This probably reflects a higher diffusion of water protons in acute benign fractures with bone marrow edema in comparison to vertebral bodies filled with tumor cells.

# Differentiation of transient bone marrow edema-syndrome and avascular necrosis

Areas of subchondral epiphyseal bone marrow edema adjacent to weight bearing joints have to be differentiated in transient bone marrow edema syndrome, subchondral farcture and avascular bone necrosis (AVN) (Figs. 11-13). An AVN lesion is typically a well-demarcated epiphyseal area of variable signal intensity, often associated with a double-line signal intensity pattern. A transient bone marrow edema lesion is ill-delimited with low-signal-intensity in the epiphyseal area on T1weighted images with high-signal-intensity on water sensitive images. Contrast-enhanced MR images may increase diagnostic confidence by showing homogeneous hypervascularization in bone marrow edema lesions and by depicting hypovascular marrow areas in AVN lesions.



Fig. 11a, b. Transient bone marrow edema syndrome (PDw fatsat image). Progression and migration of the bone marrow edema form the medial condylus ( $\mathbf{a}$ ) to the lateral condylus of the left knee ( $\mathbf{b}$ )



Fig. 12. Bone marrow edema of the medial condylus due to insufficiency fracture and overload (PDw fatsat image)



Fig. 13a, b. Focal bone necrosis in medial osteoarthrosis and meniscal desinteration. T1w image (a) and PDw fatsat image (b)

# **Suggested Readings**

- Allison JW, James CA, Arnold GL, Stine KC, Becton DL, Bell JM (1998) Reconversion of bone marrow in Gaucher disease treated with enzyme therapy documented by MR. Pediatr Radiol 28:237-240
- Antoch G, Vogt FM, Freudenberg LS, Nazaradeh F, Goehde SC, Barkhausen J, Dahmen G, Bockisch A, Debatin JF, Ruehm SG (2003) Whole-body dual-modality PET/CT and whole-body MRI for tumor staging in oncology. JAMA 290:3199-3206
- Avrahami E, Tadmor R, Dally O, Hadar H (1989) Early MR demonstration of spinal metastases in patients with normal radiographs and CT and radionuclide bone scans. J Comput Assist Tomogr 13:598-602
- Baker LL, Goodman SB, Perkash I, Lane B, Enzmann DR (1990) Benign versus pathologic compression fractures of vertebral bodies: Assessment with conventional spin-echo, chemical shift, and STIR MR imaging. Radiology 174:495-502
- Bartl R, Frisch B, Diem H et al (1991) Histologic, biochemical, and clinical parameters for monitoring multiple myeloma. Cancer 68:2241-2250
- Baur A, Bartl R, Pellengahr C, Baltin V, Reiser M (2004) Neovascularization of bone marrow in patients with diffuse multiple myeloma: a correlative study of magnetic resonance imaging and histopathologic findings. Cancer 101:2599-2604
- Baur A, Stabler A, Arbogast S, Duerr HR, Bartl R, Reiser M (2002) Acute osteoporotic and neoplastic vertebral compression fractures: fluid sign at MR imaging. Radiology 225:730-735
- Baur A, Stäbler A, Brüning R, Bartl R, Krödel A, Reiser M, Deimling M (1998) Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic compression fractures. Radiology 207:349-356
- Caldemeyer KS, Smith RR, Harris A et al (1996) Hematopoietic bone marrow hyperplasia: correlation of spinal MR findings, hematologic parameters, and bone mineral density in endurance athletes. Radiology 198:503-508
- Cuenod CA, Laredo JD, Chevret S, Hamze B, Naouri JF, Chapaux X, Bondeville JM, Tubiana JM (1996) Acute vertebral collapse due to osteoporosis or malignancy: appearance on unenhanced and gadolinium-enhanced MR images. Radiology 199:541-519
- Daffner RH, Lupetin AR, Dash N, Deeb ZL, Sefczek RJ, Schapiro RL (1986) MRI in the detection of malignant infiltration of bone marrow. AJR Am J Roentgenol 146:353-358
- Daldrup HE, Link TM, Blasius S et al (1999) Monitoring radiationinduced changes in bone marrow histopathology with ultrasmall superparamagnetic iron oxide (USPIO)-enhanced MRI. J Magn Reson Imaging 9:643-652
- Daldrup-Link HE, Rummeny EJ, Ihssen B, Kienast J, Link TM (2002) Iron-oxide-enhanced MR imaging of bone marrow in patients with non-Hodgkin's lymphoma: differentiation between tumor infiltration and hypercellular bone marrow. Eur Radiol 12:1557-1566
- Deely DM, Schweitzer ME (1997) MR imaging of bone marrow disorders. Radiol Clin North Am 35:193-212
- Dunnill MS, Anderson JA, Whitehead R (1967) Quantitative histological studies on age changes in bone. J Path Bact 94:275-291
- Durie BGM, Salmon SE (1975) A clinical staging system for multiple myeloma. Correlation of measured myeloma cell mass with presenting clinical features, response to treatment, and survival. Cancer 36:842-854
- Engelhard K, Hollenbach HP, Wohlfart K, von Imhoff E, Fellner FA (2004) Comparison of whole-body MRI with automatic moving table technique and bone scintigraphy for screening for bone metastases in patients with breast cancer. Eur Radiol 14:99-105
- Frager D, Elkin C, Swerdlow M, Bloch S (1988) Subacute osteoporotic compression fracture: misleading magnetic resonance appearance. Skeletal Radiol 17:123-126

- Fletcher BD, Wall JE, Hanna SL (1993) Effect of hematopoietic growth factors on MR images of bone marrow in children undergoing chemotherapy. Radiology 189:745-751
- Ghanem N, Kelly T, Altehoefer C, Winterer J, Schafer O, Bley TA, Moser E, Langer M (2004) Ganzkörper-MRT vs. Skelettszintigraphie bei der Detektion ossärer Metastasen solider Tumoren. Radiologe 44:864-873
- Hundt W, Petsch R, Helmberger T, Reiser M (2000) Effect of superparamagnetic iron oxide on bone marrow. Eur Radiol 10:1495-1500
- Kattapuram SV, Khurana JS, Scott JA, el-Khoury GY (1990) Negative scintigraphy with positive magnetic resonance imaging in bone metastases. Skeletal Radiol 19:113-116
- Kyle RA (1983) Long-term survival in multiple myeloma. New Engl J Med 308:314-316
- Kyle RA (1975) Multiple myeloma review of 869 cases. Mayo Clin Proc 50:29-39
- Lauenstein TC, Freudenberg LS, Goehde SC, Ruehm SG, Goyen M, Bosk S, Debatin JF, Barkhausen J (2002) Whole-body MRI using a rolling table platform for the detection of bone metastases. Eur Radiol 12:2091-2099
- Latour LL, Svoboda K, Mitra PP, Sotak CH (1994) Time-dependent diffusion of water in a biological model system. Proc Natl Acad Sci 91:1229-1233
- Le Bihan D, Breton E, Lallemand D, Grenier P, Cabanis E, Laval-Jeantet M (1986) MR imaging of intravoxel incoherent motions: Application to diffusion and perfusion in neurologic disorders. Radiology 161:401-407
- Mentzel HJ, Kentouche K, Sauner D, Fleischmann C, Vogt S, Gottschild D, Zintl F, Kaiser WA (2004) Comparison of wholebody STIR-MRI and (99m)Tc-methylene-diphosphonate scintigraphy in children with suspected multifocal bone lesions. Eur Radiol [Epub ahead of print]
- Piney A (1922) The anatomy of the bone marrow. Br Med J 2:792-795
- Rahmouni A, Divine M, Mathieu D et al (1993) Detection of multiple myeloma involving the spine: efficacy of fat-suppression and contrast-enhanced MR imaging. AJR Am J Roentgenol 160:1049-1052
- Rahmouni A, Montazel JL, Divine M, Lepage E, Belhadj K, Gaulard P, Bouanane M, Golli M, Kobeiter H (2003) Bone marrow with diffuse tumor infiltration in patients with lymphoproliferative diseases: dynamic gadolinium-enhanced MR imaging. Radiology 229:710-717
- Ryan SP, Weinberger E, White KS et al (1995) MR imaging of bone marrow in children with osteosarcoma: effect of granulocyte colony-stimulating factor. AJR Am J Roentgenol 165:915-920
- Schmidt GP, Baur-Melnyk A, Tiling R, Hahn K, Reiser MF, Schoenberg SO (2004) Hochauflösendes Ganzkörpertumorstaging unter Verwendung paralleler Bildgebung im Vergleich zur PET-CT. Erste Erfahrungen auf einem 32-Kanal-MRT-System. Radiologe 44:889-898
- Seiderer M, Stäbler A, Wagner H (1999) MRI of bone marrow: opposed-phase gradient-echo sequences with long repetition time. Eur Radiol 9:652-661
- Seneterre E, Weissleder R, Jaramillo D et al (1991) Bone marrow: ultrasmall superparamagnetic iron oxide for MR imaging. Radiology 179:529-533
- Stäbler A, Baur A, Bartl R, Munker R, Lamerz R, Reiser MF (1996) Contrast enhancement and quantitative signal analysis in MR imaging of multiple myeloma: assessment of focal and diffuse growth patterns in marrow correlated with biopsies and survival rates. AJR Am J Roentgenol 167:1029-1036
- Stäbler A, Doma AB, Baur A, Krüger A, Reiser MF (2000) Quantitative Assessment of Reactive Bone Marrow Changes in Infectious Spondylitis. Radiology, Radiology 217:863-868
- Stäbler A, Krimmel K, Seiderer M, Gartner C, Fritsch S, Raum W. Stäbler A, Krimmel K, Seiderer M, Gärtner C, Fritsch S, Raum W (1992) Kernspintomographische Differenzierung osteo-

porotisch und tumorbedingter Wirbelkörperfrakturen: Wertigkeit von subtraktiven Gradientenechosequenzen mit verlängerter Repetitionszeit, STIR Sequenzen und Gd-DTPA. Fortschr Röntgenstr 157:215-221

- Stäbler A, Schneider P, Link TM, Schöps P, Springer OS, Dürr HR, Reiser M (1999) Intravertebral vacuum phenomenon following fractures: CT study on frequency and etiology. J Comput Assist Tomogr 23:976-980
- Steinborn MM, Heuck AF, Tiling R, Bruegel M, Gauger L, Reiser MF (1999) Whole-body bone marrow MRI in patients with metastatic disease to the skeletal system. J Comput Assist Tomogr 23:123-129
- Steiner RM, Mitchell DG, Rao VM, Schweitzer ME (1993) Magnetic resonance imaging of diffuse bone marrow disease. Radiol Clin North Am 31:383-409
- Tunaci M, Tunaci A, Engin G et al (1999) Imaging features of thalassemia. Eur Radiol 9:1804-1809
- Vande Berg BE, Malghem JJ, Labaisse MA, Noel HM, Maldague BE (1993) MR imaging of avascular necrosis and transient marrow edema of the femoral head. Radiographics 13:501-520
- Vande Berg BC, Malghem JJ, Lecouvet FE, Jamart J, Maldague BE (1999) Idiopathic bone marrow edema lesions of the femoral

head: predictive value of MR imaging findings. Radiology 212:527-535

- Vande Berg BC, Michaux L, Scheiff JM et al (1996) Sequential quantitative MR analysis of bone marrow: differences during treatment of lymphoid versus myeloid leukemia. Radiology 201:519-523
- Vanel D, Missenard G, Le Cesne A, Guinebretiere JM (1997) Red marrow recolonization induced by growth factors mimicking an increase in tumor volume during preoperative chemotherapy: MR study. J Comput Assist Tomogr 21:529-531
- Vogler JB, Murphy WA (1988) Bone marrow imaging. Radiology 168:679-693
- Wilson AJ, Murphy WA, Hardy DC, Totty WG (1988) Transient osteoporosis: transient bone marrow edema? Radiology. 167(3):757-60
- Wismer GL, Rosen BR, Buxton R, Stark DD, Brady TJ (1985) Chemical shift imaging of bone marrow: preliminary experience. AJR Am J Roentgenol 145:1031-1037
- Yuh WTC, Zachar CK, Barloon TJ, Sato Y, Sickels WJ, Hawes DR (1989) Vertebral compression fractures: Distinction between benign and malignant causes with MR imaging. Radiology 172:215-218

# Metabolic and Systemic Bone Diseases\*

J. Freyschmidt

Department of Radiology, Central Hospital, Bremen, Germany

# Introduction

Metabolic bone diseases represent a fascinating nosologic group, caused by inborn as well as acquired disturbances of bone metabolism. Any part of the complex bone metabolism may be involved, resulting in various clinical and radiological signs and symptoms. I will focus this course on hyperparathyroidism, osteomalacia, chronic renal failure and hemodialysis-associated diseases, and osteitis deformans.

# Hyperparathyroidism

Hyperparathyroidism (HPT) is defined as an increased level of parathormone and parathormone peptides in the serum. It can be devided into three types:

- Primary hyperparathyroidism (PHPT) may be the result of diffuse hyperplasia, adenomas or carcinomas. In all cases the serum calcium level is increased. The annual incidence of PHPT is calculated to be 25-28 cases per 100 000.
- Secondary hyperparathyroidism (SHPT) is the result of chronic renal failure, rarely from malabsorption. If the production of active vitamin D in disordered kidneys is reduced or extinguished, intestinal calcium absorption is disturbed with subsequent hypocalcemia and stimulation of the parathyroid glands. Simultaneously phosphate excretion is reduced with hyperphosphatemia leading to regulatory hypocalcemia (calcium homeostasis). This stimulates the parathyroid glands, which in turn increases the level of circulating parathyroid hormone.
- Tertiary hyperparathyroidism is the result of longstanding secondary hyperparathyroidism due to chronic renal failure. In tertiary hyperparathyroidism, the parathyroid glands function autonomously.

#### Radiology of hyperparathyroidism

To understand the radiology of HPT, it is important to know that only osteoblasts have classic receptors for parathormone. In contrast, osteoclasts are stimulated by intercellular messengers (e.g. interleukin-1, prostaglandins, tumor necrosis factor) expressed by osteoblasts.

Thus bone-resorbing osteoclasts are stimulated by parathormone only via osteoblasts. This means that an elevated level of parathormone – at least in an early phase of HPT – generally promotes bone mass, especially that of trabecular bone. Only in later stages of the disease when the parathormone level has been increased over a longer period trabecular bone resorption may occur. Because HPT today is usually detected early through increased levels of serum calcium, we observe more patients with more bone and fewer patients with less bone, as in former times. Patients with early PHPT consult the physician not because of musculoskeletal complaints but because of symptoms of nephrocalcinosis, hypertension, arrhythmia, coronary heart disease, neuromuscular disturbances as well as mental alterations (e.g. reduction of drive, disturbance of memory). In follow-up laboratory studies, elevated serum calcium levels are usually found. If the patient then is referred to the radiologist, the latter must be aware of the various radiologic symptoms of PHPT.

The described pathogenetic mechanisms are congruent with histopathologic findings in PHPT [1].

In 90% of patients with PHPT the trabecular bone of the *iliac crest* is intact. The number of osteoclasts indeed is increased but their depth of resorption is less than normal. The number of BMU (bone mineral units) is also increased, meaning that the number of osteoblasts is elevated, producing more osteoid matrix, but without a disturbance of mineralization. In about 50% of all cases of PH-PT, there is additional endosteal fibrosis.

These results demonstrate that bone mass in those patients is increased. Only in 4% of all cases, advanced bone resorption, formation of primitive woven bone and brown tumors can be observed.

While the trabecular bone mass of the iliac crest and spine in early stages of PHPT (and also in SHPT) may be

<sup>\*</sup> This chapter originally appeared in: von Schulthess GK, Zollikofer Ch L (2001) Musculoskeletal Diseases - Diagnostic Imaging and Interventional Techniques. Springer-Verlag Italia, Milan

increased, cortical bone resorption may take place simultaneously. This is best visualized with X-rays of the hands (magnification technique, or using high-resolution films for mammography). I believe that the preference of resorption of cortical bone is a problem of vascular perfusion, especially in the subperiosteal compartment.

Classic signs of advanced PHPT (Fig. 1) include:

- Rarefication of trabecular bone (especially metaphyses and spine).
- Wispy and woolly coarsening of trabeculae, if the primitive woven bone ossifies.
- Tunneling and striation of the compact bone, especially of the tubular bones of the hand skeleton, by intracortical resorption.
- Periosteal ("subperiosteal") and subtendinous resorption, especially at the tubular bones of the hand skeleton. The outer surface of the cortex appears irregular and sometimes spiculated.
- Acro-osteolysis, especially in the terminal tufts of the hand skeleton.
- Endosteal resorption (endosteal and periosteal resorption lead to a thinned cortex).
- Osteolysis by brown tumors.
- Patchy osteosclerosis, especially in the skull, pelvis, vertebral end plates and metaphyseal sites of the long bones.

*Scintigraphic bone scans* of PHPT reveal an increased tracer uptake, sometimes as a so-called superscan.

The *hand skeleton* is the primary radiologic test region, and yields positive results in 30%-50% of all cases of PH-PT (Fig. 1). The higher rate of detection of pathologic findings in PHPT by X-ray of the hand skeleton compared with histopathologic results of the iliac crest may be explained by the fact that cortical bone resorption may al-



Fig. 1. PHPT (X-ray of the second to fourth fingers in magnification technique). Note the typical signs of PHPT: cortical borders of the terminal tufts have vanished: coarsened trabeculae with a woolly aspect; thinned cortices with a slightly spiculated outline, especially of the radial margins of the middle phalanges; osteolysis under the radial distal cortex of the second proximal phalanx (brown tumor); erosion at the metacarpal heads (by crystal-deposition synovitis and/or collapse because of reduced resistance against mechanical stress)

ready be taking place when the trabecular structures of the iliac crest are still normal.

A total of 6 possible radiologic patterns of PHPT can be distinguished [2]:

- 1. No changes; normal bone scan.
- 2. Slight or moderate diffuse or patchy osteosclerosis (skull, spine, innominate bones, ribs, femora, long bones) without resorptive changes at the hand skeleton. An early increase of bone mass is best demonstrated by quantitative computed tomography (QCT).
- 3. Osteosclerosis with additional resorptive changes at the hand skeleton (so-called late early stage).
- 4. Slight osteopenia (QCT), combined with resorptive changes at the hand skeleton (early advanced stage of PHPT).
- 5. The classic form of PHPT, as described by von Recklinghausen, includes massive "osteoporosis", thinned and laminated cortex, woven-bone phenomenon, brown tumors, spontaneous fractures.
- 6. A contradictory pattern with osteosclerosis, bone resorption and repaired brown tumors, caused by a condensorlike course of the disease, by inbleeding of thyroid adenomas and by transient inhibition of hormone secretion. In patterns 2-4, brown tumors may be possible.

#### Signs of secondary hyperparathyroidism

Typical SHPT is characterized by a mixture of osteomalacia with HPT, mostly observed in chronic renal failure, chronic hemodialysis, malabsorption or pancreatic insufficiency.

# Osteomalacia

Osteomalacia (OM) is characterized by an inhibition of mineralization of the osteoid matrix. Because of the steady remodelling of bone, under- or nonmineralized osteoid increases and normal bone decreases.

The etiologic spectrum of osteomalacia is extremely broad and cannot be discussed here. However, I would like to draw the reader's attention to a generally unknown form, *oncogenic OM* [3]. It is a paraneoplastic syndrome in which vitamin D-resistant OM or rickets occurs due to the presence of a bone or soft-tissue tumor (e.g. hemangioma, osteoblastoma, osteosarcoma, fibrous dysplasia) as well as to a reactive or proliferative condition. The metabolic abnormality is usually completely reversed when the tumor is identified and removed. Patients with oncogenic OM have low serum phosphorus levels, variably elevated alkaline phosphatase and increased urinary phosphate excretion; 1,25-dihydroxycholecalciferol, the active metabolite of vitamin D, is low or undetectable.

Radiologically the bone density, especially that of trabecular bone, decreases. Because of the unmineralized or nonmineralized osteoid matrix, the trabecular structures loose their sharp delineation and they appear like groundglass or if they had been treated with India rubber (Fig. 2). **Fig. 2.** Osteomalacia. The bony structures appear as if they had been treated with India rubber (groundglass phenomenon)



Because of the reduced strength of the bone, bowing of the long bones may occur as well as an acetabular protusion and basilar impression. Dense lines adjacent to the vertebral endplates are the result of insufficiency fractures and excessive callus formation (rugger-jersey appearance). Another typical sign of OM is insufficiency fractures (pseudofractures, Looser zones), which are often symmetric and typically located in the innominate bones, scapulae, femoral necks, proximal ulnae, ribs etc. They can best be demonstrated by scintigraphic bone scans. Especially in elderly women we observed pseudotumors in the innominate bones caused by epiphenomena of previous stress fractures. The stress fractures on the other hand were the result of unstable pseudofractures in the lateral portion of the sacrum.

The radiologic appearance of OM may be complicated by SHPT.

# Chronic Renal Failure and Dialysis-associated Diseases

During the past 15 years hemodialysis and renal transplants have enabled patients to live longer. However, musculoskeletal changes have become manifest in these patients, and are attributable to the complex therapy (e.g. highly effective metabolites of vitamin D, aluminium-containing phosphate binders, the dialysis procedure itself, steroids). For the radiologist, the classic renal osteodystrophy, aluminium-related changes,  $\beta_2$ -microglobulin amyloidosis and bone infarction and necrosis are of main interest.

## **Classic Renal Osteodystrophy**

Classic renal osteodystrophy occurs when the glomerular filtration rate drops below 30%. Renal osteodystrophy represents a mixture of secondary hyperparathyroidism (SHPT) and osteomalacia. An excellent review of the complex mechanisms of renal osteodystrophy has been published by Hruska and Teitelbaum [3]. The radiologic test region is the hand skeleton, where initial changes of SHPT can be best demonstrated. Radiology is even more reliable than are biochemical parameters. Especially in patients with insufficiently treated hyperphosphatemia, tumorous soft-tissue calcification (of bursae, para-articular structures) may occur.

#### **Aluminium-related Changes**

An overload with aluminium is common in patients on chronic hemodialysis (high dialysate aluminium concentration, intake of phosphate binders, increased intestinal resorption) but also by excessive oral intake of aluminium-containing phosphate binders (antacids) for management of peptic ulcer disease. Because aluminium is readily bound to transferrin in the plasma, it is not removed with dialysis even if the dialysate is aluminium-free. Aluminium absorption is significantly increased in the gut after oral aluminium load in uremic patients. Because the kidney is the major organ for elimination of absorbed aluminium, aluminium overload is common in patients with chronic renal failure. Predominant clinical features of aluminium toxicity include dementia, myopathy, anemia and bone disease. The latter includes osteopenia with multiple non-traumatic fractures, especially of the ribs, long bones, pelvis, hips, and spine [5]. The presence of more than 3 fractures without evidence of healing, particularly in the upper ribs, in combination with low alkaline phosphatase activity, low serum calcium, low or normal serum parathormone levels and an aluminium serum level more than 300 ng/ml is highly suspicious of aluminium intoxication [6]. Scintigraphy shows poor tracer uptake in the bone with high soft-tissue activity; the fractures may only be faintly detectable. There are two reasons for these phenomena:

- Aluminium deposited at the mineralization front slows osteoblastic and osteoclastic activity with the consequence of osteomalacia.
- Aluminium deposition in the thyroid gland leads to inhibition of their activity, resulting in hypoparathyroidism.

Both factors and other complex mechanisms finally cause the so-called adynamic bone disease [4].

The unequivocal diagnosis of aluminium bone disease is made by bone biopsy, stained for aluminium. Prompt treatment with deferoxamine usually leads to an improvement of symptoms and to the healing of the fractures.

## β<sub>2</sub>-Microglobulin Amyloidosis

After 10 and 15 years of hemodialysis,  $\beta_2$ -microglobulin deposition is observed in articular and periarticular structures in 65% and 100% of patients, respectively.

 $\beta_2$ -Microglobulin deposition stimulates cytokine secretion by macrophages, causing bone resorption and collagenase synthesis, and subsequently leading to collagen degradation and connective tissue breakdown. The leading clinical and radiological symptoms are

- Carpal-tunnel syndrome.
- Radiologic evidence of destructive arthropathy, including subchondral juxta-articular erosions or cysts mainly in the wrists (Fig. 3), shoulders, hips and knees



Fig. 3. Osteolysis in the scaphoid and capitatum by  $\beta_2$ -microglobulin deposition. Note the groundglass phenomenon in the metacarpals by osteomalacia [7, 8]. Periarticular soft tissue (amyloid) formation. Thickening of the supraspinatus tendon and hip joint capsule.

- Destructive spondylarthropathy that may affect the cervical spine (Fig. 4) with risk of cord compression [9, 10], less common in the thoracolumbar spine. Additional crystal and aluminium deposition in the ligamentous and capsular structures may contribute to their weakness with consecutive instability.
- Tears of ligaments, avulsion fractures of the patellae.

# **Osteitis Deformans (Paget's Disease)**

Paget's disease is characterized by a focal, rarely multifocal or generalized transformation of normal bone into primitive woven bone, which leads to an increased volume and variable deformation of the affected region, with variable amount of mineralization. According to a generally accepted pathogenetic concept, the "count down" of the disease begins with an infection of the osteoclasts (in Paget's bone) by measles virus and/or respiratory syncytial virus. Both viruses belong to the RNA-paramyxovirus family that may be responsible for other slow virus diseases. Regional and familial spread of Paget's disease may be explained by an RNA-DNA transcriptase or by the diaplacental transmission of the virus. The infected osteoclasts are stimulated to enormous regional activity with a consecutive increase in unbalanced bone remodelling.

Historically the disease may have its roots in southern Great Britain, where the highest incidence is regis-



Fig. 4. a Destructive spondylarthropathy in C5/6 by  $\beta_2$ -microglobulin amyloidosis. b Magnetic resonance imaging (MRI) helped to exclude a soft tissue mass as an indicator of infectious spondylodiscitis



Fig. 5. a Paget's disease with large osteolyses in the skull as an incidental finding (osteoporosis circumscripta). b In contrast to metastases, the "bone matrix" is preserved and only demineralized, best demonstrated by CT. c Massively increased tracer uptake in the affected region

tered worldwide. With the great emigration to the east coast of North America, to New Zealand and to Australia the disease has spread. In countries or regions to which people from Spain or France had emigrated, the incidence of Paget's disease is very low. The incidence in the middle-European population older than 50 years amounts to 3%-5%; the disease will be detected rarely in subjects under age 40 years, although it may begin in younger people. Approximately 90% of patients, especially those with limited disease, are asymptomatic. Clinical symptoms depend on the location, extent and activity of the disease. The latter can be best evaluated from the level of serum alkaline phosphatase.

From the pathologic and radiologic viewpoints, there are 3 stages of Paget's disease that may occur meta- or synchronously in 1 or more bones [2]:

- Lytic stage: osteolytic lesions in the skull (osteoporosis circumscripta), flameshaped osteolysis corresponding to the "cutting cone" of bone resorption in the distal parts of the involved long bones (Figs. 5, 6).
- Mixed osteolytic and osteosclerotic stage can be interpreted as a reaction of the osteoblasts and activated fibroblasts with unbalanced excessive production of woven bone. In this stage the volume of the affected region increases and the irregularly coarsened structures get an overall wispy aspect.
- *Sclerotic stage*: densely calcified woven bone and further enlargement of the involved region.

Scintigraphic bone scans reveal massively increased tracer uptake (high perfusion of Paget's bone, high affinity of the tracer in woven bone) in a typically homogeneous manner and usually involving a large segment of the affected bone. Epi- or metaphyses are usually involved.

Complications of Paget's disease include severe neuro-

logic disturbances (by means of compression), cardiovascular affections (by increased circulating blood volume) and the development of sarcomas (in about 5%-10% of polyostotic manifestations) and giant-cell tumor-like reactions (Fig. 7) [11, 12].



**Fig. 6.** Paget's disease (lytic stage) in an 86year-old man. Flameshaped osteolysis in the distal tibia with a slightly increased volume of the affected region





Therapy with modern, highly effective diphosphonates (bisphosphonates) may prevent the disease from progressing.

#### References

- 1. Delling G (1989) Osteopathie bei primärem Hyperparathyreoidismus [Osteopathy by primary hyperparathyroidism]. In: Hesch RD (ed) Endokrinologie. Teil A. Urban & Schwarzenberg, Munich (Innere Medizin der Gegenwart Bd. 4. S. 529)
- 2. Freyschmidt J (1997) Skeletterkrankungen. Springer, Berlin Heidelberg New York
- Sundaram M (2000) Oncogenic osteomalacia. Skeletal Radiol 29:117
- Hruska KA, Teitelbaum SC (1995) Renal osteodystrophy (review article). N Engl J Med 333:166
- Sundaram M, Dessner D, Ballal S (1991) Solitary, spontaneous cervical and large bone fractures in aluminium osteodystrophy. Skeletal Radiol 20:91



Fig. 7a-c. Giant cell tumorlike reaction in the right lower leg of a patient with polyostotic Paget's disease. In this 61-year-old patient, we find multiple osteolytic giant cell tumor-like reactions in the contralateral side as well as in the femora. a X-ray of the right tibia and fibulo. b CT scan. c MRI

- Kriegshauser JS, Swee RG, McCarthy JT et al (1987) Aluminium toxicity in patients undergoing dialysis: radiographic findings and prediction of bone biopsy results. Radiology 164:399
- Naidich JB, Karmel MJ, Mossey RT et al (1987) Osteoarthropathy of the hand and wrist in patients undergoing long-term hemodialysis. Radiology 164:205
- Gielen JL, van Holsbeek MT, Hauglustaire D et al (1990) Growing bone cysts in long-term hemodialysis. Skel Radiol 19:43
- Naito M, Ogata K, Nakamoto M et al (1992) Destructive spondylarthropathy during long-term hemodialysis. J Bone Joint Surg Br 74:686
- Stäbler A, Kröner G, Seiderer M (1991) MRT der dialyseassoziierten, destruierenden Spondylarthropathie der Atlantoaxialregion. RöFo 154:469
- Potter HG, Schneider R, Ghelman B (1991) Multiple giant cell tumors and Paget's disease: radiographic and clinical correlations. Radiology 180:261
- Mirra JM (1986) Paget's disease and giant cell tumor. N Engl J Med 314:105

# **Metabolic Bone Disease**

J.E. Adams

The Medical School, University of Manchester, Manchester, UK

# Introduction

Metabolic diseases of the skeleton affect bone as a tissue. All bones are involved histologically, although radiological abnormalities are not always evident. Such disorders can be caused by genetic, endocrine, nutritional, or biochemical factors. Knowledge of bone structure, development, and physiology is essential to the understanding of the effects that metabolic bone disorders have on the skeleton, and in interpreting the abnormalities which they cause as seen on radiographs and using other imaging techniques.

## **Bone Structure and Physiology**

Bone is normally present in anatomical bones in two forms: (a) compact (cortical) bone, which forms the outer cortex of bones, and (b) trabecular or cancellous bone, which is found mainly in vertebral bodies, the pelvis, and the metaphyseal regions of long bones.

All bones contain both types of bony tissue, although the relative amounts of each vary, and both contribute to bone strength. Bone is a specialised tissue made up of a matrix of collagen fibers, mucopolysaccharides (35% by weight), and inorganic crystalline mineral matrix (calcium hydroxyapatite), which is distributed along the length of the collagen fibers. This inorganic component accounts for 65% of osseous tissue by weight. Despite its hardness, bone remains a metabolically active tissue throughout life, being constantly resorbed and accreted by bone cells, the activity of which can be modified by many factors [1]. As a consequence, bones remodel from birth to maturity, thereby maintaining their basic shape, and continue to be able to carry out repair following fracture and to respond to physical forces (i.e., mechanical stresses related to bone deformity) throughout life. The strength of bone is related not only to its hardness and other physical properties, but also to the size and architectural arrangement of compact and trabecular bone.

The skeleton contains 99% of the total body calcium and therefore plays a vital role in the maintenance of calcium homeostasis.

## **Bone Cells**

#### Osteoblasts

These are bone-forming cells that synthesize and secrete type I collagen and mucopolysaccharides to form layers of bone matrix (osteoid), which subsequently becomes mineralized. Osteoblasts also synthesize collagenase, prostaglandin E2 (PGE 2) and bone-associated proteins, osteocalcin, and osteonectin. Osteoblasts have receptors for parathyroid hormone, vitamin D, prostaglandin E2, and glucocorticoids. The action that some of these substances have on osteoclasts may be mediated through their action on the osteoblast.

#### Osteocytes

These cells are derived from osteoblasts. They are initially present on the surface of bone but subsequently become encased within bone. Each osteocyte lies within a lacuna and is interconnected to other osteocytes and osteoblasts by cytoplasmic extensions within canaliculi. The osteocyte has a role in the maintenance of bone matrix, which is facilitated by the transport of material and fluid via the canaliculi. At one time, osteocytes were considered to be relatively inactive, but it has been shown that they respond to calcitonin and parathyroid hormone, and so play an important role in maintaining constant levels of calcium within the body fluids.

## Osteoclasts

These multinuclear giant cells resorb both calcified bone and cartilage. They are derived from the mononuclear phagocytic cell line of hematopoeitic stem cells. Osteoclasts lie on the surface of bone causing active resorption and forming Howship's lacunae. The osteoclasts that are in contact with bone develop motile microvillae, which cause the cell to adhere to the bone surface. The adherence of the osteoclast to the bone surface results in a microenvironment between the osteoclast and the mineralized bone. This brush border of microvillae increases with activation by such factors as prostaglandin, vitamin D, and parathyroid hormone. Osteoclasts secrete acid hydrolases and neutral proteases that degrade bone matrix following its demineralization. As there are no receptors for 1,25 dihydroxy vitamin D on the surface of the osteoclast, it is probable that factors such as vitamin D and parathyroid hormone activate osteoclasts through their effects on osteoblasts. Calcitonin inhibits bone resorption by reducing the number and activity of osteoclasts.

#### **Bone Turnover**

Bone formation (osteoblastic activity) and bone resorption (osteoclastic activity) constitute bone turnover, a process that takes place on bone surfaces and continues throughout life [2]. Trabecular bone has a much greater surface to volume area than compact bone and is therefore some eight times more metabolically active. Bone formation and bone resorption are linked in a consistent sequence under normal circumstances. Precursor bone cells are activated at a particular skeletal site to form osteoclasts, which erode a fairly constant amount of bone. After a period of time, bone resorption stops and osteoblasts are recruited to fill the eroded space with new bone tissue. This coupling of osteoblastic and osteoclastic activity constitutes the basal multicellular unit (BMU) of bone. If this process becomes uncoupled, excessive osteoclastic resorption or defective osteoblastic function result in a net loss of bone (osteoporosis). If there is both increased bone resorption and formation, this constitutes increased bone turnover. Woven immature, instead of mature lamellar, bone is laid down in Paget's disease of bone. Increased activation frequency of resorption units also results in a high turnover state (hyperparathyroidism, post-menopausal bone loss). Bisphosphonate therapy reduces the activation of resorption units by inhibiting osteoclasts, and reversal in the mineral deficit contributes to an increase in bone mineral density.

Bones grow in length by enchondral ossification and remodel by periosteal apposition of bone, endosteal resorption, and osteoclastic resorption along the periosteal surface of the metaphysis. Defective osteoclastic function prevents this normal resorption of bone, which is essential to maintain bone health by continual slow renewal throughout life. Defective osteoclastic function [3] in some diseases (i.e. osteopetrosis) can result in abnormal bone modeling and sclerosis, as seen on radiographs. Bone resorption by osteoclasts is a single-stage process in which collagen and mineral are removed together, whereas bone formation is a two-stage process: osteoblasts lay down osteoid, which subsequently becomes mineralized. Pre-requisites for normal mineralization are vitamin D (1,25  $[OH]_2$  D), normal levels of phosphorus and alkaline phosphatase, and a normal pH. Defects in the mineralization process will result in rickets or osteomalacia.

Early in fetal development, the framework of the skeleton is in place, but without mineralization. At about 26 weeks of gestation, the long bones assume their future shape and proportion. Skeletal growth occurs primarily by enchondral ossification at the metaphyses and epiphyses. At birth, about 25 g of calcium is present in the skeleton. The mineral required for the fetal skeleton is acquired from the mother. The bones grow during the first two decades of life with a pubertal spurt during adolescence. Skeletal maturity is achieved at an earlier age in girls (16-18 years) than in boys (18-20 years). Some disorders (hypothyroidism, chronic ill health) may retard skeletal development. Skeletal maturation is assessed radiologically from a hand radiograph (including wrists) which is then compared with an atlas of hand radiographs of normal American Caucasian boys and girls of different ages (Greulich and Pyle 1959) [4)] or using the Tanner and Whitehouse [5] bone score (TW2) method, which assesses changes in presence, size, and shape of certain bones with age. The latter method is more time-consuming; both methods provide comparable results and reproducibility [6]. Automated, computer-based techniques have potential in the quantitative assessment of the skeleton [7]. Following attainment of skeletal maturity, there then follows a period of consolidation during which peak bone mass is achieved. For cortical bone, this is reached at about 35 years of age and a little earlier for trabecular bone. Although the long bones grow in length at the metaphyses, they are remodeled in shape during development by endosteal resorption and periosteal apposition.

The size and shape of the skeleton and its individual bones are determined by genetic factors, but are influenced by endocrine and local growth factors, nutrition and physical activity [8]. Remodeling allows the skeleton to adjust to those mechanical forces to which it is exposed.

There is considerable variation in skeletal size and weight, both within and between races [9]. Black races have larger and heavier bones than whites, and Chinese have a small skeletal mass and size. Although genetic factors are important, they are modified by environmental differences such as diet and physical activity [10, 11, 12].

After the attainment of peak bone mass, bone loss, particularly of trabecular bone, is believed to occur from the third decade of life. Bone loss is a phenomenon that occurs in all races. Generally, both men and women lose bone as they grow older, but women lose more than men. Women lose approximately 15-30% of their total bone mass between maturity and the seventh decade, whereas men lose only about half this amount. Relatively more trabecular bone is lost (40-50%) than

compact bone (5%). After the age of 35, women lose bone at an annual rate of approximately 0.75-1.0%, which increases to a rate of 2-3% after the menopause. This loss affects both cortical and trabecular bone, but the effect on trabecular bone predominates. In contrast, cortical bone is well preserved until the fifth decade of life, when there is a linear loss in both sexes, such that men lose about 25% of their cortical bone whilst women lose about 30%. Low bone mineral density can be the result of either low peak bone mass or accelerated bone loss.

The amount of bone in the skeleton at any moment in time depends on peak bone mass and the balance between bone resorption and formation. The most common metabolic disorders of bone are:

- 1. Hyperparathyroidism, in which a tumor or hyperplasia of the parathyroid glands causes increase in parathormone production and stimulation of osteoclasts.
- 2. Rickets and osteomalacia, in which there is defective mineralization of bone osteoid due to vitamin D deficiency, hypophosphatemia, lack of alkaline phosphatase, or severe acidaemia.
- 3. Osteoporosis, in which there is a deficiency of bone mass leading to insufficiency (low trauma) fractures.

Other metabolic bone disorders include osteogenesis imperfecta, hyperphosphatasia, hyperphosphatemia, and osteopetrosis.

# **Parathyroid Disorders**

Most parathyroid tumous are functionally active and result in the clinical syndrome of primary hyperparathyroidism. This is the most common endocrine disorder after diabetes and thyroid disease, with an incidence within the population of about 1 in 1000 (0.1%). The incidence is higher in the elderly than in those under 40, and is most common in women age 60 or older. Over the past 50 years, the prevalence of the condition has increased some tenfold; this increase is due principally to the detection by chance of hypercalcemia in patients, many of whom are asymptomatic, through routine use of multichannel autoanalysis of serum samples since the 1970s.

#### Hyperparathyroidism

## Primary Hyperparathyroidism

The majority (80%) of patients with primary hyperparathyroidism have a single adenoma [13]. Multiple parathyroid adenomas may occur in 4% of patients. Chief-cell hyperplasia of all glands occurs in 15-20% of patients; the histological diagnosis depends on the finding that more than one parathyroid gland is affected. Genetic factors are relevant in a proportion of these patients (familial hyperplasia, multiple endocrine neoplasia [MEN] syndromes).

Carcinoma of the parathyroid is an infrequent cause of primary hyperparathyroidism (0.5%) [14]. The malignant tumor is slow growing but locally invasive. Cure may be obtained by adequate surgical excision and there is a 50% or greater 5-year survival rate. However, recurrence is common (30%) and metastases to regional lymph nodes, lung, liver and bone occur late in 30% of patients. Biochemical remission may occur spontaneously, presumably due to infarction of the tumor, but this is extremely rare, since the parathyroid glands have a very rich blood supply from both the inferior and superior thyroid arteries. Metastases, when solitary, may be resected with benefit.

#### Secondary Hyperparathyroidism

Secondary hyperparathyroidism is induced by any condition or circumstance that cause serum calcium to fall. This occurs in vitamin D deficiency, intestinal malabsorption of calcium, and in chronic renal failure (through lack of the active metabolite of vitamin D,  $(1,25 \text{ (OH)}_2 \text{ D})$ , and retention of phosphorus. If this secondary hyperparathyroidism is of sufficiently long standing, an autonomous adenoma may develop in the hyperplastic parathyroid glands, a condition referred to as tertiary hyperparathyroidism. This condition is usually associated with chronic renal disease but it has also been observed in patients with long-standing vitamin D deficiency and osteomalacia from other causes.

## **Clinical Presentation**

Most patients with primary hyperparathyroidism have mild disease and commonly have no symptoms, the diagnosis being made by the finding of asymptomatic hypercalcemia. The most common clinical presentations, particularly in younger patients, are related to renal stones and nephrocalcinosis (25-35%), high blood pressure (40-60%), and acute arthropathy (pseudogout), caused by calcium pyrophosphate dihydrate deposition (chondrocalcinosis). Osteoporosis, peptic ulcer and acute pancreatitis, depression, confusional states, proximal muscle weakness, and mild non-specific symptoms such as lethargy, arthralgia and difficulties with mental concentration may also occur.

#### Treatment

Surgical removal of the overactive parathyroid tissue is generally recommended. In experienced hands, surgical excision is successful in curing the condition in over 90% of patients [15]. The decision to operate, particularly in the elderly and those with asymptomatic disease, requires careful assessment [16]. Conservative treatment may be judged to be the management of choice, with monitoring of the serum calcium, renal function, blood pressure, and bone density at regular intervals [17, 18].

## **Radiological Findings**

With the increased number of patients with primary hyperparathyroidism being diagnosed with asymptomatic hypercalcemia, the majority (95%) of patients will have no radiological abnormalities.

Sub-periosteal erosions (Fig. 1) of cortical bone, particularly in the phalanges, is pathognomonic of hyperparathyroidism [13, 19]. The most sensitive site to identify this early subperiosteal erosion is along the radial aspects of the middle phalanges of the index and middle fingers. Other sites may be involved including the distal phalanges (acro-osteolysis), the outer ends of the clavicle, the symphysis pubis, the sacroiliac joints, the proximal medial cortex of the tibia, the proximal humeral shaft, ribs, and femur. However, if no subperiosteal erosions are identified in the phalanges, they are unlikely to be identified radiographically elsewhere in the skeleton. Subperiosteal erosions in sites other than the phalanges indicate more severe and long-standing hyperparathyroidism, such as may be found secondary to chronic renal impairment.

#### Intracortical Bone Resorption

Intracortical bone resorption results from increased osteoclastic activity in haversian canals. Radiographically, this causes linear translucencies within the cortex (cortical "tunnelling"). This feature is not specific for hyperparathyroidism, and can be found in other conditions in which bone turnover is increased (e.g., normal childhood, Paget's disease of bone).



**Fig. 1.** Hyperparathyroidism: there are subperiosteal erosions along the radial cortex of the middle phalanges and of the terminal phalanges of the second and third fingers

#### **Chondrocalcinosis**

The deposition of calcium pyrophosphate dihydrate (CP-PD) causes articular cartilage and fibrocartilage to become visible on radiographs [20]. This is most likely to be identified on radiographs of the hand (triangular ligament), the knees (articular cartilage and menisci), and symphysis pubis. Other joints less commonly involved are the shoulder and the hip. Clinically, the patients may present with acute pain resembling gout, but on joint aspiration pyrophosphate crystals, rather than urate crystals, are found. Affected joints, however, may be asymptomatic, and chondrocalcinosis noted radiographically might bring the diagnosis of hyperparathyroidism to light in an asymptomatic patient. The combination of chondrocalcinosis in the symphysis pubis and nephrocalcinosis on an abdominal radiograph is diagnostic of hyperparathyroidism. Chondrocalcinosis is a feature of primary disease, rather than occurring secondary to chronic renal impairment.

#### Brown Tumors (Osteitis Fibrosa Cystica)

These are cystic lesions within bone in which there has been excessive osteoclastic resorption. Histologically, the cavities are filled with fibrous tissue and osteoclasts, with necrosis and hemorrhagic liquefaction. Radiographically, brown tumors appear as low-density, multiloculated cysts that can occur in any skeletal site and may cause expansion of bones. They are now rarely seen.

## Osteosclerosis

Osteosclerosis occurs uncommonly in primary hyperparathyroidism [21] but is a common feature of disease secondary to chronic renal impairment [22]. In primary disease, with normal renal function, it results from an exaggerated osteoblastic response following bone resorption. In secondary causes of hyperparathyroidism, it results from excessive accumulation of poorly mineralized osteoid, which appears more dense radiographically than normal bone. The increase in bone density affects particularly the axial skeleton. In the vertebral bodies, the end plates are preferentially involved, giving bands of dense bones adjacent to the end plates with a central band of lower normal bone density. These alternating bands of normal and sclerotic bone give a stripped pattern described as a "rugger jersey" spine (Fig. 2).

#### **Osteoporosis**

With excessive bone resorption, the bones may appear reduced in density in some patients. This may particularly occur in postmenopausal women and the elderly, in whom bone resorption exceeds new bone formation, with a net reduction in bone mass. This can be con-



Fig. 2. Secondary hyperparathyroidism in chronic renal insufficiency: bone sclerosis of vertebral endplates giving the appearance of a "rugger jersey" in the thoracic spine



**Fig. 3.** Azotemic osteodystrophy: phosphate retention due to reduced glomerular function associated with secondary hyperparathyroidism causes metastatic calcification in soft tissues around the left hip joint

firmed by bone densitometry, which is an integral component in the evaluation of hyperparathyroidism. In primary hyperparathyroidism, there is a pattern of skeletal involvement that preferentially affects the cortical, as opposed to the trabecular, bone. Bone mineral density measurements made in sites in which cortical bone predominates, e.g. in the distal forearm, may show the most marked reduction [23]. Bone density increases after parathyroidectomy in primary hyperparathyroidism [24].

#### Metastatic Calcification

Soft-tissue calcification (Fig. 3), other than in articular cartilage and fibrocartilage, does not occur in primary hyperparathyroidism, unless there is associated reduced glomerular function resulting in phosphate retention. The latter results in an increase in the calcium phosphate product, and as a consequence amorphous calcium phosphate is precipitated in organs and soft tissues [25]. If there are features of hyperparathyroidism, i.e., subperiosteal erosions and, additionally, vascular or soft-tissue calcifications, e.g., around joints and in tendons, this implies impaired renal function in association with hyperparathyroidism.

# Hypoparathyroidism

## Etiology

Hypoparathyroidism can result from reduced or absent parathyroid hormone production or from end-organ (kidney, bone or both) resistance. This may be the result of the parathyroid glands failing to develop, the glands being damaged or removed, the function of the glands being reduced by altered regulation, or the action of PTH being impaired [26]. The biochemical abnormality that results is hypocalcemia; this can clinically cause neuromuscular symptoms and signs such as tetany and fits.

Acquired hypoparathyroidism results either from surgical removal of the parathyroid glands or from autoimmune disorders. Post-surgical hypoparathyroidism is more common, and occurs in approximately 13% of patients following surgery. Idiopathic hypoparathyroidism usually presents during childhood, is more common in girls, and rare in black races. It may be associated with pernicious anemia and Addison's disease. There may be antibodies to a number of endocrine glands as part of a generalized autoimmune disorder.

#### **Radiological** Abnormalities

There may be localized (23%) or generalized (9%) osteosclerosis in affected patients [27]. This particularly affects the skull, where the vault is thickened. At an early age of onset, the dentition is hypoplastic. Metastatic calcification may be present in the basal ganglia or in the subcutaneous tissue, particularly about the hips and shoulders.

A rare but recognized complication of hypoparathyroidism is an enthesopathy with extraskeletal ossification in a paraspinal distribution and elsewhere. In the spine this skeletal hyperostosis resembles most closely that described by Forestier as "senile" hyperostosis [28, 29]. Differentiating features from ankylosing spondylitis are that there is no erosive arthropathy and the sacroiliac joints appear normal. Clinically, the patients may have pain and stiffness in the back with limitation of movement. Extraskeletal ossification may be present around the pelvis, hip, and in the interosseous membranes and tendinous insertions elsewhere.

## Pseudohypoparathyroidism

Pseudohypoparathyroidism (PHP) describes a group of genetic disorders characterised by hypocalcemia, hyperphosphatemia, raised PTH, and target-tissue unresponsiveness to PTH [31, 32]. The condition was first described by Albright et al. in 1942 [33]. Affected patients are short in stature, have reduced intellect, rounded faces, and shortened metacarpals, particularly the fourth and fifth. Metastatic calcification, bowing of long bones and exostoses can occur. Clinical features include tetany, cataracts, and nail dystrophy. Some of the clinical and radiological features of PHP may resemble those in other hereditary syndromes, including Turner's syndrome, acrodysostosis, Prader-Willi syndrome, fibrodysplasia ossificans progressiva, and multiple hereditary exostosis.

In PHP there is an end-organ unresponsiveness to PTH since the parathyroid glands are normal and produce the hormone. This usually involves unresponsiveness of both bone and kidneys. However, there is a rare variation of PHP in which the kidneys are unresponsive to PTH, but the osseous response to the hormone is normal [34]. The condition is referred to as pseudohypohyperparathyroidism, and the histologic and radiological features resemble those of azotemic osteodystrophy.

#### **Radiographic Abnormalities**

Abnormalities may not be evident at birth but subsequently there develops premature epiphyseal fusion, calvarial thickening, bone exostoses, and calcification in the basal ganglia and in the soft tissue. Metacarpal shortening is present, particularly affecting the fourth and fifth digits. This may result in a positive metacarpal sign in which, if a line is drawn tangential to the heads of the fourth and fifth metacarpals, the line should not normally intersect the third metacarpal, but does if there is shorting of the fourth metacarpal. This feature is not specific for PHP and can occur in other congenital (Beckwith-Weidemann and basal-cell nevus syndromes, multiple epiphyseal dysplasia) and acquired (juvenile chronic arthritis, sickle-cell disease with infarction) conditions. Soft-tissue calcification occurs in a plaque-like distribution in the subcutaneous area. Rarely, soft-tissue ossification can occur in a periarticular distribution, usually involving the hands and feet.

#### Pseudo-pseudohypoparathyroidism (Pphp)

In these affected individuals, the dysplastic and other features are the same as PHP, but there are no associated parathyroid or other biochemical abnormalities. The abnormalities of metacarpal and metatarsal shortening, calvarial thickening, exostoses, soft-tissue calcification, and ossification are best identified on radiographs. Computed tomography (CT) of the brain may be more sensitive at identifying basal ganglia calcification. Bone density may be normal, reduced, or increased.

## **Rickets and Osteomalacia**

#### Introduction

The mineralization of bone matrix depends on the presence of adequate supplies of 1,25 di-hydroxy vitamin D (1,25 (OH)<sub>2</sub>D), calcium, phosphorus and alkaline phosphatase, and on a normal body pH. If there is a deficiency of any of these substances, or if there is severe systemic acidosis, the mineralization of bone will be defective. This results in a qualitative abnormality of bone, with a reduction in the mineral to osteoid ratio, resulting in rickets in children and osteomalacia in adults. Rickets and osteomalacia are therefore synonymous, and represent the same disease process, but are the manifestation in either the growing or the mature skeleton. In the immature skeleton, the radiographic abnormalities predominate at the growing ends of the bones, where enchondral ossification is taking place, giving the classic appearance of rickets. At skeletal maturity, when the process of enchondral ossification has ceased, the defective mineralization of osteoid is evident radiographically as Looser's zones (pseudofractures, Milkman's fracture), which are pathognomonic of osteomalacia [35, 36]. Many different conditions can cause the same radiological abnormalities of rickets and osteomalacia. In the past, there was much confusion between these conditions, which had similar clinical and radiological features but different patterns of progression and responses to therapies of the day. Much of the causes of confusion have been clarified with the increased understanding during the twentieth century of the structure and function of vitamin D and its metabolites.

The pro-hormone forms of vitamin D require two hydroxylation stages to form the active metabolite, through which the hormone exerts its physiological action. There are two pro-hormonal forms of 1,25 di-hydroxy D in humans: vitamin  $D_2$  and vitamin  $D_3$ . Vitamin  $D_2$  is prepared by irradiation of ergosterol, obtained from yeast or fungi, and is used for food supplementation and pharmaceutical preparations. Vitamin  $D_3$  occurs naturally through the interaction of ultraviolet light on 7-dehydrocholesterol, in the deep layers of the skin. Vitamin  $D_2$  and  $D_3$  are initially hydroxylated at the 25 position to form 25-OH- $D_2$  and 25-OH- $D_3$ , the latter predominating and circulating bound to a specific protein. This hydroxylation occurs predominantly in the liver. A further hydroxylation in the 1 position in the kidney produces 1,25 (OH)<sub>2</sub>  $D_3$ , which is the active form of the hormone.

# Vitamin D Deficiency

Deficiency of vitamin D may occur as a consequence of simple nutritional lack (diet, lack of sunlight), malabsorption states (vitamin D is fat soluble and absorbed in the small bowel), chronic liver disease (which affects hydroxylation at the 25 position), and chronic renal disease (in which the active metabolite 1,25 di-hydroxy D is not produced). Consequently, a wide variety of diseases may result in vitamin D deficiency; the radiological features will be similar, being those of rickets or osteomalacia. This similarity of radiological features, but variation in response to treatment, contributed to some of the early confusion. Rickets due to nutritional deprivation was cured by ultraviolet light or physiological doses of vitamin D (400 IU per day), but that associated with chronic renal disease was not, except if very large pharmacological doses (up to 300,000 IU per day) were used. This led to the terms "refractory rickets" and "vitamin D resistant rickets" being used for these conditions. Within these terms were included the diseases that cause the clinical and radiological features of rickets, but were related to phosphate, not vitamin D, deficiency, such as X-linked hypophosphatemia, and genetic disorders involving defects in 1a-hydroxylase and the vitamin D receptor.

# Genetic Disorders of Vitamin D Metabolism

Prader et al., in 1961, described the condition in which rickets occurred within the first year of life and was characterized by severe hypocalcemia and dental enamel hypoplasia, and which responded to large amounts of vitamin D. The term "vitamin D dependency" was used for this syndrome. It is now recognized that this disease is due to an in-born error of metabolism in which there is defective hydroxylation of 25(OH)D in the kidney due to defective activity of the renal 25(OH)D 1 $\alpha$ -hydroxylase. This results in insufficient synthesis of 1,25(OH)<sub>2</sub>D. The preferred term for this condition is pseudo-vitamin-D deficiency rickets (PDDR) and it is inherited as an autosomal recessive trait [37].

Another in-born error of vitamin D metabolism was described in 1978 [38]; it clinically resembles PDDR but there are high circulating concentrations of  $1,25(OH)_2D$ . This condition results from a spectrum of mutations that

affect the vitamin D receptor (VDR) in target tissues, causing resistance to the action of  $1,25(OH)_2D$  (end-organ resistance). Affected patients have complete alopecia.

# **Radiological Appearance**

## Rickets

In the immature skeleton, the effect of vitamin D deficiency and the consequent defective mineralization of osteoid is seen principally at the growing ends of bones [35, 36, 39] (Fig. 4). In the early stage, there is apparent widening of the growth plate, which is the translucent "unmineralised" gap between the mineralized metaphysis and epiphysis. More severe change produces "cupping" of the metaphysis, with irregular and poor mineralization. Some expansion in width of the metaphysis results in swelling around the ends of the affected long bones. This expansion of the anterior ends of the ribs is referred to as a "rachitic rosary". There may be a thin ghost-like rim of mineralization at the periphery of the metaphysis which occurs by membranous ossification at the periosteum. The margin of the epiphysis appears indistinct as enchondral ossification at this site is also defective. These changes predominate at the sites of bones that are growing most actively, around the knee, the wrist (particularly the ulna), the anterior ends of the middle ribs, the proximal femur and the distal tibia, and depend on the age of the child. If rickets is suspected it is these anatomical sites that are most likely to show radiographic abnormality.

Rachitic bone is soft and bends and this results in genu valgum or genu varum, deformity of the hips (coxa valga or more commonly coxa vara), in-drawing of the ribs



**Fig. 4.** Rickets: widening of the growth plate with "flaring" and poor mineralization of the metaphyses

at the insertion of the diaphragm (Harrison's sulcus), protrusio acetabuli, and triradiate deformity of the pelvis, which can cause problems with subsequent parturition. Involvement of the bones in the thorax and respiratory tract (larvnx and trachea) rarely result in stridor and respiratory distress. In very severe rickets, when little skeletal growth is taking place (i.e. owing to nutritional deprivation or chronic ill health), radiological features of rickets may not be evident at the growth plate. In rickets of prematurity, little abnormality may be present at the metaphysis since no skeletal growth is taking place in the premature infant. However, the bones are osteopenic and prone to fractures. In mild vitamin D deficiency, the radiographic features of rickets may only become apparent at puberty, during the growth spurt, with the metaphyseal abnormalities predominating at the knee.

With appropriate treatment of vitamin D deficiency, the radiographic features of healing lag behind the improvement in biochemical parameters (2 weeks) and clinical symptoms. With treatment, the zone of provisional calcification will mineralize. This zone is initially separated by translucent osteoid from the shaft of the bone and may be mistaken for a metaphyseal fracture of child abuse [40]. Reduced bone density and poor definition of epiphyses are helpful distinguishing features for rickets. The section of abnormal bone following healing of rickets may be visible for a period of time, and give some indication as to the age of onset and duration of the period of rickets. Eventually, this zone will become indistinguishable from normal bone with remodeling over a period of 3 to 4 months. The zone of provisional calcification that was present at the onset of the disturbance to enchondral ossification may remain (Harris growth arrest line) [41] as a marker of the age of skeletal maturity at which the rickets occurred. However, this is not specific for rickets and can occur in any condition (i.e. period of ill health, lead poisoning) that inhibits normal enchondral ossification. There will be evidence of retarded growth and development in rickets, but in my experience this tends to be more marked when the vitamin D deficiency is associated with chronic diseases that reduce calorie intake, general well-being, and activity (i.e. malabsorption, chronic renal disease) than with simple nutritional vitamin D deficiency.

Vitamin D deficiency is associated with hypocalcemia. In an attempt to maintain calcium homeostasis, the parathyroid glands are stimulated to secrete PTH. This results in another important feature of vitamin D deficiency rickets. Evidence of secondary hyperparathyroidism, with increased osteoclastic resorption, is always evident histologically, although not always radiographically.

Metaphyseal chondrodysplasias encompass a variety of inherited bone dysplasias in which there are metaphyseal abnormalities ranging from mild (Schmit Type) to severe (Jansen) [42]. Normal serum biochemistry serves to differentiate these dysplasias from other rachitic disorders that the radiographic abnormalities at the metaphyses may simulate.

#### Osteomalacia

At skeletal maturity, the epiphysis fuses to the metaphysis with obliteration of the growth plate and cessation of longitudinal bone growth. However, bone turnover continues throughout life in order to maintain the tensile integrity of the skeleton. Vitamin D deficiency in the adult skeleton results in osteomalacia, the pathognomonic radiographic feature of which is Looser's zone, named after E. Looser, who described them in 1908 [43]. Looser's zones (pseudofractures, Milkman's fractures) [44] are translucent areas in the bone that are composed of unmineralized osteoid (Fig. 5). They are typically bilateral and symmetrical. Radiographically, they appear as radiolucent lines that are perpendicular to the bone cortex, do not usually extend across the entire bone shaft, and characteristically have a slightly sclerotic margin. Looser's zones can occur in any bone, but most typically are found in the medial aspect of the femoral neck, the pubic rami, the lateral border of the scapula, and the ribs. They may involve the first and second ribs, in which traumatic fractures are uncommon, being usually associated with severe trauma. Other less common sites for Looser's zones are the metatarsals and metacarpals. the base of the acromiom, and the ilium. They may not always be visible on radiographs; radionuclide bone scans are more sensitive in identifying radiographic occult Looser's zones [45].

Looser's zones must be differentiated from insufficiency fractures that can occur in osteoporotic bone, particularly in the pubic rami, sacrum, and calcaneus. Such insufficiency fractures consist of multiple microfractures in brittle osteoporotic bone and often show florid callus formation, serving to differentiate them from Looser's zones [46, 47]. Incremental fractures occur in Paget's disease of bone and resemble Looser's zones in appearance, but tend to occur on the convexity



**Fig. 5.** Osteomalacia: Looser's zone in the medial aspect of the right femoral neck

of the cortex of the bone involved [48, 49], rather than medially, as in osteomalacia. The other typical features of Paget's disease serve as distinguishing radiological features.

Complete fractures can occur through Looser's zones, but with no evidence of callus formation until the osteomalacia is treated with vitamin D. Then there will be quite florid callus formation around fractures and healing of fractures and Looser's zones with little residual deformity. However, as in rickets, osteomalacic bone is soft and bends. This is evident radiographically by protrusio acetabuli, in which the femoral head deforms the acetabular margin so that the normal "teardrop" outline is lost. There may be bowing of the long bones of the legs and a triradiate deformity of the pelvis, particularly if the cause of the vitamin D deficiency has persisted since childhood, and has been inadequately treated or untreated.

In osteomalacia, as in rickets, hypocalcemia acts as stimulus to secondary hyperparathyroidism. This may be manifested radiographically as subperiosteal erosion, particularly in the phalanges but other sites (sacroiliac joints, symphysis pubis, proximal tibia, outer ends of the clavicle, skull vault – "pepperpot" skull) may be involved, depending on the intensity of the hyperparathyroidism and how long it has been present. There may also be cortical tunnelling and a hazy trabecular pattern. Generalised osteopenia may occur and vertebral bodies may have biconcave endplates, due to deformation of the malacic bone by the cartilaginous intervertebral disc ("cod fish" deformity) [50].

# **Renal Osteodystrophy**

The bone disease associated with chronic renal impairment is complex and multifactorial, and has changed over the past decades [22, 51]. Whereas, originally, features of vitamin D deficiency (rickets/osteomalacia) and secondary hyperparathyroidism (erosions, osteosclerosis, brown cysts) predominated, improvement in management and therapy have resulted in such radiographic features being present in a minority of patients. Metastatic calcification (Fig. 3) and "adynamic" bone develop as a complication of disease (phosphate retention) and treatment (phosphate binders). New complications (amyloid deposition, noninfective spondyloarthropathy, osteonecrosis) are now seen in long-term hemodialysis and/or renal transplantation. Radiographs remain the most important imaging techniques, but occasionally other imaging and quantitative techniques (CT, MRI, bone densitometry) are relevant to diagnosis and management.

In extreme cases of soft-tissue calcification, there may be ischemic necrosis of the skin, muscle and subcutaneous tissue, referred to as "calciphylaxis". This condition can occur in patients with advanced renal disease, in those on regular dialysis, and also those with functioning renal allografts [52].

#### **Renal Tubular Defects**

Glucose, inorganic phosphate, and amino acids are absorbed in the proximal renal tubule; concentration and acidification of urine in exchange for a fixed base occur in the distal renal tubule. Renal tubular disorders may involve either the proximal or the distal tubule, or both. These disorders will result in a spectrum of biochemical disturbances that may result in loss of phosphate, glucose, or amino acids alone, or in combination, with additional defects in urine acidification and concentration. Such defects of tubular function may be inherited and present from birth (Toni-Fanconi syndrome, cystinosis, X-linked hypophosphatemia), or later in life (e.g. tubular function being compromised by deposition of copper in Wilson's disease, hereditary tyrosinemia), or be acquired by tubular dysfunction induced by the effects of toxins or therapies (paraguat, lysol burns, toluene "glue sniffing" inhalation, ifosfamide, gentamicin, streptozotocin, valproic acid), deposition of heavy metals or other substances (multiple myeloma, cadmium, lead, mercury), in relation to immunological disorders (interstitial nephritis, renal transplantation), or to the production of a humoral substance in tumor-induced osteomalacia, also know as "oncogenic rickets" [53, 54]. In these renal tubular disorders, rickets or osteomalacia can be caused by multiple factors, including hyperphosphaturia, hypophosphatemia, and reduced  $1\alpha$  hydroxylation of 25(OH)D. When serum calcium is generally normal, secondary hyperparathyroidism does not occur.

#### X-Linked Hypophosphatemia

X-linked hypophosphatemia (XLH) (Fig. 6) is a genetic disorder transmitted as an X-linked dominant trait [55]. Sporadic cases also occur through spontaneous mutations. The incidence is approximately 1 in 25,000, and XLH is now the most common cause of genetically induced rickets. The disease is characterised by phosphaturia throughout life, hypophosphatemia, rickets and osteomalacia. Clinically affected individuals may be short in stature, principally due to defective growth in the legs, which are bowed; the trunk is usually normal [59]. Rickets becomes clinically evident at about 6-12 months of age or older. Treatment with phosphate supplements and large pharmacological doses of vitamin D (hence the term "vitamin D-resistant rickets") may heal the radiological features of rickets, and also increase longitudinal growth [57]. The radiological features of XLH are characteristic [58].

There is defective mineralization of the metaphysis and widening of the growth plate (rickets). The metaphyseal margin tends to be less indistinct than in nutritional rickets and the affected metaphysis is not as wide. Changes are most marked at the knee, wrist, ankle, and proximal femur. Healing can be induced with appropriate treatment (phosphate supplements,  $1,25(OH)_2D$ ) [57]. The growth plates fuse normally at skeletal maturation.



**Fig. 6a, b.** X-linked hypophosphatemic rickets/osteomalacia. **a** Pelvis and femora: the bones are increased in density and undertubulated, with ricketic changes at the metaphyses. **b** Pelvis: evidence of an enthesopathy with new bone formation around the hips, paraspinal ossification (but normal sacro-iliac joints, to differentiate this condition from ankylosing spondylitis), and dense bones with a coarse trabeular pattern

The bones are often short and under-tubulated (shaft wide in relation to bone length) with bowing of the femur and tibia, which may be marked. Following skeletal maturation, Looser's zones appear and persist in patients with XLH. They tend to be in sites that are different from those in nutritional osteomalacia and often affect the outer cortex of the bowed femur, although they also occur along the medial cortex of the shaft. Looser's zones in the ribs and pelvis are rare. Although Looser's zones may heal with appropriate treatment, those that have been present for many years persist radiographically and are presumably filled with fibrous tissue.

Although there is defective mineralization of osteoid in XLH, the bones are commonly and characteristically increased in density, with a coarse and prominent trabecular pattern. This is a feature of the disease and is not related to treatment with vitamin D and phosphate supplements, as it is present in those who have not received treatment. This bone sclerosis can involve the petrous bone and structures of the inner ear, and may be responsible for the hydropic cochlea pattern of deafness that these patients can develop in later life [59].

X-linked hypophosphatemia is characterised by an enthesopathy, in which there is inflammation in the junctional area between bone and tendon insertion that heals by ossification at affected sites [60]. As a result, ectopic bone forms around the pelvis and spine. This may result in complete ankylosis of the spine, resembling ankylosing spondylitis, and clinically limiting mobility. However, the absence of inflammatory arthritis, with normal sacroiliac joints, serves to differentiate XLH from ankylosing spondylitis. Ossification can occur in the interosseous membrane of the forearm and in the leg between the tibia and the fibula. Separate, small ossicles may be present around the joints of the hands and ossification of tendon insertions in the hands cause "whiskering" of bone margins.

A rare, but recognized, complication of XLH is spinal cord compression caused by a combination of ossification of the ligamentum flavum, thickening of the laminae, and hyperostosis around the apophyseal joints [61]. Ossification of the ligamentum flavum causes the most significant narrowing of the spinal canal and occurs most commonly in the thoracic spine, generally involving two or three adjacent segments. Affected patients may be asymptomatic, even when there is severe spinal-canal narrowing. Acute cord compression can be precipitated by guite minor trauma. It is important to be aware of this rare complication of the disease since surgical decompression by laminectomy is curative, and is best performed as an elective procedure by an experienced surgeon rather than as an emergency. The extent of intraspinal ossification cannot be predicted by the degree of paraspinal or extra skeletal ossification at other sites. Computed tomography is a useful imaging technique for demonstrating the extent of intraspinal ossification.

Extraskeletal ossification is uncommon in patients with XLH before the age of 40 years. The extent to which radiographic abnormalities of rickets and osteomalacia, osteosclerosis, abnormalities of bone modeling and extraskeletal ossification are present varies between affected individuals [62]. In some, all the features are present and are thus diagnostic of the condition. In others, there may only be minor abnormalities and the diagnosis of XLH rickets may be overlooked [63].

## Tumor Induced "Oncogenic" Rickets/Osteomalacia

Tumor-induced osteomalacia (TIO) or "oncogenic" rickets and osteomalacia was first reported in 1947 [64]. The condition is characterized by phosphaturia and hypophosphatemia induced by a factor (phosphatonin) produced by the tumor which has various effects (inhibiting production of 1,25(OH)<sub>2</sub>D; direct effect on the renal tubule) and is associated with the clinical and radiographic features of rickets and osteomalacia. Such features may precede identification of the causative tumor by long periods (1-16 years). The tumors are usually small, benign, and of vascular origin (hemangiopericytoma), but there is now known to be a wide spectrum of tumors that may result in this syndrome, some of which may be malignant [65]. The causative lesions may originate in the skeleton and occur in neurofibromatosis. The biochemical abnormalities will be cured, and the rickets and osteomalacia will heal, with surgical removal of the tumor [66]. Often the tumors are extremely small and elude detection for many years. It is important that the affected patient is vigilant about self examination and reports any small palpable lump or skin lesion. More sophisticated imaging (CT, MRI) may be helpful in localizing deep-seated lesions [65].

# Other Causes of Rickets and Osteomalacia Not Related to Vitamin D Deficiency or Hypophosphatemia

#### Hypophosphatasia

Hypophosphatasia is a rare disorder that was first described by Rathbun, in 1948 [67]. It is generally transmitted as an autosomal recessive trait, but autosomal dominant inheritance has also been reported. The disease is characterized by reduced levels of serum alkaline phosphatase (both bone and liver isoenzymes), with raised levels of phosphoethanolamine in the blood and the urine. Serum calcium and phosphorus levels are not reduced; in perinatal and infantile disease there can be hypercalciuria and hypercalcemia attributed to the imbalance between calcium absorption from the gut and defective growth and mineralization of the skeleton. The latter results in rickets in childhood and osteomalacia in adults. The severity of the condition varies greatly, being diagnosed either in the perinatal period, in infancy, or during childhood, but in some patients it only becomes apparent in adult life [68]. The condition can wax and wane and tends to be more severe in children than when it becomes apparent in later life. In severely affected neonates, there is little, if any, evidence of mineralization of the skeleton; in extreme cases there may be such poor mineralization that only the skull base is visualized radiographically. Death ensues soon after birth since there is inadequate bony support for the thorax or brain. Less se-

verely affected children survive with rachitic metaphyseal changes appearing soon after birth as growth proceeds. The abnormalities at the growth plates resemble nutritional vitamin D deficiency rickets, but in hypophosphatasia there are larger, irregular lucent defects that often extend into the metaphyses and diaphyses. There may be a generalized reduction in bone density with a coarse trabecular pattern. The long bones, particularly those in the lower limbs, become bowed, fractures may occur and be the presenting feature. Such fractures may or may not heal; when they do unite, it is through subperiosteal new bone formation. In severe disease, multiple fractures may cause deformity and limb shortening. Initially, the skull sutures are widened due to poor mineralization of the skull vault; later, premature fusion may lead to craniostenosis. This can result in raised intracranial pressure, bulging of the anterior fontanelle, proptosis and papilloedema. Wormian (intersutural) bones may be identified.

In adult onset of the disease, the presenting clinical feature is usually a fracture, occurring after relatively minor trauma, particularly in the metatarsals. Fracture healing is slow or absent with little callus formation, but will occur following intramedullary nailing [69]. The features of osteomalacia may be present, with Looser's zones, a coarse trabecular pattern and bowing deformities of the limbs. Chondrocalcinosis and extraskeletal ossification of tendinous and ligamentous insertions to bone may occur [70]. The diagnosis is confirmed by the biochemical changes of reduced alkaline phosphatase and raised blood and urine phosphoethanolamine. As there is no effective treatment for hypophosphatasia, severely affected patients can prove a challenge to orthopedic management [69].

## Osteoporosis

## Introduction

Osteoporosis is the most common metabolic bone disease, and affects one in three postmenopausal women and one in twelve men in their lifetime. The disease is characterized by reduced bone mass and deterioration in trabecular structure. The clinical consequence is low-trauma fractures, particularly in the spine, wrist, and hip [71, 72]. All may be associated with pain and deformity, and hip fractures cause significant mortality. In the past, there was little effective preventative or bone-enhancing therapy, but now this is not the case. Supplementary calcium and vitamin D, bisphosphonates, and selective estrogen receptor modulators (SERMs) increase bone density and reduce future fracture risk [73]. It is therefore relevant to identify patients at risk, preferably before fractures occur. Radiologists have an important role to play in this objective. There are features on radiographs that should be recognized and reported clearly; if present, it should be suggested that the patient be referred to a clinician with a special interest in osteoporosis management and that bone densitometry be performed to confirm the diagnosis [74]. Imaging techniques also play a role in differentiating acute from old and stable osteoporotic fractures (relevant to the selection of patients appropriate for vertebroplasty), and in confirming that fractures are not related to pathologies (metastases, myeloma) other than osteoporosis [75].

#### **Causes of Osteoporosis**

Osteoporosis may be generalized or regional [72].

The most common cause of generalized osteoporosis is the bone loss that occurs with aging (senile) and in women after the menopause (postmenopausal). Osteoporosis can be associated with endocrine disorders (Cushing's disease, thyrotoxicosis, hyperparathyroidism), medications (glucocorticoid therapy, heparin), deficiency states (scurvy, malnutrition), osteogenesis imperfecta [76], and other conditions (excess alcohol consumption, celiac disease, cystic fibrosis).

Regional osteoporosis can occur in a limb with disuse (e.g., following a fracture or stroke) and around joints in inflammatory diseases (rheumatoid arthritis). There are also specific conditions, which include reflex sympathetic osteodystrophy (Sudeck's atrophy) and transient osteoporosis of the hip.

#### **Radiographic Features**

With loss of bone mass, the bones appear more radiolucent (osteopenic). The cortex of bones becomes thinned and the number of trabeculae is reduced. In the vertebrae the horizontal trabeculae are the first to be lost with preservation of the primary vertical trabeculae. This results in a prominent vertical striated appearance in the spine. In the proximal femur there is accentuation of the principal compressive and tensile trabeculae with reduction in trabecular number in Ward's area.

### Fractures (Fig. 7)

Vertebral fractures are defined as wedge, endplate, or crush [77, 78]. They are powerful predictors of future fracture (hip X2; vertebral X5). It is therefore extremely important that if they are present they are accurately reported by radiologists. There is currently a joint initiative between the International Osteoporosis Foundation (IOF), the European Society of Skeletal Radiology (Osteoporosis Group – Chairman Professor JE Adams), and the National Osteoporosis Society in the UK to improve the sensitivity and accuracy of reporting of vertebral fractures by European radiologists and an interactive teaching CD is being prepared. For further information contact the author (judith.adams@manchester.ac.uk) or manderson@osteofound.org.

Fractures also occur in the wrist and hip. Micro-fractures can occur, particularly in the sacral alar, pubic rami,



**Fig. 7.** Osteoporosis: lateral thoracic spine showing reduced bone density, several wedge vertebral fractures, and resulting kyphosis

and calcaneus [79]. There may be profuse callus formation mimicking other pathologies. These micro-fractures may be difficult to identify on radiographs due to superimposition of other structures (e.g. bowel, overlying sacrum), and radionuclide bone scans (RNS), CT, and MR imaging may be required ("Honda" sign of sacral fractures) for identification [45]. These imaging methods are also relevant to differentiating osteoporotic fractures from fractures due to other pathologies (metastases, myeloma) [80].

#### Vertebroplasty

This approach has selected application in patients with osteoporotic vertebral fracture that are persistently painful. Although performed by several medical specialists, radiologists are probably the most appropriate group to perform this image-guided interventional technique, particularly as imaging (radiographs, RNS, CT and MR) plays a role in selecting patients appropriate for the procedure [81].

#### **Bone Densitometry**

Bone mineral density (BMD) predicts about 70% of bone strength; BMD methods are therefore predictors of fracture [82]. Dual energy X-ray absorptiometry (DXA) (Fig. 8) uses ionizing radiation (albeit extremely low





densitometry: dual energy X-ray absorptiometry (DXA). a PA spine image showing analysis of vertebrae L1-4, and the result shown in relation with an appropriate genderand race-specific reference range for comparison. b PA hip image showing areas of analyses (total, neck, trochanter, Ward's area)

doses: 1-6 Sv) and is most often performed by radiographers. The equipment would therefore be appropriately housed in radiology departments. DXA of the hip and spine is currently the "gold standard" for the diagnosis of osteoporosis by bone density (WHO definition T score below -2.5) [83]. Images must be scrutinized for abnormalities that can result in errors in DXA measurements (osteophytes in lumbar spine) and for identifying vertebral fractures and other pathologies on DXA images [84]. The latter is now feasible through improvement in spatial resolution of DXA images (0.35 mm), faster fan beam scanning and, on some scanners, a "C", arm so that repositioning in the lateral position is not required; computer-assisted diagnosis is also possible [85, 86]. Bone densitometry is relevant to research and pharmaceutical trials, and thus provides scientific opportunities as well as generating income. There are some limitations of DXA (size dependency) that do not apply to QCT, which can be applied to axial and peripheral sites. The size dependency of DXA is a particular limitation of its use in growing children, in whom QCT has advantages [87]. There is increasing interest in examining how

bone size influences bone strength, and the interaction of muscle and bone. CT can provide not only true volumetric bone density (mg/cm<sup>3</sup>), but also cross-sectional area of muscle and bone, and from the latter can be derived biomechanical parameters (stress strain index: moment of inertia) [88].

## Other Metabolic Bone Disorders

#### Introduction

A number of congenital and familial disorders can be associated with increased bone density (osteosclerosis) and abnormal bone modeling. These include osteopetrosis, pyknodysostosis, metaphyseal dysplasia (Pyle's disease), craniometaphyseal dysplasia, frontometaphyseal dysplasia, osteodysplasty (Melnick-Needles syndrome), progressive diaphysial dysplasia (Camurati-Engelmann disease), hereditary multiple diaphysial sclerosis (Ribbing's disease), craniodiaphysial dysplasia, endosteal hyperostosis (Worth and Van Buchem types), dysosteosclerosis, tubular stenosis and occulodento-osseous dysplasia [89]. All are rare and have different natural histories, genetic transmission, complications and radiographic features. Many are dysplasias rather than metabolic bone disorders. The only condition to be considered in this chapter is osteopetrosis.

#### **Osteopetrosis**

In this condition, there is defective osteoclastic resorption of the primary spongiosa of bone. Osteoclasts in affected bone are usually devoid of the ruffled borders by which osteoclasts adhere to the bone surface and through which their resorptive activity is expressed. In the presence of continued bone formation, there is generalised osteosclerosis and abnormalities of metaphyseal modeling (Fig. 9) [90]. There have been reports of reversal of the osteosclerosis following successful bone-marrow transplantation.

Osteopetrosis was first described by Albers-Schonberg, in 1904, and is sometimes referred to as marble bone disease, osteosclerosis fragilis generalisata, or osteopetrosis generalisata. There are two main clinical forms:

- 1. The lethal form of osteopetrosis with precocious manifestations and an autosomal recessive transmission
- 2. Benign osteopetrosis with late manifestations inherited by autosomal dominant transmission

There is also a more rare autosomal recessive (intermediate) form that presents during childhood, with the signs and symptoms of the lethal form, but the outcome on life expectancy is not known. The syndrome previously described as osteopetrosis with renal tubular acidosis and cerebral calcification is now recognized as an inborn error of metabolism, carbonic anhydrase II deficiency. Neuronal storage disease with malignant osteopetrosis has been described, as has the rare lethal, transient infantile, and post-infectious form of the disorder.





Fig. 9a, b. Osteopetrosis. a PA chest and b AP pelvis images show very dense bones, with loss of the medullary/cortical differentiation, rib fractures, and loss of the normal modeling of the proximal femora

#### Autosomal Recessive Lethal Type

In affected individuals, there is obliteration of the marrow cavity leading to anemia, thrombocytopenia, and recurrent infection. Clinically, there is hepatosplenomegaly, hydrocephalus, and cranial nerve involvement resulting in blindness and deafness. Radiographically, all the bones are dense, with lack of corticomedullary differentiation. Modeling of affected bones is abnormal, with expansion of the metaphyseal region and undertubulation of bone. This is most evident in the long bones, particularly the distal femur and proximal humerus. Although the bones are dense, they are brittle, and horizontal pathological fractures are common. The entire skull, particularly the base, is involved and the paranasal and mastoid air cells are poorly developed. Sclerosis of endplates of the vertebral bodies produce a "sandwich" appearance. MR imaging may assist in monitoring those with severe disease who undergo marrow transplantation, since success will be indicated by expansion of the marrow cavity. Findings on MR and CT scanning of the brain have been described.

There is an intermediate recessive form of the disease which is milder than that seen in infants and distinct from the less severe autosomal dominant disease. Affected individuals suffer pathological fracture and anemia and are of short stature, with hepatomegaly. The radiographic features include diffuse osteosclerosis with involvement of the skull base and facial bones, abnormal bone modeling and a "bone within a bone" appearance.

#### **Benign**, Autosomal Dominant Type

This type of osteopetrosis (Albers-Schonberg disease) is often asymptomatic, and the diagnosis may come to light either incidentally or through the occurrence of a pathological fracture. Other presentations include anemia and facial palsy or deafness from cranial nerve compression. Problems may occur after tooth extraction, and there is an increased incidence of osteomyelitis, particularly of the mandible. Radiographic features are similar to those of the autosomal recessive form of the disease, but less severe. The bones are diffusely sclerotic, with thickened cortices and defective modeling. There may be alternating sclerotic and radiolucent bands at the ends of diaphyses, a "bone within a bone" appearance, and the vertebral endplates may appear sclerotic. In 1987, Andersen and Bollerslev classified this form of the disease into two distinct radiological types. In type 1, fractures are unusual, in contrast to type II in which fractures are common. Transverse bands in the metaphyses are more commonly a feature in type II disease, as is a raised serum acid phosphatase.

#### Hyperphosphatasia

Hyperphosphatasia is a rare genetic disorder resulting from mutations in osteoprotegerin (OPG), and is characterized by markedly elevated serum alkaline phosphatase levels [91]. Affected children have episodes of fever, bone pain, and progressive enlargement of the skull, with bowing of the long bones and associated pathological fractures (Fig. 10). Radiographically, the features resemble Paget's disease of bone, and it is sometimes referred to as "juvenile" Paget's disease, osteitis deformans in children or hyperostosis corticalis. There is an increased rate of bone turnover, with woven bone failing to mature into lamellar bone. Radiographically, this increased rate of bone turnover is evidenced by decreased bone density with coarsening and disorganisation of the trabecular pattern. In the skull, the diploic space is widened and there is patchy sclerosis. The diaphyses of the long bones become expanded, with cortical thickening along their concave aspects. The long bones may be bowed, resulting in
#### Metabolic Bone Disease





coxa vara and protrusio acetabulae. The vertebral bodies are reduced in density and in height and are biconcave. The bowing of the limbs causes affected individuals to be short in height. There is often premature loss of dentition due to resorption of dentine, with replacement of the pulp by osteoid.

The radiographic features closely resemble those of Paget's disease, but are diagnostic as they involve the whole skeleton and affect children from the age of 2 years. In contrast, Paget's disease is rare before the age of 40 years, and skeletal involvement is either monostotic or asymmetrically polyostotic. On radionuclide scanning, there is a generalized increase in uptake, giving a "super scan", due to excessive osteoblastic activity, with absence of evidence of renal uptake.

#### References

Fig. 10. Hyperphospha-

taemia: a AP tibiae

show the bones to be

expanded with thinning of the cortices, and dis-

organised trabecular

pattern b PA skull

shows massive expan-

sion of the skull vault

with sclerosis, the features resembling Paget's

disease of bone

- Mundy G.R. (1999) Bone remodelling and its disorders (2<sup>nd</sup> edn) Martin Dunitz, London, pp 1-82
- Parfitt AM (1988) Bone remodelling: Relationship to the amount and structure of bone, and the pathogenesis and prevention of fracture, in Osteoporosis – Etiology, Diagnosis and Management, (Eds B.L. Riggs and L.J. Melton), Raven Press, New York, pp 45-93
- Mundy GR (1999) Osteopetrosis, in Bone remodelling and its disorders (2<sup>nd</sup> edn) Martin Dunitz, London, pp 193-199
- Greulich WW, Pyle SI (1959) Radiographic atlas of skeletal development of the hand and wrist (2<sup>nd</sup> edn). Stanford University Press, Stanford, California
- Tanner JM, Whitehouse RH, Cameron N et al (1983) Assessment of skeletal maturity and prediction of adult height (TW2 method), 2<sup>nd</sup> edn, Academic Press, London
- 6. King DG, Steventon DM, O'Sullivan MP et al (1994) Reproducibility of bone ages when performed by radiology registrars: an audit of Tanner and Whitehouse II versus Greulich and Pyle methods, Brit J Radiol 67:848-851
- Pietka E, Gertych A, Pospiecha Euro Kurkowska S et al (2004) Computer-assisted bone age assessment:graphical user interface for image processing and comparison, J Digit Imaging 17(3):175-188
- Nelson DA, Kleerekoper M, Parfitt AM (1988) Bone mass, skin color and body size among black and white women. Bone Miner 4:257-264
- 9. Jouanny P, Guillemin F, Kuntz C et al (1995) Environmental and genetic factors affecting bone mass. Similarity of bone density among members of healthy families. Arth Rheum 38:61-67
- Ralston SH (1997) The genetics of osteoporosis. Quart J Med,90:247-251
- Krall EA, Dawson-Hughes B (1993) Heritable and life-style determinants of bone mineral density. J Bone Miner Res 8:1-9
- Sambrook PN (2005) How to prevent steroid induced osteoporosis. Ann Rheum Dis 64(2):176-178
- Hayes CW, Conway WF. (1991) Hyperparathyroidism. Radiol Clin North Am 29:85-96
- Wynne AG, Van Heerden J, Carney JA, Fitzpatrick LA (1992) Parathyroid carcinoma: clinical and pathological features in 43 patients. Medicine 71, 197-205
- Kaplan EL, Yoshiro Y, Salti G (1992) Primary hyperparathyroidism in the 1990s. Ann Surg 215:300-315
- Consensus Development Conference Panel: Diagnosis and management of asymptomatic primary hyperparathyroidism: Consensus Development Conference Statement. Ann Int Med, 114:593-397
- 17. Davies M (1992) Primary hyperparathyroidism: aggressive or conservative treatment? Clin Endocrinol 36:325-332
- Davies M, Fraser WD, Hosking DJ (2002) The management of primary hyperparathyroidism. Clin Endocrinol 57:145-155
- Genant HK, Heck LL, Lanzl LH et al (1973) Primary hyperparathyroidism: A comprehensive study of clinical, biochemical, and radiographic manifestations. Radiology 109:513-524
- Dodds WJ, Steinbach HL (1968) Hyperparathyroidism and articular cartilage calcification. Am J Roentgenol 104:884-892
- 21. Genant HK, Baron JM, Strauss FH et al (1975) Osteosclerosis in primary hyperparathyroidism. Am J Med 59:104-113
- Sundaram M (1989) Renal osteodystrophy. Skelet Radiol, 18:415-426
- 23. Wishart J, Horowitz M, Need A, Nordin BE (1990) Relationship between forearm and vertebral mineral density in postmenopausal women with primary hyperparathyroidism. Arch Intern Med 150:1329-1331
- 24. Silverberg SJ, Gartenberg F, Jacobs TP et al (1995) Increased bone density after parathyroidectomy in primary hyperparathyroidism J Clin Endocrinol Metab 80:729-734

- 25. Parfitt AM (1969) Soft tissue calcification in uraemia. Arch Internal Med, 124:544-556
- Dimich A, Bedrossian PB, Wallach S (1967) Hypoparathyroidism. Arch Intern Med 120:449-458
- Steinbach H, Waldron BR (1952) Idiopathic hypoparathyroidism: analysis of 52 cases, including report of new case. Medicine 31:133-154
- 28) Salvesen HA, Boe J (1953) Idiopathic hypoparathyroidism. Acta Endocinol 14:214-226
- 29) Adams JE, Davies M (1977) Paravertebral and ligamentous ossification, an unusual association of hypoparathyroidism. Postgrad Med J 53:167-172
- Albright F, Burnett CH, Smith PH et al (1942) Pseudohypoparathyroidism - An example of "Seabright-Bantam Syndrome". Report of 3 cases. Endocrinol 30:922-932
- 31. Steinbach HL Young DA (1966) The Roentgen appearances of pseudohypoparathyroidism (PH) and pseudo-pseudohypoparathyroidism (PPH). Differentiation from other syndromes associated with short metacarpals, metatarsals and phalangeals. Am J Roentgenol 97:49-66
- 32. Posnanski AK, Werden EA, Giedion A et al (1977) The pattern of shortening of the bones of the hands in PHP and PPHP a comparison with brachydactyly E, Turner's syndrome, and acrodysostosis. Radiology 123:707-718
- Wilson LC, Hall CM (2002) Albright's osteodystrophy and pseudohypoparathyroidism Semin Musculoskel Radiol 6(4):273-283
- Kolb FO, Steinbach HL (1962) Pseudohypoparathyroidism with secondary hyperparathyroidism and osteitis fibrosa. Journal of Clinical Endocrinology 22:59-70
- Adams JE (2005) Radiology of rickets and osteomalacia. In: Feldman D, Glorieux FH, Pike JW (eds). Chapter 60, Vitamin D, Elsevier Academic Press, San Diego, California, pp 967-994
- Pitt MJ (1981) Rachitic and osteomalacic syndromes. Radiol Clin North Am 19:582-598
- 37. Glorieux FH, St-Arnaud R (1997) Vitamin D Pseudodeficiency. In: Feldman D, Glorieux FH, Pike JW (eds) Vitamin D, Academic Press, San Diego, California, pp 755-764
- Brooks MH, Bell NH, Love L et al (1978) Vitamin D dependent rickets Type II, resistance of target organs to 1,25-dihydroxyvitamin D. New Engl J Med 293:996-999
- Pitt MJ (1993) Rickets and osteomalacia are still around. Radiol Clin North Am 29:97-118
- Brill PW, Winchester P, Kleinman PK (1998) Differential diagnosis 1: diseases simulating abuse. In: 2<sup>nd</sup> edn. Ed PK Kleinman Diagnostic imaging of child abuse. Mosby, Inc. St. Louis, Missouri, pp 178-196
- Harris HA (1933) Rickets. In: Bone growth in health and disease. Oxford Medical Publications, Oxford University Press, London, p 87
- Taybi H, Lachman R (1996) Radiology of syndromes, metabolic disorders and skeletal dysplasias. 4<sup>th</sup> edn. Mosby Year Book, St. Louis, Missouri
- Looser E (1920) Uber spatrachitis und osteomalacie Klinishe ront-genologische und pathologischanatomische Untersuchungen. Drsch Z Chir 152:210-357
- 44. Milkman LA (1930) Pseudofractures (hunger osteopathy, late rickets, osteomalacia). Am J Roentgenol 24:29-37
- 45. Hain SF, Fogelman I (2002) Nuclear medicine studies inmetabolic disease. Semin Musculoskelet Radiol 6(4):323-329
- 46. De Smet AA, Neff JR (1985) Pubic and sacral insufficiency fractures: clinical course and radiological findings. Am J Roentgenol 145:601-606
- McKenna MJ, Kleerekoper M, Ellis BI et al (1987) Atypical insufficiency fractures confused with Looser zones of osteomalacia. Bone 8:71-78
- 48. Milgram JW (1977) Radiographical and pathological assessment of the activity of Paget's disease of bone. Clin Orthop 127:63-69

- Whitehouse RW (2002) Paget's disease of bone. Semin Musculoskel Radiol 6(4):313-322
- 50. Resnick DL (1982) Fish vertebrae. Arthritis Rheumat 25:1073-1077
- Adams JE (2002) Dialysis bone disease. Semin Dial 15(4)277-289
- 52. Gipstein RM, Coburn JW, Adams JA et al (1976) Calciphylaxis in man: A syndrome of tissue necrosis and vascular calcification in 11 patients with chronic renal failure. Arch Intern Med 136:1273-1280
- Lawson J (2002) Drug-induced metabolic bone disorders. Semin Musculoskel Radiol 6:285-297
- 54. Ryan EA, Reiss E (1984) Oncogenous osteomalacia: Review of the world literature of 42 cases. Am J Med 77:501-512
- Weisman Y Hochberg Z (1994). Genetic rickets and osteomalacia. Curr Ther Endocrinol Metab 5:492-495
- 56. Steendijk R, Hauspie RC (1992) The pattern of growth and growth retardation of patients with hypophosphataemic vitamin D-resistant rickets: longitudinal study. Eur J Pediatr 151:422-427
- Glorieux FH, Marie PJ, Pettifor JM, Delvin EE (1980) Bone response to phosphate salts, ergocalciferol and calcitriol in hypophosphatemic vitamin D-resistant rickets. N Engl J Med 303:1023-1031
- Milgram JW Compere CL (1981) Hypophosphatemic vitamin D refractory osteomalacia with bilateral pseudofractures. Clin Orthop 160:78-85
- 59. O'Malley SP, Adams JE, Davies M, Ramsden RT (1988) The petrous temporal bone and deafness in X-linked hypophosphataemic osteomalacia. Clin Radiol 39:528-230
- Polisson RP, Martinez S, Khoury M et al (1985) Calcification of entheses associated with X-linked hypophosphatemic osteomalacia. N Engl J Med 313:1-6
- 61. Adams JE, Davies M (1986) Intra-spinal new bone formation and spinal cord compression in familial hypophosphataemic vitamin D resistant osteomalacia. Q J Med 61:1117-1129
- 62. Hardy DC, Murphy WA, Siegal BA et al (1989) X-linked hypophosphatemia in adults: prevalence of skeletal radiographic and scintigraphic features. Radiology 171:403-414
- 63. Econs MJ, Samsa GP, Monger M et al (1994) X-linked hypophosphatemic rickets: a disease often unknown to affected patients. Bone Miner 24:17-24
- 64. McCance RA (1947) Osteomalacia with Looser's nodes (Milkman's syndrome) due to raised resistance to vitamin D acquired about the age of 15 years. Q J Med 16:33-47
- Edminster KA, Sundaram M (2002) Oncogenic osteomalacia. Semin Musculoskelet Radiol 6(3):191-196
- Linovitz RJ, Resnick D, Keissling P et al (1976) Tumor-induced osteomalacia: a surgically curable syndrome, report of two cases. J Bone Joint Surg (Am) 58:419-423.
- Rathbun JC, MacDonald JW, Robinson HMC, Wanklin JM (1961) Hypophosphatasia: a genetic study. Arch Dis Child 36:540-542
- Weinstein RS, Whyte MP (1981) Heterogeneity of adult hypophosphatasia: report of severe and mild cases. Arch Intern Med 141:727-731
- Anderton JM (1979) Orthopedic problems in adult hypophosphatasia: a report of two cases. J Bone Joint Surg (Br) 61:82-84
- Chuck AJ, Pattrick MG, Hamilton E et al (1989) Crystal deposition in hypophosphatasia: a reappraisal. Annals Rheum Dis 48:571-576
- Mayo-Smith W, Rosenthal DI (1991) Radiographic appearances of osteopenia. Radiol Clin North Am 29:37-47
- Quek ST, Peh WC (2002) Radiology of osteoporosis. Semin Musculoskel Radiol 6(3):197-206
- Eastell R (1998) Treatment of postmenopausal osteoporosis. N Engl J Med 338:736-746
- Kanis JA, Gluer C-C (2000) An update on the diagnosis and assessment of osteoporosis with densitometry. Osteoporosis Int 11:192-202

- 75. Baur A, Stabler A, Bruning R et al (1998). Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic compression fractures. Radiology 207:349-356
- Rauch F, Glorieux FH (2004) Osteogenesis imperfecta Lancet 363(9418):1377-1385
- 77. Jiang G, Eastell R, Barrington NA, Ferrar L (2004) Comparison of methods for the visualisation of prevalent vertebral fracture in osteoporosis. Osteoporos Int 15(11):887-896
- Genant HK, Wu CY, van Kuijk C, Nevitt MC (1993) Vertebral fracture assessment using a semi-quantitative technique. J Bone Miner Res 8:1137-1148
- 79. Peh W (1996) Imaging of pelvic insufficiency fracture. Radiographics 16:335-348
- Jergas M (2003) Conventional radiographs and basic quantitative methods. In: Radiology of osteoporosis. Ed. Grampp S. Springer-Verlag Berlin Heidelberg pp 61-86
- Peh WC, Gilula LA (2003) Percutaneous vertebroplasty: indications, contraindications, and technique. Brit J Radiol 76(901):69-75
- Marshall D, Johnell O, Wedel H (1996) Meta-analysis of how well measures of bone mineral density predict occurrence of osteoporotic fractures. Br Med J 312:1254-1259
- WHO Study Group on assessment of fracture risk and its application to screening for postmenopausal osteoporosis (1994)

Technical report 843, World Health Organisation, Geneva, Switzerland, pp 5

- Adams JE (2003) Dual Energy X-ray Absorptiometry. In: Radiology of osteoporosis. Ed: Grampp S. Springer Berlin Heidelberg, pp 87-100
- Rea JA, Steiger P, Blake GM, Fogelman I (1998) Optimizing data acquisition and analysis of morphometric X-ray absorptiometry. Osteoporos Int 8(2):177-183
- Smyth PP, Taylor CJ, Adams JE (1999) Vertebral shape: automatic measurement with active shape models. Radiology, 211(2):571-578
- 87. Mughal M, Ward K Adams J (2004) Assessment of bone status in children by densitometric and quantitative ultrasound techniques. In Imaging in children. Ed. Carty H, Elsevier Science, Edinburgh. In press
- Rauch F, Schonau E (2001) Changes in bone density during childhood and adolescence: An approach based on bone's biological organization. J Bone Miner Res 16:597-604
- Greenspan A (1991) Sclerosing Bone dysplasias. Skelet Radiol 20:561-583
- Stoker DJ (2002) Osteopetrosis. Semin Musculoskelet Radiol 6(4):299-305
- Cundy T (2002) Idiopathic hyperphosphatasia. Semin Musculoskelet Radiol, 6(4):307-312

## The Radiology of Hip and Knee Joint Prostheses

I. Watt<sup>1</sup>, B.N. Weissman<sup>2</sup>

<sup>1</sup> Department of Radiology, Leiden University Medical Centre, Leiden, The Netherlands

<sup>2</sup> Department of Radiology, Brigham & Women's Hospital, Boston, MA, USA

## Introduction

Hip replacement was pioneered by Sir John Charnley, in the early 1960s. Currently, approximately 800,000 total hip arthroplasties are performed each year worldwide, with the USA accounting for more than 200,000 of them [1]. The most frequent causes of failure are loosening and particle disease. Infection is an uncommon complication, occurring in less than 1% of arthroplasties. Up to 20% of patients will need revision surgery over 20 postoperative years due to loosening and wear.

Total knee replacement is becoming a common procedure, with more total knee prostheses than total hip replacements inserted in the Medicare population in the United States in 1989 [2]. Good to excellent results are expected in 95% of appropriately selected patients.

## **Currently Used Materials**

### Metal

A number of combinations are in current use, designed to be long-wearing and inert biologically. The principle alloys used are cobalt-chrome-molybdenum, cobaltchrome-tungsten and titanium-aluminium-vanadium. The prosthesis may be inserted with cement (to transfer stress from the prosthesis to bone) or have a sintered irregular ("porous") coating, allowing bone ingrowth to occur in order to provide component fixation. Bone in-growth fixation of the femoral stem tends to be used in younger patients, in whom the anticipated greater life expectancy requires the longest possible fixation. Acetabular fixation is generally by bone in-growth.

### Ultra-High-Molecular-Weight Polyethylene

This hard, high-density material provides a low-friction articular surface for metallic components, hence the increased longevity of the device. Furthermore, it allows plastic deformity increasing congruity. However, high density polyethylene is radiolucent and cannot be readily visualized non-invasively.

#### **Other Articulations**

In an effort to decrease articular wear and the shedding of particles, which can cause loosening, other articular surfaces have been investigated. A true ceramic hip has both the acetabular cup and the femoral head made of alumina ceramic. Combinations polyethylene acetabular components with a ceramic femoral head (either alumina or zirconia ceramic) are also used.

## Main Types of Devices Used

### Hip

**Unipolar:** Usually a femoral component only (or a modular femoral component). Used mainly in older patients following a femoral-neck fracture in whom the acetabulum is relatively normal.

**Bipolar arthroplasty:** Comprising both a fixed femoral component and an acetabular component that moves within the native acetabulum. Thus, motion should occur between the native acetabulum and the acetabular component and between the femoral component and the acetabular liner. These are used mostly in younger patients. Theoretically, a later revision to a total hip replacement can be performed if necessary.

*Total hip replacement:* Both components are fixed bone. This is the most commonly used device in patients with osteoarthritis and/or the elderly.

*Hybrid total hip replacement:* The acetabular component is fixed by bone in-growth while the femoral component is cemented.

*Customised:* Following tumor resection or difficult revision arthroplasty.

#### Knee

*Unicompartmental:* Used when only one compartment needs replacement; both the femoral and tibial sides of the joint are replaced.

*Total knee replacement:* Posterior-cruciate-sparing or cruciate-substituting designs may be inserted. Fixation may be using cement or bone in-growth.

*Mobile tibial polyethylene bearings:* Rotating platform allows rotation, meniscal bearings allow rotation and anteroposterior translation.

*Constrained:* Reserved for revision surgery, severe bone loss or after tumor resection. These devices do not permit knee rotation and are subject to failure (loosening) in patients whose activity level is high.

**Patellofemoral:** Either as part of a total knee replacement, when a polyethylene "button" is cemented into the articular surface of the patella, or as a specific patellofemoral joint replacement when the major knee compartments are normal.

## **Normal Appearances**

#### **Hip Replacements**

The following features on plain film suggest an ideal position for a total hip replacement. The acetabular inclination angle should be about  $40\pm10^\circ$  on an AP view. Acetabular anteversion should measure  $0-30^{\circ}$  on a true lateral view. The femoral component should be coaxial with the femoral shaft. Acetabular screws, if used, should not penetrate into the true pelvis. No cement should be extruded. A thin, well-defined, <1.5 mm radiolucency is usual between the cement and bone. The lucent line has a sclerotic margin and develops during the first 2 years after insertion. If the lucency is 1.5-2 mm, although considered acceptable, follow-up is recommended. A metalbone lucency may be present immediately after surgery but is not optimal. Stability of position is expected on sequential radiographs for cemented devices whereas slight subsidence is acceptable, and part of the design in uncemented hips.

#### **Knee Replacements**

*Alignment:* Obtain standing views to check alignment and compare with earlier radiographs.

**Total knee:** The tibial articular surface should be parallel to the floor in a weight-bearing position. The femoral component should lie in  $5-7^{\circ}$  of valgus. The patellar button should be central and well embedded in cement.

The joint-line height is drawn from the tibial tubercle to the superior surface of the tibial component (the inferior edge of the femoral component) on the lateral radiograph. Correct for magnification.

A joint line 8 mm higher than in the preoperative examination is associated with a poorer clinical outcome. [3] A low joint line causes a low patella and may result in patellar component wear or patellar tendon rupture.

**Unicompartmental arthroplasty:** The femoral and tibial components should parallel each other, with no rotatory element, and lie parallel to floor. No overcompensation into varus or valgus into the 'normal' compartment should have occurred.

## **Bone In-growth and Porous Coating**

Porous coating, while significantly adding to the cost of joint replacement, may significantly improve implant longevity. Beads of a similar alloy are sintered onto the metallic components, permitting bony in-growth to occur without the need for intervening cement. The objective is a bone-metal fixation without an intervening fibrous layer. Clearly, this requires stability to allow in-growth to occur, with implications for the postoperative period. Anticipated normal plain-film appearances include resorption of medial femoral cortex at the calcar femoris (98%), reduced bone density where it is unloaded, the absence of a thin lucent rim around the implant, although such a lucency with a sclerotic margin is common (79%) [4]. A lucent line of more than 2 mm implies unacceptable motion. It is also normal to see endosteal sclerosis at the tip of a prosthesis (36%), localized periosteal new bone and cortical thickening, representing altered stress loading (12%) and a degree of prosthetic subsidence (7%).

### **Complications of Joint Replacement**

Patients may have complaints about their prosthetic joints, including pain and other symptoms, for which no cause may be found. This is especially true of knee replacements. In addition, not all abnormal joint replacements are associated with symptoms. Overall, complications occur in 1-5% of total hip replacements annually.

The major complications are described in the following:

#### Loosening With or Without Coexisting Infection

These are the most common complications. The infection rate is approximately 1% of total hip replacements, 2% of total knee replacements and 3% of revision joints per annum. Obviously, figures vary but at 10 years after insertion as many as 50% of hips may appear radiographically loose, 30% requiring revision [5]. Loosening and infection may be difficult to differentiate.

The major plain-film findings of loosening include the presence of cement-bone lucencies and/or cement-metal lucency of more than 2 mm in width [6]. The progressive widening of an interface, especially if associated with bone destruction, is highly suggestive. The presence of a joint effusion (shown on plain X-ray or ultrasound) implies a joint that is abnormal, although a small joint effusion is usual in total knee replacements. Additional signs include excessive component migration or subsidence of uncemented components, subsidence of cemented components, cement or fatigue fractures of metallic components, displaced fragments of metal or separated beads from porouscoated devices. A periosteal reaction should always be regarded as suspicious of infection, as opposed to local cortical thickening, which represents a stress response.

The distinction between infection and aseptic loosening may be extremely difficult but features that suggest infection include excessive bone destruction, the radiolucent zone being ill defined and irregular in outline with the absence of a sclerotic 'demarcation' line. As emphasized, periosteal new bone formation is highly suggestive of infection. On rare occasions, gas formation may be seen in soft tissues, confirming infection. However, infection is often low grade and associated with a non-virulent organism, and may be difficult to detect.

#### **Further Investigations**

#### Scintigraphy [5]

Bone-seeking <sup>99m</sup>Tc compounds demonstrate abnormal uptake for 9-12 months post-operatively. Thereafter, increased activity should be regarded as abnormal. Abnormal activity in the blood pool or perfusion phase should suggest infection, particularly if the abnormality is diffuse. In the late phase, the classical 3-point scan suggests varus tilt and loosening. Again, a diffuse increase in activity suggests infection; however, the ability to separate infection from aseptic loosening on scans is limited. A normal bone scan has a strong negative predictive value. However, for reasons that have yet to be explained, bone scans are often abnormal in otherwise uncomplicated total knee replacements and thus have a poorer positive predictive value.

<sup>67</sup>Gallium citrate is no longer popular due to problems of delivery, dose and spatial resolution. It had a high negative predictive value but a poor positive predictive value, making a negative result more useful.

<sup>111</sup>Indium labeled white blood cells afford increased sensitivity and specificity when used in combination with <sup>99m</sup>Tc bone scans but also carry a significant false-negative rate.

<sup>99m</sup>Tc-labeled ciprofloxacin has been studied to distinguish loosening from infection. Suggestive signs of infection include the extension of abnormal activity outside the area of the synovial cavity and persistence of increased activity on 24-h images. The overall sensitivity is 86% with a specificity of 78% [7]. <sup>18</sup>[F]-fluorodeoxyglucose (FDG) positron emission tomography is relatively untried, but appears to perform similarly to 3-phase bone scan but less well than conventional radiographs [8].

#### Arthrography

The major objective of arthrography is to obtain fluid for culture and sensitivity and to document intra-articular needle position. It is important to remember to aspirate material for both aerobic and anaerobic cultures. Although local anaesthetics are not proven to be bacteriostatic, aspiration prior to local anesthetic or saline injection is preferred. If the joint appears 'dry', or only a small quantity of fluid is harvested, sterile saline may be injected and re-aspirated. False-positive and false-negative cultures occur, and thus synovial biopsy is preferred by some authors.

Local anaesthetic may be injected into the articulation as a therapeutic trial (does this ablate the pain of which the patient complains?). A good PPV but 8% false negative rate has been reported.

The sensitivity of arthrography is increased when contrast medium is injected under pressure (with local anesthetic to combat induced pain) and/or the patient being asked to walk about to load the joint. Subtraction techniques increase sensitivity.

Features shown on arthrography include loosening, component failure and extra-articular collections and tracks. Specific signs at the acetabulum of loosening include the leakage of contrast at the cement-bone/metalcement interface in 90% of loose replacements and extension of contrast into pseudo-bursae in about 56%. Femoral loosening is confirmed in about 98% by contrast medium tracking into the cement-bone interface below the intertrochanteric line, or in the bone-metal interface below the intertrochanteric line. In the case of longstemmed devices, contrast medium below the level of mid-component is abnormal. Other findings shown at the hip include communication with greater trochanteric bursae (50%), supra-acetabular collections (33%) and filling of the iliopsoas bursa (17%). Lymphatic filling remains a controversial finding and is probably not significant, although it was originally thought to be a sign of inflammation [9].

At the knee, contrast under the tibial tray, or the cement interface is abnormal. Component failure, especially the non-opaque component, may be shown. The finding of a Baker's cyst may explain a patient's symptoms and signs, but in most cases is not relevant. Aspiration was shown to be 100% sensitive and specific for infection in the knee in one series.

#### **Magnetic Resonance Imaging**

MRI has received relatively little attention in the assessment of total hip and total knee replacements because of the major technical artefacts associated with metallic objects. Generally, the artefacts produced by a prosthesis reflect the orientation of the prosthesis relative to the main magnetic field, the degree of the ferromagnetic effect and the shape of the implant itself [10]. Strategies to decrease MRI artefacts with orthopedic hardware include orientating the frequency-encoding direction along the long axis of the device and reducing voxel size (increasing the number of pixels in the frequency-encoding direction does this without increasing imaging time). Thus, thin scan slices are better than thick ones and a 32-kHz bandwidth is better than a 16-kHz one [1]. Increasing the readout gradient strength to decrease misregistration artefact, the use of fast spin-echo imaging techniques, lower static magnetic field strength and a fast multiplanar inversion-recovery sequence all assist in artefact reduction. The use of less ferromagnetic hardware (e.g. titanium) also helps.

MRI scanning is of value in pre-operative planning and is superior to CT in evaluating soft-tissue pathology after a total hip replacement [9]. Normal findings include that bone marrow adjacent to the device is bright due to signal from fat; no focal masses are present and cement produces very low signal areas on all sequences. Loosening is shown by low-signal fluid collections adjacent to a component, for example, paralleling the femoral stem. Poorly defined hyperintense areas suggest infection, with signal intensity similar to fluid [10]. Synovitis, as shown by lowsignal distension of the joint pseudocapsule, may precede granuloma formation. The latter is associated with focal osteolysis and appears as discrete, well-demarcated intermediate to slightly increased signal areas with low-signal linear margins at interfaces replacing normal marrow fat [9]. Granulomas may appear as focal periprosthetic intraosseous masses with low signal intensity on T1-weighted images and as heterogeneous, predominately low to intermediate signal masses on T2-weighted images. Peripheral enhancement and some internal enhancement have been noted [1].

Early work suggested that MRI allows good visualization of femoral component complications (100% in a study of 11 patients) but poorer visualization of the acetabular area (36%) with currently available sequences. The recent adaptations suggest that periprosthetic soft tissues may be visualized better [10].

Other complications that may be demonstrated include hematomata, fat-pad scarring and heterotopic bone formation. As areas of heterotopic bone mature, the T2 signal intensity and contrast enhancement decrease while the fat and cortical bone signal increase [11]. In patients in whom recurrent dislocation is a problem, the absence of the posterior capsule and disruption of external rotator muscles have been demonstrated. Lastly, MRI may be used to evaluate vascular complications, such as pseudoaneurysm, and of vascular variations that may predispose to injury [12].

#### **Computed Tomography**

A number of important roles for CT have emerged recently [13], for example, measuring limb length and alignment pre-operatively especially, in patients with fixed flexion deformities and in prosthesis planning.

Post-operatively, CT may be used to evaluate component rotation in the knee. The tibial landmarks for measuring component rotation include the tibial tubercle or posterior tibia. It is recommended that scans are obtained perpendicular to the femoral and tibial components in order to assess rotation [14]. Fixed axes are determined to evaluate component position.

CT may also be useful to evaluate component position at the hip. However, it has been noted that the position of the pelvis changes from the supine to the standing posture, limiting the value of the measurement of component anteversion from CT scans. Acetabular anteversion, measured with relation to a horizontal baseline, is normally between 0 and  $25^{\circ}$  [13]. The inclination angle in the coronal plane normally measures between 40 and  $50^{\circ}$  [13]. Overhang of the acetabular cup of greater than 12 mm has been seen in patients with iliopsoas impingement and irritation.

CT assessment of complications includes confirming component wear, identification of granulomatous masses and detection of periprosthetic infection. Signs of the latter include periostitis (100% specificity, but 16% specificity for infection), soft-tissue infection (100% sensitivity and 87% specificity) and the presence of fluid collections (100% PPV and 96% NPV). CT may be used to demonstrate and monitor developing bone radiolucencies, where it has been shown to be more sensitive than radiographs in detecting and quantifying acetabular small-particle disease [15].

#### What investigations Are The Most Useful?

Appropriateness criteria developed by the American College of Roentgenology (see National Guideline Clearing House) are as follows, graded (1=least useful, 9=most useful).

For possible loosening, with or without infection: plain radiographs, with comparison to previous studies 9 (indicated), all other studies 1.

For possible loosening, with or without infection, but radiographs normal: joint aspiration with or without an arthrograms 8, all other studies 1.

The radiographs suggest loosening, but is the joint infected also? aspiration, with or without an arthrogram 9, all other studies 1. These criteria are currently being reevaluated.

#### **Small-Particle Disease**

This is a problem associated with increasing prosthetic life expectancy, and perhaps the demands placed upon them by their increasingly active hosts! Typically, onset begins 1-5 years after insertion and is characterized by increasing focal radiolucencies with adjacent local cortical thinning. The underlying pathological process is a granulomatous reaction with histiocytic infiltration. This reaction, as yet to have an agreed terminology (small-particle disease is the most accurate), results from the shedding of microparticles of cement, metal or polyethylene into the joint pseudocapsule [16]. The exact histology varies according to particle size. Since, characteristically, no secondary bone response occurs, as in myeloma, at one stage this change was known as 'pseudomalignant' loosening. However, prosthesis loosening may or may not be present. Nonetheless, once started, this process is relentlessly progressive, leading to eventual failure of fixation and possible fracture. The bone loss that has occurred is not retrievable and presents a major problem at revision surgery. To this end, a classification of degree and extent of bone loss, from no notable loss of bone stock to periprosthetic fracture, has been proposed [17]. The granulomatous process may breach the cortex, causing a softtissue mass, which when biopsied may be erroneously considered a malignant soft-tissue tumor.

Radiographically, the areas of radiolucency associated with this process are more difficult to assess around the knee, the distal femur being best assessed on lateral view. Tibial lesions spread along screw tracks or around peripheries of the tibial implants.

#### **Abnormal Alignment and Dislocation**

The postoperative position of a knee or hip replacement at insertion may predict early failure. For example, at the hip, a varus position risks failure. Overall, a total hip replacement fails at a rate of about 0.4-0.8% per annum, but is greater, up to 16%, in revision hips. An initial acetabular angle on an AP view of greater than 50° indicates a risk of dislocation. Similarly, the acetabular angle on the lateral view of either retroversion or more than 30° of anteversion risks dislocation. Materials, for example, cement fragments, may very rarely become postoperatively interposed, sometime after closed reduction of a dislocated prosthesis.

# Failure of Union at Trochanteric Osteotomy/Abductor Tendon Re-implantation

In order to gain access to the hip joint, some surgical approaches require reflection of the abductor muscles, with or without a part of the greater trochanter. Failure of reimplantation results in poor gait and abductor weakness. Patients with poor muscle tone or general debility are at greatest risk. MRI may be used to confirm the abnormal anatomy [18].

More rarely, failure of abduction may be due to an abductor neuropathy.

#### Fractures, Non-union and Wear

The insertion of rigid metallic implants focuses loading of the skeleton at specific points, for example, the tip of the femoral stem, rather than loading a longer area of bone. Hence, stress risers occur at these sites and may be considered a normal, perhaps 'usual', finding. These sites are at risk of fatigue and failure. Similarly, a poorly fixed metallic implant may be subject to metal fatigue. Typically, this affects a femoral implant where poor fixation has been achieved, or has developed, proximally while it it remains well fixed distally.

Similarly, fixating wires and bone cement may loosen and fragment, the latter risking the development of small-particle disease. Wear of metallic components may be obvious on plain-film or CT as a radio-opaque joint effusion.

Polyethylene elements may wear and fail. Wear usually results from friction and, eventually, when the polyethylene liner wears through or breaks, metal-metal abrasion occurs. Wear results in thinning of the nonopaque liner and an abnormal position of the components, seen on a plain film or CT [19] as a typically eccentric femoral-head position. This will be indicated by migration of an opaque element, such as the femoral head, relative to a fixed marker. It is important to distinguish wear from creep in acetabular cups. Creep represents normal plasticity of the cup, with central movement of the metallic head, whereas wear is shown by superolateral displacement of the head within the cup [20]. Wear particles cause a chronic low-grade synovitis and may result in small-particle disease. This is especially true with certain silastic implants. Polyethylene components may fracture or become displaced either due to primary failure or secondary to wear and loosening.

Lastly, a nonopaque component may become detached and dislocate within a joint. The component may be whole, worn or fragmented.

#### **Heterotopic Bone Formation**

It is common to see heterotopic new bone formation, as it occurs in about 15-50% of hip replacements. It may result in a reduced range of movement in 1-5%. The risk is greater in patients who had infection, trauma, previous hip surgery, ankylosing spondylitis or paralysis prior to surgery. The extent of ossification reflects the type of osteoarthritis (OA) preoperatively, with more occurring in patients with hypertrophic OA (15/43) than in those with atrophic (9/43) OA [21]. Heterotopic ossification may be classified by the Brooker score, from minor foci to complete ankylosis. Various forms of therapy may be employed in patients who exhibit excessive new bone formation, including radiotherapy and bone-inhibiting drugs.

#### **Cement Extrusion**

Although relatively common, for example, around the acetabular cup, this is usually asymptomatic and thought to be unimportant. Occasionally, cement injected under pressure may travel into veins, such as branches of the profunda femoris, but this is not considered dangerous. Rarely, cement extrusion causes nerve, vessel, bowel or bladder injury.

#### **Associated Malignant Tumors?**

Publications have linked the finding of a malignant tumor with a joint replacement. These may be bony or soft-tissue tumours, most often malignant fibrous histiocytoma [22]. In particular, chromosomal abnormalities have been described in lymph nodes adjacent to prosthetic joints. However, a recent meta-analysis suggests that the risk of an associated malignancy, local or general, is less than expected [23].

## References

- White LM, JK Kim, M Mehta et al (2000) Complications of total hip arthroplasty: MR imaging-initial experience. Radiology 215:254-262
- 2. Wilde AH (1993) Management of infected knee and hip prostheses. Curr Opin Rheumatol 5317-5321
- 3. Figgie HE, 3rd, Goldberg VM, Heiple KG et al (1986) The influence of tibial-patellofemoral location on function of the knee in patients with the posterior stabilized condylar knee prosthesis. J Bone Joint Surg (Am volume) 68:1035-1040
- Kaplan PA, Montesi SA, Jardon OM, Gregory PR (1988) Bone-ingrowth hip prostheses in asymptomatic patients: radiographic features. Radiology 169:221-227
- Love C, Tomas MB, Marwin SE, Pugliese PV, Palestro CJ (2001) Role of nuclear medicine in diagnosis of the infected joint replacement. Radiographics 211:229-238
- Ewald FC (1989) The Knee Society total knee arthroplasty roentgenographic evaluation and scoring system. Clin Orthop Rel Res 248:9-12
- Larikka MJ, Ahonen AK, Niemela O et al (2002) 99m Tcciprofloxacin (Infecton) imaging in the diagnosis of knee prosthesis infections. Nucl Med Comm 23:167-170
- 8. Stumpe KD, Notzli HP, Zanetti M et al (2004) FDG PET for differentiation of infection and aseptic loosening in total hip replacements: comparison with conventional radiography and three-phase bone scintigraphy. Radiology 231:333-341
- 9. Heenan SD, Chirambasukwa W, Stoker DJ (1995) Lymphatic filling in arthrography following total hip replacement. Clin Radiol 50:90-94
- Potter HG, Nestor BJ, Sofka CM et al (2004) Magnetic resonance imaging after total hip arthroplasty: evaluation of periprosthetic soft tissue. J Bone Joint Surg (Am volume) 86-A(9):1947-1954
- Ledermann HP, Schweitzer ME, Morrison WB (2002) Pelvic heterotopic ossification: MR imaging characteristics. Radiology 222: 189-195

- Hartford JM, Kwolek C, Circle B (2002) Popliteal pseudoaneurysm after total knee arthroplasty: MRI of the vascular anatomy. Orthopedics 25:187-189
- Cyteval C, Hamm V, Sarrabere MP et al (2002) Painful infection at the site of hip prosthesis: CT imaging. Radiology 224:477-483
- 14. Berger RA, Rubash HE, Seel MJ, Thompson WH, Crossett LS (1993) Determining the rotational alignment of the femoral component in total knee arthroplasty using the epicondylar axis. Clin Orthop Rel Res 286:40-47
- Puri L, Wixson RL, Stern SH,et al (2002) Use of helical computed tomography for the assessment of acetabular osteolysis after total hip arthroplasty. J Bone Joint Surg (Am volume) 84-A(4):609-614
- Schmalzried TP, Callaghan JJ (1999) Wear in total hip and knee replacements. [see comment]. J Bone Joint Surg (Am volume) 81:115-136
- Saleh KJ, Holtzman J, Gafni A et al (2001) Reliability and intraoperative validity of preoperative assessment of standardized plain radiographs in predicting bone loss at revision hip surgery. [erratum appears in J Bone Joint Surg Am 2001 Nov; 83-A(11):1712]. J Bone Joint Surg (Am volume) 83-A:1040-1046
- Twair A, Ryan M, O'Connell M et al (2003) MRI of failed total hip replacement caused by abductor muscle avulsion. Am J Roentgenol 181:1547-1550
- Duryea J, Grainger AJ, Hide IG, Genant HK, Campbell RS (2001) Fully automated software to monitor wear in prosthetic knees using fluoroscopic images. Eur Radiol 11:2184-2187
- Murray DW, O'Connor JJ (1998) Superolateral wear of the acetabulum. [see comment]. J Bone Joint Surg (Am volume) 80:197-200
- Goel A, Sharp DJ Heterotopic bone formation after hip replacement. The influence of the type of osteoarthritis. J Bone Joint Surg (Am volume) 73:255-257
- 22. Langkamer VG, Case CP, Collins C et al (1997) Tumors around implants. J Arthropl 12:812-818
- 23. Visuri T, Pukkala E, Pulkkinen P, Paavolainen P (2003) Decreased cancer risk in patients who have been operated on with total hip and knee arthroplasty for primary osteoarthrosis: a meta-analysis of 6 Nordic cohorts with 73,000 patients. Acta Orthop Scan 74(3):351-360

## **Suggested Reading**

- Mihra ST, Jones MD, Hunter TB et al (2003) Joint arthroplasties and prostheses. RadioGraphics 23:1295-1314
- Taljanovic MS, Jones MD, Hunter TB et al (2003) Joint arthroplasties and prostheses. RadioGraphics 23:1295-1314
- Weissman B (1997) Imaging of total hip replacement. Radiology 202:611-623
- National Guideline Clearing House ACR Appropriateness Criteria for evaluation of the patient with painful hip or knee arthroplasty (www.guideline.gov)

## **Traumas of the Axial Skeleton**

H. Imhof<sup>1</sup>, G.Y. El-Khoury<sup>2</sup>

<sup>1</sup> Osteology, Universitäts Klinik für Radiodiagnostik, AKH, Vienna, Austria

<sup>2</sup> Department of Radiology, University of Iowa, Iowa City, IA, USA

## **Cervical Spine Trauma**

#### Introduction

In the European Union, there are over 130,000 victims of spinal injuries each year. In the majority of cases, initial radiography is performed in community hospitals and, therefore, in the majority of cases radiography remains the basis on which the initial evaluation of a spinal injury is made. It has been shown that 46% of spinal injuries are missed by radiography compared to multi-detector row computed tomography (MDCT), and there are many advocates who stress that in multitrauma patients MDCT should be the first imaging modality.

Some 10-14% of all spinal fractures and dislocations are associated with spinal cord injury. Injuries of the cervical spine are by far the ones most commonly associated with neurological deficit which occurs in about 40% of these patients [1]. In 85% of patients, cord injury occurs at the time of the accident while in 5-10% cord injury develops very shortly thereafter [2]. The majority of cord injuries are in the lower cervical spine and at the cervico-thoracic junction (Fig. 1).



Fig. 1. Frequency of spinal fractures in correlation with segments

## **Injury Assessment**

In about 30-50% of patients, radiography alone is diagnostic and no further imaging is needed, provided that the clinical symptoms are in agreement with the imaging findings. The remaining patients 50-70% of patients should be evaluated with cross-sectional imaging. Occult fractures are best studied with MDCT while soft-tissue injuries (cord, vessels, ligaments, and muscle) or bonemarrow injuries are best studied with magnetic resonance imaging (MRI). Any questionable findings on physical examination or radiography should be thoroughly investigated because of the possible devastating sequel of a missed injury.

## **Imaging** [3, 4]

Anterioposterior (AP), lateral, oblique, and open-mouth views are initially obtained. A technically adequate lateral view should include all seven cervical vertebrae along with the upper half of the  $T_1$  vertebra. This will allow the assessment of the four longitudinal lines, which should be uninterrupted: (1) a line connecting the anterior margins of the vertebral bodies; (2) a line connecting the posterior margin of the vertebral bodies; (3) the spinolaminar line joining the junctions of the laminae with the anterior margin of the spinous processes; (4) a line joining the tips of the spinous processes (Fig. 2). On the AP view, this last line should be perfectly straight (Fig. 3).

Patients with a neurological deficit are best studied with MRI, which is capable of demonstrating cord injuries (Fig. 4). Vascular (a. vertebralis) injuries, disc herniations (Fig. 5), rupture of ligaments and (or) muscles, nerve-root disruption, and bone-marrow edema can all be evaluated with MRI. Bony abnormalities and facet joint dislocations are best seen with plain radiographs supplemented with MDCT. The standard axial CT views help in visualizing the spinal canal, facet joints, and spinous processes. Bony fragments within the spinal canal can al-



**Fig. 2.** The lateral cervical spine. Parallel lines are drawn along the anterior vertebral bodies, posterior vertebral bodies, and connecting the spinolaminar lines



**Fig. 3.** Drawing of the AP cervical spine: a line through the spinous processes should be fairly straight

**Fig. 4.** MRI of cervical spine (sagittal, T2-weighted): hyperintense bleeding within the cord and paravertebral tissues



**Fig. 5.** MRI of the cervical spine (sagittal, T2-weighted): anterior subluxation of  $C_2$  on  $C_3$  with disc herniation and cord compression. Severe edema and bleeding in the dorsal spinal parts and soft tissues

so be demonstrated (Tables 1, 2). In addition, 2D sagittal and coronal reformations are an essential part of the CT examination.

When CT and MRI are available, we do not recommend open-mouth views or oblique views of the spine. The accuracy of both modalities is much higher than that

 Table 1. Systematic inspection of the images in cervical spine trauma

Alignment	Subluxation/ dislocation	Radiographs and CT	MRI
Spinal cord	Edema		+
1	Swelling		+
	Hemorrhage	+	+
	Compression		+
	Dissection		+
Epidural space	Disk herniation	+	+
I	Bone fragment	+	
	Hematoma	+	+
Spinal column	Vertebral body fracture	+	
	Posterior element fracture	e +	
	Dislocation	+	
	Bony edema		+
	Spondylosis	+	
Ligaments	Anterior longitudinal ligament-rupture		+
	Posterior longitudinal ligament-rupture		+
	Interlaminar ligament (flava)-rupture		+
	Supra- or interspinous ligament-rupture		+
Vascular	Vertebral artery: occlusion/dissection		+

Table 2. Imaging protocol

Question	Image procedure		
• Trauma with spinal involvement?	Plain radiographs (AP, lateral)		
<ul><li>Unclear bony fracture?</li><li>Bony fragments?</li></ul>	Spiral (multi-slice) CT		
<ul><li>Facet joints?</li><li>Spinal canal?</li></ul>	HR-mode		
<ul> <li>Cord injury?</li> <li>Epidural space?</li> <li>Vascular supply?</li> <li>Disc herniation?</li> <li>Lig/ament/muscle rupture?</li> <li>Bone- marrow edema?</li> </ul>	MRI: T1-SE T2-Fast SE T2-STIR T2*-GRE T1-SE T2-Fast SE		

of plain radiographs. Dynamic views (flexion and extension) are contraindicated in the acutely traumatized spine. In the unconscious patient, all three imaging modalities, radiography, MDCT and MRI, are often required. Myelography and CT-myelography are used only in cases in which MRI is not available, and to assess dural tears or nerve-root problems, which occur only rarely.

## **Cervical Spine Instability**

The statement made by Denis more than 20 years ago, that a spine that can withstand normal physiological stresses without progressive deformity or neurological abnormalities, or both, is considered stable, is still valid today [5]. After the initial emergency treatment, the longterm survival and quality of life of the patient depend on the stability of the injury. Signs of instability on plain radiographs are presented in Table 3.

The three-column concept [5] was originally intended for the thoraco-lumbar spine, but it can be used, with some modifications, in the lower cervical spine (Table 4). According to this concept, fractures affecting both the anterior and middle columns or all three columns are considered unstable. Magerl's classification is based on biomechanics and is divided into three grades of severity (Table 5). Taking

Table 3. Radiographic findings of cervical spine instability

- Widened interspinous space or facet joints
- Anterior listhesis greater than 3.5 mm
- Narrowed or widened disc space
- Focal angulation of more than 11°
- Vertebral compression more than 25%

 Table 4. Components of the three columns of the cervical spine (after Denis)

Column	Components		
Anterior	Anterior longitudinal ligament Anterior annulus fibrosus Anterior vertebral body		
Middle	Posterior vertebral body Posterior annulus fibrosus Posterior longitudinal ligament		
Posterior	Posterior elements Facet capsules Interlaminar ligaments (flava) Supra- or interspinous ligaments		

**Table 5.** Components Radiological signs of Typ-A-/B-/C-injuries of the thoraco-lumbar spine according to Magerl

Typ A injury Compression fracture A1 Impaction fracture A2 Splitting fracture A3 Burstfracture	Reduction of vertebral body height Body splitting Enlarge interpedicle distance Intraspinal fragments
Typ B injury Distraction fracture (Injury of body and dorsal parts with distraction) B1 dorsal, ligament tear B2 dorsal, osseous tear (fracture) B3 ventral tear trough disk	Enlarged spin. proc. distance Luxation/subluxation of the facett-joints Overhanging dorsal edge Transversal fracture Fragments of dorsal body-rim
Typ C injury Torsions injury (injury of body and dorsal parts with rotation) C1 Type A-injury with rotation C2 Type B-injury with rotation C3 Rotation and shearing	Lateralisation of body Pedicle asymmetry Dislocation of spin. process Fracture of transv. process One-sided luxation / subluxation Fracture of dorsal ribs Unilaterale fracture Exarticulation

 Table 6. Functional classification of cervical spine fractures and dislocations

Mechansim of injury	Туре	Stable	Unstable
Hyperflexion	Anterior subluxation (sprain)	+	
51	Bilateral interfacetal dislocation	n	+
	Simple wedge fracture	+	
	Clay-shoveler's fracture	+	
	Tear-drop fracture		+
	Odontoid fracture	+	+
Hyperextension	Dislocation (sprain or strain)	+	
	Avulsion fracture of the posterior arch of C1	+	
	Fracture of the posterior arch	of C1	+
	Tear- drop fracture of C2	+	
	Laminar fracture		+
	Hangman's fracture		+
	Fracture or dislocation		+
Vertical	Jefferson's fracture		+
compression	Burst fracture	+	

into account the mechanism of injury, cervical spine fractures and fracture dislocations can be divided into three major groups (Table 6).

#### Hyperflexion injuries

Flexion injury of the cervical spine results in anterior angulation or translation of a vertebral segement in the sagittal plane. This injury is caused by direct trauma to the head and neck while the cervical spine is in a flexed position or by other forces that cause hyperflexion of the cervical spine.

Prominent features of flexion injuries are disruption of the posterior ligamentous complex including the interlaminar ligaments, the facet joint capsules, and the posterior part of the annulus fibrosus. In the acute phase, the injury can appear stable although the incidence of delayed instability is high, ranging from 20% to 50%. Hyperflexion injuries are commonly associated with acute disc herniation. The flexion tear-drop fracture is caused by severe flexion and axial loading. The anterior and posterior ligaments as well as the intervertebral disc are disrupted. An anterior inferior corner fracture of the vertebral body is typically present. Cord injury is commonly associated with flexion tear-drop fracture (Fig. 6). The clay-shoveler's and the simple wedge compression fracture tend to be stable, whereas the bilateral interfacetal dislocation and tear-drop fracture are unstable.

When a significant rotational component accompanies hyperflexion, unilateral or bilateral facet dislocation may occur. The vertebral body of the dislocated vertebra is anteriorly displaced. Widening of the interspinous process distance is present and the articulating facets are no longer in opposition (Figs. 6, 7).



Fig. 6. Bilateral facet-joint dislocation with anterior subluxation of  $C_5$  on  $C_6$ 



Fig. 7. Axial CT section showing left-sided facet joint fracturedislocation

#### **Hyperextension Injuries**

Extension injury of the cervical spine results in posterior angulation or translation of the injured vertebral segment in the sagittal plane. It often results from an anterior impact to the face or forehead or from sudden deceleration. The facial trauma often gives a clue to the hyperextension mechanism. Rupture of the anterior longitudinal ligament is frequently accompanied by disruption of the intervertebral disc. An avulsion fracture of the anterior arch of the atlas and small extension tear-drop fractures can be seen in  $C_2$  and  $C_3$  with hyperextension injuries.

In the more severe hyperextension injuries, at least two columns are disrupted, with resultant instability. Such fractures include the hangman's fracture, which involves the pedicles or posterior portion of the vertebral body of  $C_2$ . Effendi et al. classified the hangmans fractures into three types, depending on the location of the fracture [6].

Most commonly, this fracture occurs in frontal car accidents in which the driver and (or) passenger next to the driver did not use their seat belts (Fig. 9). When the  $C_2$ - $C_3$  disc ruptures with extension, anterior subluxation of



**Fig. 8a, b.** Flexion tear-drop (Burst) fracture of  $C_5$ . **a** Lateral radiograph of the cervical spine showing the tear-drop fragment anteriorly. **b** Axial CT showing vertebral body fractures and dislocation of the facet joints



Fig. 9. Drawing of a frontal car collision. There are anterior-flexion forces on the head (a) and hyperextension forces (b)

 $C_2$  on  $C_3$  occurs, indicating instability. Hyperextension injuries assume great importance in patients with ankylosing spondylitis, and in patients with congenital or acquired cervical stensosis.

### **Vertical Compression**

Axial loading of the cervical spine results from forces transmitted through the skull and occipital condyles to the cervical spine. Typical representatives are the Jefferson's fracture and burst fractures of the lower cervical spine (Figs. 10, 11). The Jeffersons fracture consists





**Fig. 10a, b.** Jefferson fracture. **a** Drawing with lateral overlay of the atlas. **b** Coronal tomogram demonstrating overhang of the lateral masses of the atlas with tilting to the right

of simultaneous disruption of the anterior and posterior arches of  $C_1$  with or without rupture of the transverse atlantal ligament. Detection of transverse ligament disruption, which results in atlanto-axial instability, is crucial for thorough evaluation of this injury. An overhang (i.e., lateral displacement) of the lateral masses of  $C_1$  on the lateral masses of  $C_2$  greater than 7 mm implies instability due to tearing of the transverse ligament. The openmouth view and 2D coronal CT reformations can demonstrate overhang of the  $C_1$  lateral masses.

## **Atlanto-axial Fractures**

Odontoid fractures are the most frequent injury in the atlanto-axial region. The mechanism of injury in these fractures is not well understood. Hyperflexion is believed to play a major role. Anderson and D'Alonzo [7] classified odontoid fractures into three types based on the location of the fracture (Figs. 12, 13). Type I is an avulsion fracture of the tip of the dens. Type II is the most common



**Fig. 12a-c.** Three types of odontoid process fractures. **a** Avulsion fracture of the tip of the odontoid process; **b** fracture through the base of the odontoid process; **c** fracture through the body of  $C_2$ 



**Fig. 11.** Anterior compression fracture of C<sub>7</sub>. Straightening of the cervical lordosis



**Fig. 13.** Lateral tomogram: odontoid fracture type III

odontoid fracture, and it represents a transverse fracture at the base of the dens; displacement of the type II dens is frequent and the incidence of non-union is high. In type III, the fracture line extends into the superior body of  $C_2$ .

#### **Thorocolumbar Spine Injuries**

Historically, the thoracic and lumbar spines were grouped together, but based on anatomy and biomechanisms, they can be divided into three segments:  $T_1$ - $T_{10}$ ,  $T_{11}$ - $L_4$  and  $L_5$ .

## The Upper Thoracic Spine (T<sub>1</sub>-T<sub>10</sub>)

The upper thoracic spine or  $T_1$ - $T_{10}$  is the largest and most rigid segment of the spine. About 10-20% of all spinal fractures occur in the upper thoracic spine. The  $T_1$ - $T_{10}$ segment is distinguished by the presence of the rib cage, which restricts motion and adds stiffness and stability to the spine. To produce a fracture in the upper thoracic spine, considerable energy is required; therefore, such injuries are often associated with non-contiguous vertebral fractures [8]. Of the patients with upper thoracic spine trauma, 63% present with neurological deficit due to spinal cord injury [9]. The cord damage is believed to be due to the relatively small canal size and reduced blood supply to the mid-thoracic cord [10].

There are two conditions that can mimic vertebral fractures: physiologic wedging and Scheuermann's disease. Physiologic wedging is most pronounced in the lower thoracic spine and it is especially common in males. A wedging ratio of 0.80 in males and 0.87 in females, at  $T_{8}$ - $T_{12}$  levels is considered within normal limits [11].

The other condition that mimics vertebral fractures Scheuermann's disease, is an abnormality of vertebral growth cartilage that results in vertebral wedging which persists into adulthood. In the setting of trauma, this deformity can be easily confused with a compression fracture.

# Classification of Upper Thoracic Spine Injuries (T<sub>1</sub>-T<sub>10</sub>)

Fractures of the upper thoracic spine do not fit into the Denis classification, which is intended for categorizing thoraco-lumbar junction injuries [5]. Most injuries of the upper thoracic spine occur in flexion and axial loading (Fig. 14) [10]. Bohlman classified these injuries into five types: (1) wedge compression, which is a common injury and is considered to be stable; (2) sagittal slice fracture-dislocation, which is an unstable injury and is often associated with a neurological deficit (Fig. 14c); (3) complete anterior dislocation, which is rare but very unstable; (4) posterior fracture dislocation (lumberjack paraplegia), which is an unstable injury characterized by retrolisthesis of the upper segment; and (5) burst fracture, which is produced by severe axial load-ing (Fig. 14c).

Spines that are fused by diseases such as ankylosing spondylitis, diffuse skeletal hyperostosis (DISH), or severe degenerative disc disease with bridging osteophytes are discussed separately. Fractures in this group are very unstable since all three columns are disrupted. The mechanism is due to hyperextension and such fractures can occur from relatively minor trauma [12].

#### Imaging

MDCT has been shown to be superior to radiography for imaging the spine in multi-trauma patients [13]; however, radiography is still useful for clearing the upper thoracic spine in patients who are not severely injured. A supine swimmers view of the upper thoracic spine is almost always required for visualizing the upper three thoracic vertebrae. Anteroposterior and lateral views are essential for assessing vertebral alignment, height of the vertebral bodies, endplates and disc spaces. Attention should be paid to the integrity of the lateral vertebralbody margins, pedicles and posterior vertebral body line. The posterior body line should be concave anteriorly. Mediastinal widening is seen in more than two-thirds of patients with fractures above T<sub>5</sub> and in such patients differentiating a thoracic spine fracture from aortic rupture becomes a challenge (Fig. 14a-d).

MRI is indicated for all patients who present with a neurological deficit [14]. In the acute phase, MRI is useful in looking for treatable causes for the neurological deficit, such as bony fragments compressing the spinal cord, disc herniation or epidural hematoma (Fig. 14f, g). MRI is also useful for detecting cord edema and hemorrhage, which are important in predicting prognosis. Extensive cord edema or focal cord hemorrhage are indicative of a poor prognosis [14]. Ligamentous injuries can also be directly imaged with MR.

## **Thoracolumbar Junction (T<sub>11</sub>-L<sub>2</sub>) Injuries**

Thoracolumbar spine injuries are common, accounting for about 40% of all spinal fractures. Fractures below  $L_2$ are rare. In 1983, Denis proposed the three-column concept, in which the middle column is considered pivotal in maintaining spinal stability [5]. Based on the three-column concept, Denis also described four basic fracture types of the thoracolumbar junction. These are: compression fracture, burst fracture, flexion-distraction (Chance) fracture and fracture dislocation [5].

### **Compression Fracture**

This is a common fracture, accounting for about half of all thorocolumbar junction injuries, and represents failure of



**Fig. 14a-g.** A 36-year-old woman who arrived in the emergency department with paraplegia and loss of sensation in the lower extremities after being hit by a falling tree. **a** AP radiograph of the chest shows a wide mediastinum and paravertebral soft-tissue swelling. **b** A penetrated AP view of the thoracic spine shows evidence of loss of height in the vertebral bodies of  $T_4$  and  $T_8$ , suggesting fractures in these two vertebrae. The paravertebral soft tissues show swelling. **a-d** Sagittal and coronal 2D (MDCT) reformations reveal a  $T_4$  burst fracture and  $T_8$  sagittal slice fracture. **e** Axial section through  $T_8$  shows a large retropulsed bone fragment, causing severe spinal stenosis. **f**, **g** Sagittal  $T_1$ -and  $T_2$ -weighted images show the cord to be crushed at the  $T_8$  level. There is an associated epidural hematoma anterior to the cord



the anterior column while the middle column remains intact. The posterior column can remain intact or it may fail in tension. The mechanism of injury is due to axial loading acting on a flexed spine. The fracture typically involves the superior endplate of the vertebral body producing anterior wedging and disruption of the anterior cortex.

## **Burst Fractures**

Burst fractures are relatively common, and nearly 50% are associated with neurological deficit. They are characterized by either failure of the anterior and middle columns or of all three columns (Fig. 15). The majority of burst fractures are associated with retropulsion of a bony fragment resulting in spinal stenosis. Burst fractures represent a dynamic event in which the final position of the retropulsed fragment is not representative of the canal stenosis that occurred during the injury [15]. The lateral radiograph shows disruption of the posterior vertebralbody line and displacement of the retropulsed fragment into the spinal canal (Fig. 15). Most retropulsed fragments originated from the posterior superior corner of the vertebral body. CT is the modality of choice for the evaluation of burst fractures. Patients with neurological deficit require MRI.

# Flexion-Distraction Injuries (Chance Fracture)

This is a rare injury resulting from hyperflexion, in which the axis of rotation is centered anterior to the spine [16]. The posterior and middle columns fail in tension while the anterior column fails either in tension or compression depending on whether the axis of rotation is at or anterior to the anterior column. The classical Chance fracture involves the bony elements of a single vertebra. It horizontally splits the spinous process, laminae, pedicles and vertebral body. On the lateral radiograph, there is increased height of the vertebral body posteriorly while the anterior portion of the vertebral body shows mild compression. Chance fractures have a high association with intra-abdominal injuries (45%). About 15% of patients with these injuries suffer from a neurological deficit.

## **Fracture-Dislocation**

Fracture disclocations represent one of the most serious spinal injuries, as 75% are associated with a neurological deficit. The mechanisms of injury include flexion with rotation, flexion with distraction, and shear forces. This injury is characterized by displacement of one vertebral body over an adjacent vertebral body resulting in horizontal translation or rotation at the level of the injury (Fig. 16). Fracture dislocations are very unstable injuries since all three columns are disrupted. Radiography demonstrates mal-alignment of the vertebral bodies and spinous processes. Facet dislocation is often seen in severe cases. Sagittal and coronal reformatted images and 3D images are now routinely obtained for thorough evaluation of the extent of this injury.





Fig. 15a-d. A 24-year-old man was involved in a car accident. **a**, **b** AP and lateral views of the thoracolumbar junction show evidence of burst fracture of L<sub>1</sub>. The interpedicular distance at  $L_1$  is wide and there is retropulsion of the posterior superior body of  $L_1$ . c Axial section through  $L_1$  shows the injury to be a three-column burst fracture. d Sagittal T2weighted image that shows the conus to be compressed by the retropulsed L<sub>1</sub> fragment. There is no evidence of edema or bleeding within the conus



16a-c. A 19-year-old woman who arrived at the emergency department with significant lower-extremity weakness after a motorcycle accident. a, b Sagittal 2D reformation through the mid-line and sagittal 2D reformation through the facet joints on the right side show a fracture-dislocation with anterior displacement of T<sub>12</sub> on L<sub>1</sub>. The spiral canal is arrowed and the inter-spinous distance is increased. The facet joints at this level are dislocated. c T2weighted sagittal MRI image shows compression of the conus and edema within the cord. The posterior ligaments are disrupted

#### References

- 1. Rogers LF (1982) Radiology of skeletal trauma. Churchill Livingstone, Edinburgh
- Galanski M, Wippermann B (1999) Kompendium der traumatologischen Röntgendiagnostik. Springer Verlag, Berlin-Heidelberg
- 3. Harris JH, Mirvis JH (1996) The radiology of acute cervical spine trauma. Williams & Wilkins, Baltimore
- Eustace STJ (1999) Magnetic resonance imaging of orthopaedic trauma. Lippincott Williams & Wilkins, Philadelphia
- 5. Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8:817-831
- 6. Effendi B, Roy D, Cornish B et al (1981) Fractures of the ring of the axis: A classification based on the analysis of 131 cases. J Bone Joint Surg Br 63:319-327
- Anderson LD, D'Alonzo RT (1974) Fractures of the odontoid process of the axis. J Bone Joint Surg Am 56:1663-1691
- Qaiyum M, Tyrrell PN, McCall IW, et al (2001) MRI detection of unsuspected vertebral injury in acute spinal trauma: incidence and significance. Skeletal Radiol 30:299-304
- 9. Rogers LF, Thayer C, Weinberg PE et al (1980) Acute injuries of the upper thoracic spine associated with paraplegia. AJR 134:67-73
- Bohlman HH (1985) Treatment of fractures and dislocations of the thoracic and lumbar spine. J Bone Joint Surg Am 67:165-169
- Lauridsen KN, De Carvalho A, Andersen AH (1984) Degree of vertebral wedging of the dorso-lumbar spine. Acta Radiol Diagn (Stockh) 25:29-32
- 12. Weinstein PR, Karpman RR, Gall EP et al (1982) Spinal cord injury, spinal fracture, and spinal stenosis in ankylosing spondylitis. J Neurosurg 57:609-616
- Wintermark M, Mouhsine E, Theumann N et al (2003) Thoracolumbar spine fractures in patients who have sustained severe trauma: depiction with multi-detector row CT. Radiology 227:681-689
- 14. Castillo M (1999) Current use of MR imaging in spinal trauma. Emerg Radiol 6:121-123
- Wilcox RK, Boerger TO, Allen DJ et al (2003) A dynamic study of thoracolumbar burst fractures. J Bone Joint Surg Am 85:2184-2189
- Vaccaro AR, Kim DH, Brodke DS et al (2003) Diagnosis and management of thoracolumbar spine fractures. J Bone Joint Surg Am 85:2456-2470

## Trauma of the Appendicular Skeleton

J.J. Kaye<sup>1</sup>, M.K. Dalinka<sup>2</sup>

<sup>1</sup> Department of Radiology and Radiological Sciences, Vanderbilt University Medical Center, Nashville, TN, USA

<sup>2</sup> Department of Radiology, University of Pennsylvania, Philadelphia, PA, USA

## Introduction

Trauma to the appendicular skeleton is exceedingly common. An understanding of normal radiographic anatomy is therefore essential in interpreting images of the appendicular skeleton. Certain anatomic sites are associated with particular injuries, and knowledge of the injuries that are common at those sites is important.

Problems in the diagnosis of trauma of the appendicular skeleton arise due to incomplete or inadequate examinations. In multitrauma patients, shortcuts are often taken to decrease the examination time. The examinations are frequently incomplete and the images obtained may be marginal or subopitimal. Consequently, attempts to interpret such images may lead to diagnostic errors. Incorrect diagnosis also may result from a failure to appreciate abnormalities or the significance of abnormalities on imaging studies. In addition, many errors are secondary to an inadequate or incomplete history; it has been shown that, when the history is specific, the miss rate in cases of subtle injury can be reduced by approximately 50% [1].

In addition to detecting acute injuries, in many cases radiologists are called upon to do follow-up imaging in patients with persistent pain following trauma. In these cases, the fracture may have been initially occult, or subtle findings may have been overlooked. Chronic repetitive trauma is another cause of skeletal injury in which radiographic abnormalities may not be detectable on the initial images and for which follow-up studies are frequently necessary. The ACR has developed appropriateness criteria to help address this issue [2].

## Techniques

In the long bones of the extremities, two views are generally adequate. It is important that the examinations include the proximal and distal joints, which sometimes require additional images particularly in large patients. In the case of paired long bones (the leg or forearm), the detection of the fracture of a single bone should always prompt a search for a fracture of the other paired bone or for a dislocation of the proximal or distal joint. In the case of examination of joints and in the hands and feet, three views are traditionally obtained, although the use of the opposite oblique has been suggested in the hand [3]. The exception to this may be in the hip, shoulder, or the knee, where two views are commonly sufficient. Nonetheless, whenever doubt occurs, supplemental views are often helpful.

Computed tomography (CT) may aid in the diagnosis, particularly in areas of complex anatomy (e.g., in the detection of fractures in the mid-foot or in the wrist). CT with reformatted images is often used in treatment planning in patients with fractures at the ends of long bones, particularly when joint involvement is suspected or known.

Magnetic resonance imaging (MRI) is helpful in demonstrating occult fractures (those fractures not visualized with conventional imaging techniques), in detecting incomplete fractures (particularly in the femoral neck and about the hip), and in establishing a diagnosis of fatigue and insufficiency fractures.

## Specific Sites – Upper Extremity

## Shoulder

As noted above, two views of the shoulder are often obtained in order to evaluate for traumatic abnormalities. In the past, these were typically AP views in both internal and external rotation. Currently, many centers also use a direct AP view or a Grashey view (which is a 45° oblique view of the glenohumeral joint), some combination of a scapular "Y" projection (60° anterior oblique of scapula), or an axillary or apical oblique (Garth) view (45° posterior oblique with 45° of caudal angulation) [4, 5]. Axillary views are particularly helpful in evaluating posterior dislocations of the shoulder. These views require moving the injured shoulder rather than the patient, and are sometimes difficult to obtain. They are more readily obtained by technologists than scapular-Y views. The axillary view is helpful in evaluating the glenoid process of the scapula as well as the precise location of the humeral head with respect to the glenoid.

The most common shoulder injury in the elderly population is fracture of the surgical neck of the humerus. There may also be displaced portions of the tuberosities. It is important to mention displacement of fragments, as this affects management. Fractures of the scapula, which may extend to the glenoid process and become intra-articular, are commonly seen in younger patients who have sustained severe trauma. We often obtain CT examinations in these patients to detect glenoid involvement. Other injuries commonly seen in the young and middleaged population include fractures of the clavicle.

Dislocations occurring commonly at the shoulder include acromioclavicular joint separations; these may require stress radiographs when initial images show no separation at the acromioclavicular joint. The most important aspect of the examination is the determination of the integrity of the coracoclavicular ligaments as disruption of these structures may sometimes lead to surgical therapy. When assessing the acromioclavicular joint, the inferior surface of the acromion and clavicle should be used to determine the appropriate alignment. The coracoclavicular distance may be assessed by comparison to the opposite side with both sides included on a crosswise AP image.

Anterior dislocations of the shoulder are the most common. These may be associated with fractures of the greater tuberosity or with compression fractures of the posterolateral aspect of the humeral head (Hill-Sachs deformity). Fractures of the anterior rim of the glenoid (bony Bankart deformity) may also be identified; these are best seen on axillary views of the shoulder. Posterior dislocations are far less common than anterior dislocations at the shoulder. Importantly, this injury is commonly missed; it is estimated that nearly half are missed on the initial evaluation. The use of the Grashey view, an axillary view, or scapular-Y view facilitates diagnosis. Overlap of the humeral head on the glenoid in a Grashey view indicates the presence of a dislocation. On the internal and external views, a clue may arise from the fact that the technologist is unable to obtain images in external rotation. Compression fractures of the anteromedial aspect of the humeral head are found in association with posterior dislocations, analogous to the Hill-Sachs defect seen with anterior dislocations. These compression deformities of the anteromedial aspect of the humeral head present as a trough in the humeral head and are often best seen on axillary images. When doubt exists concerning the presence of a posterior dislocation, CT may be extremely valuable to determine that a dislocation is present and to assess fracture of the humeral head. The history may be helpful in patients with posterior dislocations, which may occur following seizure disorders or electroconvulsive therapy.

Sternoclavicular dislocations may be difficult to demonstrate on conventional imaging, but are readily

seen on CT examinations. Sternoclavicular dislocations may be anterior or posterior; of these, posterior dislocations may be associated with tracheal or vascular compression, and are very serious injuries. CT allows evaluation of the airway in addition to demonstrating the dislocation.

#### Elbow

Conventional radiographic imaging of the elbow should include AP, lateral, and oblique views. Care should be taken in the evaluation of the soft tissues about the elbow, in particular, the anterior and posterior fat pads. Displacement and elevation of these fat pads is a reliable sign of intra-articular fluid. In the setting of trauma, the presence of displaced anterior and posterior fat pads at the elbow should be considered presumptive evidence of an intra-articular fracture.

In the adult, the most common elbow fractures are those of the radial head or radial neck. The cortical surfaces and margins of the radial head should be examined carefully for fracture, as should the contours of the radial neck. Comminuted fractures of the radial neck may be associated with radial shortening and malalignment at the distal radial ulnar joint. This combination is referred to as the Essex-Lopresti fracture. In view of this possibility, fractures of the radial neck with shortening should prompt an evaluation of the wrist and the forearm.

Fractures of the coronoid process of the ulna are another common fracture at the elbow. These are almost always seen in association with or following posterior dislocation of the elbow. Fractures of the olecranon are often displaced and readily identified, but some olecranon fractures are non-displaced.

Supracondylar fractures are the most common fracture of the elbow in children. Knowledge of the normal relationship of the anterior cortex of the humerus to the condyles is thus important. A line drawn along the anterior cortex of the humerus (anterior humeral line) should intersect the mid-third of the condyles; if the line intersects the anterior third of the condyle, there may be a posteriorly displaced supracondylar fracture. Fractures of the lateral condyle and medial epicondyle are also common.

The most common dislocation of the adult elbow is the posterior dislocation. These are usually quite obvious and not difficult to diagnose. A commonly missed injury is the Monteggia fracture-dislocation, in which an angulated or displaced fracture of the proximal ulna is associated with a radial head dislocation. As in the shoulder, overlap of the radial head on the capitellum on the AP image should indicate the presence of a dislocation. A line drawn along the long axis of the radial neck should intersect the capitellum in every projection (radio-capitellar line).

Avulsion fractures, especially those of the medial epicondyle, are not uncommon in children; knowledge of the order of appearance of the ossification centers at the elbow facilitates correct diagnosis, as the epicondyle may be displaced into the joint simulating the trochlear ossification center, which does not ossify until later.

#### Wrist

Radiographic examination of the wrist usually consists of three views: PA, lateral, and pronation-oblique projections. Additional views, including angle views of the scaphoid (with ulnar deviation), a "clenched fist" view, and carpal tunnel views, may be helpful in specific situations. Recently, a semisupinated oblique view was recommended for detecting subtle distal radial fractures [6].

The most common fracture at the wrist in the adult population is the Colles fracture, which is much more frequent in elderly women than in men. In this injury, there is a resultant dorsal tilt to the distal radial articular surface. Subtle injuries may be difficult to detect when displacement is minimal; these may be recognized only by the loss of the normal volar tilt to the distal radial articular surface. Fractures of the distal radius are frequently associated with injuries to the ulnar side of the wrist, including tears of the triangular fibrocartilage, dislocation of the distal radio-ulnar joint and fractures of the ulnar styloid. Such injuries often give rise to ulnar-sided wrist pain with instability and the ulnar abutment syndrome, which may require surgery.

In the carpus, fracture of the scaphoid is by far the most common fracture, accounting for approximately 70% of fractures of the carpal bones [7, 8]. A scaphoid fracture may be virtually impossible to detect on conventional imaging but clinically suspected due to pain in the anatomic snuff box. These suspected fractures may be splinted for 10-14 days and re-imaged. If there is a strong suspicion and an immediate diagnosis is necessary, MRI is an expensive but highly accurate method of diagnosis. At least one author stated that MRI is cost effective in patients with negative X-rays in whom there is a strong clinical suspicion of a scaphoid fracture [9]. The diagnosis can be made with a brief limited examination. While scaphoid fractures most commonly occur at the waist, they may also occur more proximally and more distally. The more proximal the scaphoid fracture, the more common the complications of osteonecrosis and non-union.

The next most common fracture of the carpus is the dorsal chip fracture, most often from the triquetrum. This is usually identified only on the lateral image. Another fracture along the dorsal surface of the carpus is that of the hamate. This fracture is commonly seen following carpometacarpal dislocations.

In children, epiphyseal separations at the distal radius are common, as are metaphyseal injuries. These include torus fractures, which can be identified by a buckling of the cortex (usually dorsal) of the distal radius.

An analytic approach to the diagnosis of wrist injuries was evaluated by Gilula [10], who focused primarily on conventional imaging findings of the carpal arcs, parallelism, and the overlap of articular surfaces. These criteria have withstood the test of time. Dislocation and fracture dislocations of the wrist fall into two major recognizable patterns: (1) lesser arc injuries, which are dislocations about the lunate bone, and (2) greater arc injuries, which are fractures about the vulnerable zone of the wrist [9]. Dislocations at the wrist include perilunate and lunate dislocations. Perilunate dislocations are frequently associated with fractures through the scaphoid waist (a trans-scaphoid perilunate dislocation). Virtually all perilunate dislocations are dorsal. Lunate dislocations, in contrast, are virtually all volar in direction and are rarely seen in association with other fractures at the wrist. Scapho-lunate dissociation (rotary subluxation of the scaphoid) results in abnormal rotation of the scaphoid and is due to a disruption in the scapho-lunate and volar wrist ligaments. This may occur alone or be part of other more complex injuries about the lunate axis [7].

The Galeazzi fracture is a fracture is of the distal radial shaft associated with a dislocation at the distal radial ulnar joint (i.e., the reverse of the Monteggia fracture). Isolated dislocations at the distal radioulnar joint are extremely difficult to diagnose because slight degrees of rotation of the wrist from the lateral projection may cause difficulty. When a question exists concerning the possibility of distal radioulnar dislocations, CT is the recommended technique for evaluation. Scans done in both pronation and supination are most helpful.

Less common injuries at the wrist include fractures of the hook of the hamate and of the pisiform or capitate. CT may be needed to demonstrate these fractures; reformatted images in coronal and sagittal planes should be a part of the examination.

#### Hands

Conventional imaging of the hand should include PA, a lateral view, and pronation oblique views. The internal oblique view may detect fractures that were overlooked or significantly underestimated on standard views of the hands [3].

One of the more common injuries in the hand is fracture of the distal portion of the fifth metacarpal, the "boxers fracture". While most of these fractures are identified on the PA radiograph, oblique and lateral radiographs are necessary to determine the degree of angulation.

Fractures at the bases of the metacarpals occur but are much less common than shaft fractures, except for the thumb metacarpal. Fractures at the base of the thumb metacarpal are typically associated with a dislocation and proximal displacement of the metacarpal fragment, caused by the pull of the abductor pollicis longus muscle. The dorsal fragment almost always remains in place secondary to its strong attachments (Bennett's fracture); when these are comminuted, this is termed a Rolando's fracture [8].

Carpometacarpal dislocations occur most commonly at the base of the fourth and fifth metacarpals; however, these may be difficult to recognize. The only radiographic sign on PA images may be overlap of the bases of the metacarpals on the hamate [11]. Dorsal chip fractures of the hamate may be seen on the lateral radiograph. The degree of displacement is best appreciated on this image.

In the digits, AP, lateral, and oblique projections of the digit in question should be obtained. Some authors believe that the addition of a reverse (internal) oblique is helpful [3]. The so-called baseball finger or mallet finger is a fracture of the dorsal aspect of the base of the distal phalanx of the digit, almost always accompanied by flexion of the distal interphalangeal (DIP) joint. This injury may be purely tendinous, and manifested only by flexion deformity at the DIP joint. Volar plate fractures are quite common and are seen at the volar aspect of the base of the middle phalanx. These may be impossible to identify on PA radiographs but are usually evident on oblique or lateral images [12]. Dislocations at the interphalangeal joints may be seen in association with volar plate injuries; a dislocation may have been reduced prior to imaging.

A gamekeeper's (skier's) thumb is a disruption of the ulnar collateral ligament of the metacarpo-phalangeal joint. This is often accompanied by a fracture at the site of avulsion and may require stress views for evaluation when the injury is purely ligamentous. If the adductor aponeurosis is entrapped within the joint (Stenner lesion), then surgery may be necessary. Ultrasound [13] and MRI have been advocated for the diagnosis

### **Specific Sites - Lower Extremity**

#### Hip

Fractures of the femoral neck may be displaced, with resultant shortening and external rotation of the lower extremity. Although these are readily diagnosed by conventional imaging, at times there is an apparent radiolucency in the femoral neck, suggesting that the fracture is pathologic. Usually this is not the case, and this appearance has been termed the pseudo-pathologic fracture. The area of lucency is due to rotation of the fracture fragments. When femoral-neck fractures are impacted, diagnostic problems increase. The position of the hip is usually in valgus and these fractures may be recognized as bands of density extending across the femoral neck or by a "squared-off" contour to the head-neck junction along the lateral aspect of the femoral neck. Patients with impacted fractures of the femoral neck do not present with shortening or external rotation of the extremity; they may be ambulatory at presentation, although with a limp.

Intertrochanteric fractures are common, and are frequently seen as radiolucent lines extending through the intertrochanteric region; the lesser trochanter may represent a separate bony fragment in these cases. "Isolated" fractures of the greater trochanter should raise the possibility of an incomplete intertrochanteric fracture. In patients with conventional images indicating an avulsion of the greater trochanter, MRI should be preformed in order to evaluate the intertrochanteric region for incomplete fracture. MRI has been clearly demonstrated to reveal occult fractures, that is, those not demonstrable on conventional radiography. When the clinical index of suspicion of hip fracture is high, particularly in the elderly, an MRI is often useful. A limited examination may be performed, and in many patients without a hip fracture, MRI will demonstrate other abnormalities about the hip that are responsible for the symptoms. When taken in a timely fashion, MRI can establish or exclude the diagnosis of fracture [14].

Stress and insufficiency fractures about the hip are also relatively common and may occur either in the young (fatigue fractures) or in the elderly with osteoporosis or other underlying disease (insufficiency fractures) [15]. Conventional imaging signs may be subtle or non-existent. Some patients will show vague bands of sclerosis extending across the femoral neck. Others will have no findings on conventional imaging and the presence of the stress or insufficiency fracture may only be demonstrable on MRI.

In children and adolescents, avulsion fractures about the hip are not uncommon, particularly in athletes. The most common of these include avulsion fractures from the site of origin of the hamstring muscles (the ischial tuberosity), avulsions from the straight or reflected heads of the rectus femoris (seen at the anterior inferior iliac spine or in the supra-acetabular region), and avulsions of the lesser trochanter.

Dislocations of the hip are most commonly posterior and are frequently associated with fractures of the posterior wall of the acetabulum. Osteochondral or shear fractures of the femoral head (Pipkin fractures) occur where the femoral head strikes the acetabulum at the time of posterior dislocation [16]. In a posterior dislocation, the hip is displaced posteriorly and often slightly superiorly; the thigh is held in adduction. Much less common are anterior dislocations of the hip, in which the femoral head is seen in a medial and inferior position; the thigh is held in abduction.

#### Knee

Routine imaging includes at least two views, AP and lateral. Tangential views of the patella and tunnel views may be used to supplement these, particularly when joint effusions are demonstrable. In addition, oblique views may be helpful in detecting fractures of the tibial plateau.

The presence of a knee-joint effusion following trauma to the knee in the absence of a visible fracture is frequently secondary to ligamentous injury, such as a tear of the anterior cruciate ligament, or to an intra-articular fracture. If a lipohemarthrosis is demonstrable on horizon-beam images, this is presumptive evidence for an intra-articular fracture. In these cases, CT is often the most expeditious way to demonstrate these fractures. However, CT may not be able to detect other intra-articular abnormalities. For this reason, MRI may be even more useful as it can detect ligamentous injuries, meniscal tears and chondral fractures as well as osseous and osteochondral fractures. Osteochondral injuries of the femoral condyles are often difficult to detect on conventional imaging. Tangential views of the patella may show fracture of the femoral condyle.

Avulsions of the tibial spine may occur in both children and adults, and the anterior cruciate ligaments may be avulsed with the tibial eminence. Avulsion fractures at the insertion of the posterior cruciate ligament are often only visualized on lateral images. Knowledge of the insertion point of the posterior cruciate ligament in this location should allow ready diagnosis.

Avulsion of the lateral margin of the tibial plateau, the Segond fracture, occurs at the insertion of the lateral menisco-femoral ligament. This fracture, which can be demonstrated on conventional imaging, has an extremely high association with tears of the anterior cruciate ligament. When this fracture is identified, MRI will clearly demonstrate the ligamentous injury.

In children and adolescents, Salter fractures are common in the distal femur but less common in the proximal tibia. In adolescent athletes, epiphyseal separations are more common than ligamentous injuries. Careful examination of the growth plate is warranted. Asymmetry in the width of the growth plate or small fracture fragments on the metaphyseal side of the growth plate should be sufficient to establish the diagnosis in most cases. MR may be a valuable technique when the nature of the injury is in question and also allows evaluation of ligamentous structures about the knee.

#### Ankle and Hindfoot

Conventional imaging of the ankle should include AP, internal oblique ("mortise") and lateral images. Fractures of the malleoli are common ,and careful examination for the presence of posterior malleolar fracture is necessary when another malleolar fracture is demonstrated. If a shift of the talus in the ankle mortise has occurred and no lateral malleolar fracture is demonstrated, examination of the entire length of the fibula is necessary to demonstrate higher-level fractures (Maisoneuve fracture). Isolated fractures of the posterior malleolus do occur and may only be demonstrable on lateral images.

Careful evaluation for fractures of the lateral process of the talus is necessary. These fractures typically occur in snowboarders [17]. Avulsion fractures from the neck of the talus occur at the site of insertion of the capsule. Pilon fractures represent comminuted fractures of the tibial plafond secondary to axial compression forces. They are often associated with supra-malleolar fibular fractures and they commonly exhibit depressed tibial fragments. There is typically a posterior tibial fragment that remains attached to the fibula and which is often used as a platform for repair; this frequently follows CT evaluation [18].

Calcaneal fractures are commonly depressed and frequently involve either the posterior subtalar or calcaneocuboid joint. The medial subtalar joint remains intact in approximately 75% of cases, although there may be displacement of the medial fragment [19]. The sustentacular fragment typically includes the posterior subtalar joint [20]. Involvement of these structures can sometimes be seen on tangential views of the calcaneus, although CT is typically employed in preoperative evaluation of these injuries. Isolated fractures of the anterior process of the calcaneus occur and must be distinguished from normal variants in this location. They are often missed on conventional imaging and diagnosis often follows MRI performed for persistent ankle pain. Splitting fractures of the calcaneus may only be visible on the mortise view of the ankle, during examination of the calcaneal surface of the posterior subtalar joint. They are usually readily demonstrable on tangential views of the calcaneus or on CT.

In the hindfoot, stress fractures are most common in the calcaneus and navicular. Calcaneal stress fractures are usually recognized as bands of increased density running roughly perpendicular to the normal calcaneal trabecular pattern. Stress fractures of the navicular are often difficult to see on conventional imaging and may require CT or MRI for precise diagnosis.

#### The Forefoot

The Lisfranc fracture-dislocation of the tarso-metatarsal joints is a frequent injury. This injury is easily overlooked, and a careful examination of the relationships of the metatarsal bases to the cuniforms is essential for diagnosis.

In the forefoot, stress and other fractures of the metatarsals are not uncommon. Avulsions of the base of the fifth metatarsal, at the point of insertion of the peroneus brevis muscle, should be distinguished from "dancer's fracture" or Jones fracture. These occur near the base of the fifth metatarsal, approximately 2.5 cm distal to the base, in a relatively avascular area of the metatarsal and may go on to non-union. Fractures of the phalanges in the foot are quite common. It is important to evaluate all images so that these are not overlooked. Fractures of the sesamoids may also occur; most commonly at the first metatarsal phalangeal joint. They must be distinguished from bipartite or multipartite sesamoids. The degree of separation of the parts is usually sufficient to establish the diagnosis of sesamoid fractures.

### Summary

Fractures and dislocations in the appendicular skeleton are common and careful examination of the appropriate images obtained from each site is required for diagnosis. When aware of the common locations for fractures and dislocations, the radiologist is better prepared to make the diagnosis or to recommend additional imaging techniques. When doubt occurs based upon conventional imaging findings, CT and MRI will often be useful to establish the diagnosis or to exclude a fracture.

### References

- Berbaum KS, El-Khoury GY, Franken EA, Kathol M, Montogomery WJ, Hesson W (1988) Impact of clinical history on fracture detection with radiography. Radiology 168:507-511
- 2. American College of Radiology (2000) ACR appropriateness criteria 2000. Am Coll Radiol 215
- Street JM (1993) Radiographs of phalangeal fractures: importance of the internally rotated oblique projection for diagnosis. Am J Roentgoenol 160:575-576
- Garth WP, Slappey CE, Ochs CW (1984) Roentgenographic Demonstration of Instability of the Shoulder: The Apical Oblique Projection. J Bone Joint Surg 66-A (9):1450-1452
- Kornguth PJ, Salazar AM (1987) The apical oblique view of the shoulder: Its usefulness in acute trauma. Am J Roentgoenol 149:113-116
- Russin LD, Bergman G, Miller L, Griffin WM, Walter M, Bhargavan M, Sunshine J (2003) Should the routine wrist examination for trauma be a four-view study, including a semisupinated oblique view? Am J Roentgoenol 181:1235-1238
- Johnson RP (1980) The acutely injured wrist and its residuals. Clin Orthop 149:33-44
- Jarvik JG, Dalinka MK, Kneeland JB (1991) Hand Injuries in Adults. Semin Roentgenol 25.4:282-299
- Dorsay TA, Major NM, Helms CA (2001) Cost-effectiveness of immediate MR imaging versus traditional follow-up for revealing radiographically occult scaphoid fractures. Am J Roentgoenol177:1257-1263
- Gilula LA (1979) Carpal injuries: Analytic approach and case exercises. Am J Roentgoenol 133:503-517

- Fisher MR, Rogers LF, Hendrix RW (1983) Systematic approach to identifying fourth and fifth carpometacarpal joint dislocations. Am J Roentgoenol 140:319-324
- Nance EP, Kaye JJ, Milek MA (1979) Volar plate fractures. Radiology 113:61-64
- O'Callaghan BI, Kohut G, Hoogewoud HM (1994) Gamekeeper thumb: Identification of the Stener lesion with US. Radiology 192:477-480
- 14. Bogost GA, Lizerbram EK, Crues JV III (1995) MR Imaging in evaluation of suspected hip fracture: Frequency of unsuspected bone and soft-tissue injury. Radiology 1097:263-267
- May DA, Purins JL, Smith DK. MR (1996) Imaging of occult traumatic fractures and muscular injuries of the hip and pelvis in elderly patients. Am J Roentgoenol 166-1075-1078
- Tehranzadeh J, Vanarthos W, Pais MJ (1990) Osteochondral impaction of the femoral head associated with hip dislocation: CT study in 35 patients. Am J Roentgoenol 155:1049-1052
- Boon AJ, Smith J, Zobitz ME, Amrami KM (2001) Snowboarder's talus fracture: mechanism of injury. Am J Sports Med (29)3:333-338
- Mainwaring BL, Daffner RH, Riemer BL (1988) Pylon fractures of the ankle: a distinct clinical and radiologic entity. Radiology 168:215-218
- Rosenberg ZS, Feldman F, Singson RD (1987) Intra-articular calcaneal fractures computed tomographic analysis. Skeletal Radiol 16:105-113
- Heger L, Wulff K, Seddiqi MSA (1985) Computed tomography of calcaneal fractures. AM J Roentgoenol 145:131-137

## **Inflammatory Diseases of the Spine**

V. Jevtic<sup>1</sup>, V. Pullicino<sup>2</sup>

<sup>1</sup> Clinical Radiology Institute, Clinical Centre Ljubljana, Slovenija

<sup>2</sup> The Robert Jones & Agnes Hunt Orthopaedic Hospital, Oswestry, Shropshire, UK

## **Spinal Infection**

Infective spondylitis represents 2-4% of all cases of osteomyelitis and is increasing in prevalence. Men are affected more frequently than women (2-3 to 1), usually in the fifth and six decades of the life, but infective spondylitis may appear in all age groups. The most common causative pyogenic organisms are Staphylococcus aureus, followed by streptococcus and pneumococcus. On rare occasions, spondylitis is the result of infection with gram-negative bacteria (Escherichia coli, Pseudomonas, Klebsiella, Salmonella), nonpyogenic microorganisms (Mycobacterium tuberculosis, fungi) or parasites. Typically, the lumbar spine is involved, followed in frequency by thoracic, sacral and cervical spine infection. Pyogenic spondylitis frequently follows a recent infection or surgical procedure. Immunocompromised patients are at particular risk. The general clinical signs are similar to those seen in other infectious diseases. The intensity and distribution of pain depends on the level of infection, the causative microorganism, and on host resistance. In tuberculus spondylitis, the clinical symptoms may be minor. Paraplegia or tetraplegia, the most serious complications, occurs in approximately 1% of patients, especially if the cervical spine is affected.

The hematogenous spread of infection via the arterial or venous system is the most common route of contamination. Arterial spread seems to be more frequent than transmission through Batson's paravertebral plexus. Some spinal operations and operative procedures may lead to postoperative infections. The extension of infection per continuitatem from a contiguous focus is an infrequent mechanism of spread. Direct implantation of microorganisms may follow diagnostic or therapeutic disc puncture, leading to primary infection of the intervertebral disc (discitis). In the vast majority of cases, the inflammation begins as vertebral ostemyelitis with subsequent extension of the infection into the disc space (spondylodiscitis). Septic embolus occludes vertebral end-arteries, whose location may differ according to age group. Under the age of 4 years, end arteries perforate the vertebral endplates and enter the disc space. Therefore this age group is particularly likely to develop discitis. However, during disc degeneration vascular invasion of the disc space may occur, therefore primary discitis is possible in older patients as well. In adults, arteries are distributed equatorially, with the richest networks of nutrient arterioles in the subchondral "metaphyseal" region of the vertebral bodies, which is the most common site of initial inflammation. From an anterior subchondral focus, infection spreads through the vertebral endplate into the disc space. Later on, the neighboring endplate is also destroyed, with affection of the opposite vertebral body as well. Infection may continue into the paravertebral and epidural spaces. extending subligamentously and further on through the ligaments to the paravertebral tissues. Epidural abscess may compress the spinal cord and cause paraplegia. Local pathoanatomic changes are similar to those seen in osteomyelitis at the metaphyseal region of the long bones. During the initial destructive phase, edema, hyperemia and cellular infiltration are present, leading to local bone loss. Ingrowth of fibrovascular tissue demarcates necrotic areas from the surrounding structures. Exclusion of a part of the bone from the circulation results in osteonecrosis (sequestrum). Late regenerative changes are characterised by new bone formation with osteosclerosis [1].

Radionuclide studies are highly sensitive in the detection of early inflammatory changes. An increased uptake of radiopharmaceuticals affecting two neighboring vertebral bodies may be demonstrated well before bone destruction is seen on routine radiographs or computed tomography (CT) examination. Scintigraphy is capable of showing eventual multiple infectious foci [2].

In the early stage of infection, plain films may be normal. The first radiographic signs include discrete radiolucency localized within the subchondral region, frequently anteriorly, followed by loss of definition of the endplate and narrowing of the intervertebral disc. Progression of the infection is characterized by further destruction of the vertebral body, with affection of the opposite endplate and eventual extension of inflammation through the anterior and posterior surfaces of the vertebral bodies. When the disease is well-established, paravertebral soft-tissue mass with displacement of the surrounding structures may be seen. Generally, the presence of a vacuum phenomenon excludes disc-space infection. Radiographic differentiation between pyogenic and specific spondylitis is difficult. Signs indicating tuberculosis include an absence of sclerosis, affection of the posterior elements, tendency for subligamentous and epidural extension, affection of the anterior vertebralbody surfaces, affection of a single vertebral body, preservation of the disc spaces and pronounced soft-tissue abscesses with calcifications (Fig. 1a-c). Healing is characterized by osteosclerosis and ankylosis of the intervertebral disc.

Early destructive changes are clearly demonstrated by CT examination. Effacement of paravertebral fat may be seen at the beginning of infection. Due to its high-contrast resolution, paravertebral abscess, especially its extension into the spinal canal and eventual cord compression, is best seen on CT. Calcification and sequestra are better seen on CT than on radiography or magnetic resonance imaging (MRI). In addition, CT-guided biopsy enables microbiological identification of the causative microorganism.

MRI combines the high sensitivity of scintigraphy with the high specificity of radiography and CT (Fig. 2a-d). In the acute phase, the vertebral bodies adjacent to ill-defined endplates are diffusely hypointense on T1-weighted spin-echo non-contrast images, and hyperintense on T2-weighted images. The disc space is also of high signal intensity. T1-weighted Gd-DTPA post-contrast images reveal enhancement of the vertebral bodies and of the fibrovascular tissue at the periphery of the intervertebral disc and the necrotic areas. These findings resemble the MR appearances of Modic type I lesions, seen in early disc degeneration [3]. Findings indicative of infection include high-signal-intensity intervertebral disc and the lack of a normal intranuclear cleft on T2-weighted images; widespread bone marrow edema, which frequently extends to the opposite endplates; and eventual paraspinal and epidural extension of infection, clearly demonstrated by gadolinium contrast enhancement [4]. The healing phase of spondylitis is characterized by gradual reduction of bone-marrow edema and hyperemia, leading to different degrees of fatty bone-marrow transformation, osteosclerosis, and residual vascularized fibrous tissue. MR features may suggest Modic type II and III lesions, which are seen in advanced disc degeneration.



**Fig. 1a-c.** Tuberculous spondylitis with subligamentous extension of infection from the upper cervical to the thoracic region. **a** Plain-film radiography demonstrates the widened retropharyngeal space. **b** Control radiography 2 months later shows a large retropharyngeal abscess with erosions of the anterior vertebral-body surfaces. Note also the anterior atlantoaxial subluxation due to a laxity of the ligamentum transversum, with widening of predental spaces. **c** Gd-DTPA post-contrast T1-weighted spin-echo image. Contrast enhancement of chronic inflammatory tissue within the prevertebral and epidural spaces, which extend from the cervical to the thoracic spine

Inflammatory Diseases of the Spine





Fig. 2a-d. Subacute pyogenic spondylitis at the level of L4-L5. a Plain-film radiography. Narrowing of the disc space, eroded vertebral-body endplates with pathological fracture of vertebral body L4. b CT examination. Pathological fracture is clearly demonstrated. Sequestra within the vertebral body and the paravertebral abscess. c T2weighted spin-echo image. High-signal-intensity inflammatory changes within the disc space with extension into the epidural space. d T1-weighted Gd-DTPA post-contrast images. High-signal-intensity contrast accumulation within fibrovascular tissue and the epidural abscess. Central necrotic areas are of low signal intensity

#### **Spinal Inflammation**

Inflammation of different spinal articulations may occur in rheumatic inflammatory diseases, mainly in seronegative spondyloarthritis and, less frequently, in rheumatoid arthritis. The term seronegative spondyloarthritis (or spondyloarthropaties) was introduced by Moll et al. [5] to distinguish a group of heterogeneous inflammatory rheumatic diseases from rheumatoid arthritis. The criteria for a disease to be included in this group are: the absence of rheumatoid factor in the serum (in contrast to rheumatoid arthritis, hence the term "seronegative") and the absence of subcutaneous nodules. Common features include peripheral inflammatory arthritis (often asymmetri-



cal), radiological sacroiliitis with or without radiological spondylitis, evidence of clinical overlap between members of the group, tendency of familial aggregation, significant prevalence of HLA-B27 antigen association, and the presence of different skin, nail and mucosal manifestations. The spondyloarthritides consist of several rheumatic diseases: ankylosing spondylitis, psoriatic arthritis, reactive arthritis and Reiter's syndrome, enteropathic arthritis (Crohn's disease, ulcerative colitis), juvenile chronic arthritis, and undifferentiated spondyloarthritis.

Clinically, the spondyloarthritides usually represent a combination of four syndromes: pelvic and axial manifestations, peripheral arthritis, enthesopathic syndrome and extraskeletal features. Pathoanatomically, two types of joint diseases occur in the seronegative spondyloarthritides, an inflammatory enthesopathy and a peripheral inflammatory arthritis. Inflammatory enthesopathy affects different sites of ligamentous insertions into the bone, most often those of ligaments around the intervertebral discs and the sacroiliac joint, hence the name of the whole group (spondyloarthritis). The initial phase is characterized by bone destruction at the margins of the vertebral bodies (radiological erosion) followed by exuberant bone repair and ossification within the fibers of the annulus fibrosus, forming a radiological lesion known as a syndesmophyte. The final outcome is complete ossification of the annulus. The joint is immobilized, resorbed, and replaced by bone, with a high risk of fracture [6].

During the course of the different seronegative spondyloarthritides, a variety of changes affect the discovertebral junctions, apophyseal and costovertebral joints, atlantoaxial articulations, and ligaments. Spondvlitis occurs in approximately half of all patients with ankylosing spondylitis and usually begins at the thoracolumbar and lumbosacral junctions. Subsequently, the rest of the lumbar, upper thoracic and cervical spine are affected. Radiographically, the enthesitis at the insertion of the outer fibers of the annulus fibrosus is initially demonstrated as discrete erosions of the superior and inferior portions of the vertebral bodies followed by surrounding sclerosis ("shiny" corners). These early destructive and reactive changes are called Romanus lesions, spondylitis anterior, or spondylitis marginalis (Fig. 3a, b).

More pronounced inflammatory destruction may cause "planed-down" corners which, together with anterior periosteal apposition, produce "squaring" or "barreling" of the vertebral bodies. Initial marginal destruction is followed by ossification of the outer fibers of the annulus fibrosus which extends vertically between the two neighboring vertebral bodies forming syndesmophytes, which are typical of ankylosing spondylitis. The syndesmophytes are delicate and symmetric, and connect the vertebral-body margins (marginal syndesmophytes). They are different from the parasyndesmophytes (or nonmarginal syndesmophytes) seen in psoriatic arthritis and



Fig. 3a, b. Ankylosing spondylitis with Romanus lesions. a T1weighted spin-echo image. Erosions of vertebral-body endplates surrounded by low-signal-intensity bone-marrow edema. b T1weighted spin-echo post-contrast image. Marked contrast enhancement is demonstrated within hyperemic active inflammatory tissue

Reiter's syndrome, which extend above the vertebralbody margins, are asymmetric, and relatively robust.

Complete fusion of the vertebral bodies by syndesmophytes in the late stage of ankylosing spondylitis results in what is called the "bamboo spine". Another late manifestation of ankylosing spondylitis is the advanced, discovertebral destructive Andersson's lesion, of which there are two types: type A or inflammatory; and type B or non-inflammatory. Both types occur after long-term disease. Inflammatory type A is characterized by defects of the vertebral-body endplates surrounded by broad perifocal sclerosis with narrowing of the intervertebral discs. Usually, several intervertebral discs are affected. Paucity or absence of syndesmophytes is another feature. Non-inflammatory type B may be seen even later, 10 or more years after the beginning of the disease. Typically, an ankylosed spine with numerous syndesmophytes is demonstrated. As a rule, only one intervertebral disc is affected, usually at the thoracolumbar junction. It may be widened or narrowed with pronounced bone destruction. The lesion resembles disc-space infection. It represents pseudoarthrosis due to trauma or stress and extends from the intervertebral disc to the posterior elements. The radiological differentiation between type A and type B discovertebral lesions is of practical importance since pseudoarthrosis may require spinal stabilization. Erosions, reactive sclerosis, and bony ankylosis are seen

in the apophyseal and costovertebral joints as well as ossification of interspinous and supraspinous ligaments.

Experience in the use of MRI to demonstrate the pathological processes of seronegative spondylitis is still limited. It has been shown that on Gd-DTPA MRI seronegative spondulitis has a variable signal pattern and degree of contrast enhancement, which may reflect the evolutionary stages of discovertebral enthesitis in ankylosing spondylitis. At an early stage of spinal enthesitis, the discovertebral junctions are of low signal intensity on T1-weighted spin echo images. High signal intensity on T2-weighted images and marked contrast enhancement on T1-weighted post-contrast images surrounding bone erosion reflect inflammatory edema and hyperemia. MRI may identify early erosive changes in radiographically normal vertebra. In the later stage, with syndesmophytes, MR studies show high-signal-intensity vertebral-body corners on T1-weighted and T2-weighted images without contrast enhancement. Such findings are compatible with fatty marrow transformation [7].

Affection of the occipito-atlanto-axial synovial articulations with potentially life-threatening complications is frequent in rheumatoid arthritis but may occur in psoriatic arthritis and ankylosing spondylitis. Most frequently, an anterior atlanto-axial subluxation due to laxity of the ligamentum transversum is present. Widening of the predental space by more than 3 mm in adults and 5 mm in children during flexion may be demonstrated by radiographic, CT or MR functional studies. Gd-DTPA postcontrast MRI is useful for evaluation of the inflammatory activity of synovial proliferation and for the presence of spinal cord compression. In late rheumatoid or psoriatic arthritis, pronounced erosive changes may cause vertical subluxation of the axis with basilar invagination of the odontoid. MRI studies clearly demonstrate the degree of compression of the medulla oblongata.

#### References

- 1. Resnick D, Niwayama G (1995) Diagnosis of bone and joint disorders. Saunders, Philadelphia
- 2. Rothman MI, Zoarski GH (1993) Imaging basis of disc space infection. Seminars in ultrasound, CT & MR 14(6):437-445
- 3. Modic TM, Masaryk TJ, Ross JS (1992) Magnetic resonance imaging of the spine. Year book Medical, St Louis
- 4. Jevtic V. Vertebral Infection (2004) Eur Radiol 14:43-52
- Moll JMH, Haslock I, Mac Rae IF, Wright V (1974) Associations between ankylosing spondylitis, psoriatic arthritis, Reiter's disease, the intestinal arthropathies and Behcet's syndrome. Medicine (Baltimore) 53:343-364
- Freemont AJ (1988) The pathology of ankylosing spondylitis. In: Calabro DC, Carson Dick W (eds) New clinical applications: Rheumatology: Ankylosing spondylitis. MTP, Lancaster, Penn., pp 1-22
- Jevtic V, Kos-Golja M, Rozman B, McCall I (2000) Marginal erosive discovertebral "Romanus" lesions in ankylosing spondylitis demonstrated by contrast enhanced Gd-DTPA magnetic resonance imaging. Skeletal Radiol 29:27-33

## **Degenerative Diseases of the Spine**

D. Weishaupt<sup>1</sup>, I. McCall<sup>2</sup>

<sup>1</sup> Department of Radiology, University Hospital, Zurich, Switzerland

<sup>2</sup> Department of Diagnostic Imaging, The Robert Jones & Agnes Hunt Hospital, Shropshire, UK

## Introduction

Degenerative disease of the spine, in particular low back pain (LBP), is one of the most common causes of work disability. Back pain is a pervasive problem that affects twothirds of adults at some time in their lives. Most often, back pain is benign and self-limited. However, it is occasionally the presenting symptom of systemic diseases such as cancer or infection. Some causes of back pain, especially those with neurologic symptoms, are surgically treatable. Thus, the major diagnostic task is to distinguish the 95% of patients with simple back pain from the 5% with serious underlying diseases or neurologic impairments. In this article, an overview of the spectrum of degenerative disease of the spine is provided. Special emphasis is directed to the magnetic resonance imaging (MRI) appearance of degenerative spine disorders, since MRI has become the standard of reference regarding the evaluation of patients with back pain with or without neurological deficits [1, 2].

## **Anatomical Considerations**

The intervertebral disk is a complex structure consisting of hyaline cartilage, fibrocartilage, and mucopolysaccharide and dense fibrous tissue, which together gives the spine its flexibility and stability. The layer of the hyaline cartilage attached to the vertebral endplate and encircled by the ring apophysis is called the cartilaginous endplate. Within the endplate are numerous vascular channels through which nutrients or contrast medium diffuse into the disk. One of the theories of disk degeneration is that degenerative changes in the vertebral endplate impair diffusion into and out of the disk, impeding the function of chondrocytes and fibroblasts in the disk.

The anulus fibrosus can be divided into outer and inner components, or rings. The outer ring contains the densest fibrous lamellae, which display low signal intensity on T2-weighted MR images due to the absence of ground substance. The cells in the outer ring of the anulus are almost exclusively fibroblasts. Unlike the outer ring of the anulus, the inner ring contains predominantly chondrocytes and has ground substance. Therefore, the inner ring has high signal intensity on T2-weighted images.

The second component of the intervertbral disk is the nucleus fibrosus. The nucleus fibrosus consists of collagen and hydrophilic proteoglycans.

The disk usually lacks innervation and vascularity. The anterior and posterior ligaments, facet joints, vertebral endplates, and the peripheral layer of the anulus fibrosus are innervated. Therefore, the disk is not usually a source of pain, although degeneration in the disk may lead to pain by stretching of ligamentous tissue, nerve compression, or inflammation.

### **Disk Degeneration**

With aging, the nucleus pulposus becomes dehydrated and tears occur in the anulus fibrosus. Increases in collagen and decreases in glycosaminoglycane are believed responsible for a decrease in the water content. Radial or type 3 tears are of special interest in the setting of disk degeneration since these types of anular tears concern the entire anulus fibrosus, and they correlate with shrinkage and disorganization of the nucleus [3]. Hydration and anular integrity seem to be important for the disk to absorb and transmit compressive loads to the vertebral column. As the disk ages and degenerates, it progressively loses this capacity. This results in disk-space narrowing and reduced load-bearing capacity. Occasionally, gas or calcification develops within a degenerating disk.

MRI is the most important method for clinical assessment of disk degeneration. The signal characteristics of the disk in T2-weighted sequences reflect changes caused by aging or degeneration [4]. Pfirrmann et al. [5] proposed a classification system for lumbar disk degeneration based on routine MRI. This classification system uses five grades to describe the different stages of lumbar-disk degeneration. The grading system is based on MR signal intensity, disk structure, distinction between nucleus and annulus, and disk height (Table 1, Fig. 1). The kappa coefficients for intra- and interobserver agreement were excellent for this system; thus it is useful in daily practice.

Grade	Differentiation of nucleus pulposus	Signal intensity of nucleus pulposus from anulus	Disk height	
I	Yes	Homogeneously hyperintense	Normal	
II	Yes	Hyperintense with horizontal dark band	Normal	
III	Blurred	Slightly decreased, minor irregularities	Slightly decreased	
IV	Lost	Moderately decreased, hypointense zones	Moderately decreased	
V	Lost	Hypointense, with or without horizontal hyperintense band	Collapsed	

Table 1. Classification of disk degeneration based on sagittal T2-weighted magnetic resonance (MR) images (according to [5])



Fig. 1. Grading system for the assessment of lumbar disc degeneration. Sagittal T2-weighted images show the different degrees of disk degeneration according to the classification system proposed by Pfirrmann et al. [5]

When the disk loses its anular integrity, it begins to expand outward, resulting in a variety of morphologic abnormalities. Several classification systems have been proposed to describe disk abnormalities. Currently, the most widely accepted terms are: normal, bulging, protrusion, extrusion and sequestration [6]. A disk is considered normal when it does not reach beyond the border of the adjacent vertebral bodies. Bulging is defined as circumferential, symmetric disk extension around the posterior vertebral border. The anulus fibrosus remains intact. Protrusion is defined as focal or asymmetric extension of the disk beyond the vertebral border, with the disk origin broader than any other dimension of the protrusion. Extrusion is defined as a more extreme extension of the disk beyond the vertebral border, with the base against the disk of origin narrower than the diameter of the extruding material and a connection between the material and the disk of origin (Fig. 2). Sequestration is defined as a free disk fragment that is distinct from the parent disk and has intermediate signal intensity on T1-weighted images but increased signal intensity on T2-weighed images (Fig. 3). The above-mentioned classification system for disk abnormalities does not use the term disk herniation, which is defined as displacement of disk material beyond the normal margins of the intervertebral disk space [6]. The herniated material may include nucleus pulposus, cartilage, fragmented apophyseal bone, or fragmented anular tissue. Some authors use the term disk herniation to collectively designate protrusions and extrusions.

Intervertebral disk herniation or Schmorl's node represenst an intervertebral displacement of nuclear material through a break in the vertebral endplate. Occasionally, it may present as a well-delineated cystic lesion within the vertebral body, the so-called giant cystic Schmorl's node [7]. Intravertebral disk displacement may be associated with any disease process that weakens or disrupts the endplate or subchondral bone, including intervertebral osteochondrosis, Scheuermann disease, trauma, hyper-





**Fig. 2.** T2-weighted images in the sagittal and axial planes demonstrate disk extrusion at the L4/5 disk level with compression of the right-sided L5 nerve root



Fig. 3. Sagittal T1- and T2-weighted images demonstrate a sequestrated disk at the L3/4 level. In addition, high-signal-intensity zones are visible at L3/4, L4/5 and L5/S1 disk levels

parathyroidism, osteoporosis, infection and neoplasm [8]. Chronic Schmorl's node is asymptomatic and most commonly occurs in the thoracolumar region. Acute Schmorl's node may cause pain and the surrounding vertebral bone marrow may show diffuse marrow edema [9].

When reporting imaging findings in patients with back pain, it is not only important to report the morphology, location, and size of the disk abnormality, but also to describe the relationship between the disk and the nerve root. Recently, an MR-image-based grading system of lumbar nerve-root compromise due to disk herniation was described by Pfirrmann et al. [10]. According to this classification system, the relationship between the disk and the nerve root is described as follows: no contact, contact of the disk with the nerve root without deviation of the nerve root, nerve-root deviation, or nerve-root compression. Although the grading system is primarily based on the assessment of axial images, sagittal images are also useful, in particular to detect compromise of the nerve root within the neuroforamina.

Aprill and Bogduk used the term high-signal-intensity zone (HIZ) to describe the high signal in the posterior anulus fibrosus on T2-weighted sagittal images [11]. Anular tears (synonym: anular fissure) are separations between anular fibers, avulsion of fibers from their vertebral-body insertions, or breaks through fibers involving one or many layers of the anular lamellae. The HIZ appears to be a sign of a severe form of a type II fissure [11].

In addition, to degenerative changes involving the disk structure, biochemical and structural changes also occur within the bone marrow adjacent to the endplates of the vertebral bodies. Modic et al. [2, 12] defined three different types of endplate abnormalities according to the signal abnormalities of the adjacent bone marrow: type I (inflammatory type), low signal intensity on T1-weighted images, high signal intensity on T2-weighted images when compared to fatty bone marrow; type II (fatty type), high signal intensity on both sequences; and type III (sclerotic type), low signal intensity on both sequences (Fig. 4).

**Fig. 4.** Sagittal T1- and fat-suppressed T2-weighted images demonstrate endplate changes (Modic type II) at the L4/5 disk level (*arrows*). In addition, anterolisthesis at the same level is noted

# Spondylosis Deformans and Degenerative Facet Disease

The most obvious changes in degenerative diseases of the spine are bony outgrowths along the anterior and lateral aspects of the spinal column. The outgrowths are called osteophytes or spondylosis deformans. Osteophytes arise in the setting of disk degeneration when Sharpey fibers are torn from their attachments along the vertebral-body margins, inducing a localized inflammatory response in the adjacent vertebra. Osteophytes may protrude ventrally, particularly in the lateral recesses of the spinal canal or in the intervertebral foramen.

Osteoarthritis of the facet joints is common and usually accompanies degenerative disk disease. The facet joints are true synovial joints, with hyaline articular cartilage, a synovial membrane and a joint capsule. Facet joint osteoarthritis does not differ from degenerative changes found in other synovial articulations. It is characterized by fibrillation and, later, fissuring and ulceration of the articular cartilage, which presses from the superficial to the deep cartilage layers. There is commonly a proliferative response involving the formation of osteophytes and sclerosis of subchondral bone. In addition, subchondral cysts and synovial inflammation may be present. Traditionally, CT has been used to assess the severity of facet joint osteoarthritis. However, MRI is also suitable for evaluating osteoarthritis of the facet joints [13].

## **Spinal Canal Stenosis**

Spinal canal stenosis is classified into congenital developmental stenosis (e.g., idiopathic achondroplasia, osteopetrosis) and acquired stenosis. Acquired spinal canal stenosis includes degenerative spinal canal stenosis which may further be divided into central degenerative stenosis, stenosis of the lateral recess, foraminal stenosis and degenerative spondylolisthesis. Other forms of acquired central stenosis include iatrogenic stenosis, traumatic stenosis, and miscellaneous causes of stenosis (e.g., acromegaly, Paget's disease, fluorosis, ankylosing spondylitis).

## **Cervical Spinal Stenosis**

In the cervical spine, central canal stenosis is caused by osteophytosis and ligamentous thickening. In most cases, osteophytes and disk bulge or herniation cannot be differentiated by either MRI or CT imaging and thus are sometimes referred to as disk-osteophyte complex. In the cervical spine, the width of the spinal canal is often quantitatively assessed on radiographs since such measurements are predictive for the presence of spinal canal stenosis. The spinal canal width is calculated as the ratio between the anteroposterior diameter of the spinal canal and the anteroposterior diameter of the vertebral body. In



**Fig. 5.** A 68-year-old woman with clinical symptoms of cervical spinal stenosis. Sagittal T1- and T2-weighted MR images demonstrate a narrowing the of the spinal canal at the C4-5 disk level. Hyper-intense signal is noted within the spinal cord at this level, consistent with myelomalacia

normal volunteers, this ratio is about 1. If the ratio is below 0.8, a developmental spinal canal stenosis may be present [14]. On conventional lateral radiographs the distance between the posterior surface of the vertebral body and the spinolaminar line can be measured. A spinal cord compression may be diagnosed if this distance is 10 mm or less, whereas if this distance is 13 mm or more then spinal canal stenosis is unlikely. For cross-sectional imaging modalities, measuring the cross-sectional area of the dural sac is reliable parameter for assessment of cervical spine stenosis. A cross-sectional area of 60 mm<sup>2</sup> has been reported to be predictive for cervical spine stenosis. Spinal canal stenosis may result in cervical myelopathy, which presents as high signal intensity on T2-weighted images (Fig. 5). Ossification of the posterior longitudinal ligament (OPLL) often results in a central cervical spinal stenosis. OPLL is more frequently present in men than in women and typically manifests in the fifth to seventh decade of life. The diagnosis of OPLL is established by its characteristic appearance on conventional radiography or CT imaging. In the cervical and, infrequently, in the thoracic spine, a dense ossified strip of variable thickness is evident along the posterior margins of the vertebral bodies and the intervertebral disk. OPLL may extend over multiple levels, but also can be segmental.

#### Lumbar Spinal Stenosis

Myelography has for many years been the method of choice for assessment of lumbar spinal canal stenosis. For clinical purposes, an anteroposterior diameter of the dural sac of 10 mm is indicative of absolute stenosis and 12 mm suggests relative stenosis. Using CT and MRI, measurement of the cross-sectional area of the dural sac is probably the most reliable technique for assessment of the width of the spinal canal. If the area of the dural sac is below 75 mm<sup>2</sup>, the likelihood of a stenosis is high. Loss of high-signal cerebrospinal fluid (CSF) around the nerve roots or cord on T2-weighted axial images is also valuable for evaluating clinically relevant spinal stenosis. CT and MRI are not only useful to detect central lumbar stenosis, but also to diagnose stenosis at the lateral recess. The lateral recess is bordered posteriorly by the superior articular facet, laterally by the pedicle and anteriorly by the vertebral body and disk. Lumbar lateral recess stenosis occurs when a hypertrophic superior facet encroaches on the recess, often in combination with narrowing due to a bulging disk and osteophyte. Foraminal stenosis occurs when a hypertrophic facet, vertebral-body osteophyte, or bulging disk narrows the neural foramen and encroaches on the nerve roots. When the epidural fat surrounding the nerve root within the foramen is obliterated, as seen on sagittal T1-weighted scans, marked encroachment is present.

# Correlation Between Clinical and Imaging Findings

Magnetic resonance imaging has extensively been used in the identification of abnormal conditions of the lumbar spine and has become the gold standard in evaluation of spinal pathology. However, particularly in studying patients with low back pain, there is often a discrepancy between symptoms reported by the patient and findings as documented by MRI. In addition, previous studies reported a high rate of abnormal imaging findings in the lumbar spine of asymptomatic volunteers (Table 2) [15-22].

Since disk abnormalities, including disk bulging, disk protrusion and disk extrusion, are common in asymptomatic volunteers, they cannot be used easily as parameters in clinical management. Only the presence of a disk extrusion may represent a clinical significant finding if the symptoms of the patient correspond to the imaging findings. In a study in asymptomatic volunteers who were matched according to age, sex and occupational risk factors to patients with symptoms of disk herniation, Boos et al. [19] showed that the only substantial morphological difference between symptomatic patients and asymptomatic volunteers was the presence of neural compromise (83% vs. 22%). Thus, neural compromise may have a more important role in explaining pain than does morphologic extension of disk material beyond the intervertebral space.

The pathophysiologic mechanisms that cause nerveroot symptoms are still not completely understood. Currently, two concepts are discussed: mechanical nerveroot compression and chemically induced nerve-root inflammation caused by the nucleus pulposus [23]. The latter concept may explain why patients suffer from radiculopathy even though nerve root compression is not visible on MRI.

Table 2. Prevalence of disk abnormalities in healthy volunteers

			-				
Author, year, test	Age group (n)	HNP	Bulging disk	Degenerative disk	Stenosis	Annular tear (high signal intensity zone)	Other
Weishaupt (1998), MRI	20-50 years, mean=35, <i>n</i> =60	60% <sup>c</sup>	20%	72%	_	33%	Nerve-root contact or deviation, 26%; nerve-root compression, 2%; thecal sac impression, 17%
Stadnik, (1998), MRI	17-71 years, median 42; <i>n</i> =36 <sup>a</sup>	33%	81%	72%	_	56%	-
Savage (1997), MRI	20-30 years, <i>n</i> =78; 31-58 years, <i>n</i> =71	_	_	_	_	-	Any abnormality 32%
Burns (1996), MRI	21-31 yreas, <i>n</i> =41	_	_	_	_	_	Any abnormality 85%
Boos (1995), MRI	20-50 years, mean=36 years, $n=46^{b}$	76%	51% of disks	85%	_	_	No sequestered disks; nerve- root contact or deviation, 22%
Jensen (1994), MRI	20-80 years, mean=42, <i>n</i> =98	28%	52%	_	7%	14%	64% had disk bulge, protrusion or extension
Boden (1990), MRI	<60 years, <i>n</i> =53; >60 years, <i>n</i> =14	22%, 36%	54%, 79%	46%, 93%	1%, 21%	-, -	-
Weinreb (1989), MRI	Women 19-24 years, mean=28, <i>n</i> =86	9%	44%	_	_	_	-

HNP, herniated nucleus polposus

<sup>a</sup> Referred for head or neck imaging

<sup>b</sup> Patients matched for age and occupational exposure

<sup>c</sup> Numbers represent prevalence per subject

Another important MRI finding that is probably related to discogenic pain is endplate abnormalities. Our own study [24] in 50 patients with discogenic pain has shown that type I and II endplate abnormalities are highly predictive for the presence of painful disk when compared to discography. A recent longitudinal study has shown that type I endplate changes are dynamic lesions that either increase in size or convert to type II, and that if the type I lesion does convert to type II, it starts to do so within 2 years in most cases. There is also evidence, although not at a statistically significant level, that conversion from type 1 to type 2 is related to an improvement in the patient's back pain [25].

The clinical significance of HIZ is also debatable. The results of two studies [11, 26] have shown a high correlation between the presence of HIZ and pain concordant with the usual symptoms at discography. However, the high prevalence of HIZ in asymptomatic volunteers, as reported by three studies [16-18], indicates that these results should be interpreted with care. A recent study of the natural history has shown that HIZ often remains unchanged for several years, and there was no correlation between resolution or increased severity of HIZ and changes in symptoms [27].

Another controversial issue in imaging of the degenerative spine is the influence of the body position on MRI findings. Several cadaver studies have shown that flexion and extension, lateral bending, and axial rotation significantly change the anatomic relationships of the ligamentum flavum and intervertebral disk to the spinal nerve roots in the lumbar spine. The authors refer to compression of the spinal canal or spinal nerve roots occurring exclusively during axial loading and/or spinal motion as "dynamic stenosis" [28]. Based on these data, clinically relevant spinal canal and foraminal stenosis, as well as the degree of nerve-root compression, may not be demonstrable on conventional MR systems with the patient in the supine position. Several attempts have been undertaken in order to overcome the limitations of conventional MRI. Willen and coworkers [29] have described a portable device allowing for axially loaded lumbar-spine imaging using conventional MR scanners and with the patient in supine position. Alternatively, MRI with the patient in an upright position can be performed in vertically open-configuration MR systems, which allow imaging in seated or even in standing positions. Preliminary experience [30] has shown that, although these systems may demonstrate abnormal findings that are not visible in supine body position, the use of axially loaded MRI should be reserved for selected patients with low back pain.

### References

- Kent DL, Haynor DR, Longstreth WT Jr, Larson EB (1994) The clinical efficacy of magnetic resonance imaging in neuroimaging. Ann Intern Med 120:856-871
- Modic MT, Masaryk TJ, Ross JS, Carter JR (1988) Imaging of degenerative disk disease. Radiology 168:177-186
- Yu SW, Haughton VM, Ho PS, Sether LA, Wagner M, Ho KC (1988) Progressive and regressive changes in the nucleus pulposus. Part II. The adult. Radiology 169(1):93-97
- Pearce RH, Thompson JP, Bebault GM, Flak B (1991) Magnetic resonance imaging reflects the chemical changes of aging degeneration in the human intervertebral disk. J Rheumatol Suppl 27:42-43
- Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N (2001) Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine 26(17):1873-1878
- Fardon DF, Milette PC (2001) Nomenclature and classification of lumbar disc pathology. Recommendations of the Combined task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine 26(5):E93-E113
- Hauger O, Cotten A, Chateil JF, Borg O, Moinard M, Diard F (2001) Giant cystic Schmorl's nodes: imaging findings in six patients. Am J Roentgenol 176(4):969-972
- Resnick D, Niwayama G (1995) Degenerative disease of the spine. In: Resnick D (ed) Diagnosis of bone and joint disorders. 3rd edn. Saunders, Philadelphia, pp. 1372-1462
- Wagner AL, Murtagh FR, Arrington JA, Stallworth D (2000) Relationship of Schmorl's nodes to vertebral body endplate fractures and acute endplate disk extrusions. Am J Neuroradiol 21(2):276-281
- Pfirrmann CW, Dora C, Schmid MR, Zanetti M, Hodler J, Boos N (2004) MR image-based grading of lumbar nerve root compromise due to disk herniation: reliability study with surgical correlation. Radiology 230(2):583-588
- Aprill C, Bogduk N (1992) High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol 65(773):361-369
- Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR (1988) Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. Radiology 166(1 Pt 1):193-199
- Weishaupt D, Zanetti M, Boos N, Hodler J (1999) MR imaging and CT in osteoarthritis of the lumbar facet joints. Skeletal Radiol 28(4):215-219
- Pavlov H, Torg JS, Robie B, Jahre C (1987)Cervical spinal stenosis: determination with vertebral body ratio method. Radiology 164(3):771-775
- Weinreb JC, Wolbarsht LB, Cohen JM, Brown CE, Maravilla KR (1989) Prevalence of lumbosacral intervertebral disk abnormalities on MR images in pregnant and asymptomatic nonpregnant women. Radiology 170(1 Pt 1):125-128

- 16. Weishaupt D, Zanetti M, Hodler J, Boos N (1998) MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. Radiology 209:661-666
- Stadnik TW, Lee RR, Coen HL, Neirynck EC, Buisseret TS, Osteaux MJ (1998) Annular tears and disk herniation: prevalence and contrast enhancement on MR images in the absence of low back pain or sciatica. Radiology 206(1):49-55
- Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS (1994) Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 331(2):69-73
- Boos N, Rieder R, Schade V, Spratt KF, Semmer N, Aebi M (1995) Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. Spine 20:2613-2625
- Burns JW, Loecker TH, Fischer JR Jr, Bauer DH (1996) Prevalence and significance of spinal disc abnormalities in an asymptomatic acceleration subject panel. Aviat Space Environ Med 67(9):849-853
- Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW (1990) Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am 72(3):403-408
- 22. Savage RA, Whitehouse GH, Roberts N (1997) The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. Eur Spine J 6(2):106-114
- 23. Olmarker K, Rydevik B, Nordborg C (1993) Autologous nucleus pulposus induces neurophysiologic and histologic changes in porcine cauda equina nerve roots. Spine 18(11):1425-32
- Weishaupt D, Zanetti M, Hodler J, Min K, Fuchs B, Pfirrmann CW, et al (2001) Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. Radiology 218(2):420-427
- Mitra D, Cassar-Pullicino VN, McCall IW (2004) Longitudinal study of vertebral type-1 end-plate changes on MR of the lumbar spine. Eur Radiol 14(9):1574-1581
- 26. Schellhas KP, Pollei SR, Gundry CR, Heithoff KB (1996) Lumbar disc high-intensity zone. Correlation of magnetic resonance imaging and discography. Spine 21(1):79-86
- Mitra D, Cassar-Pullicino VN, McCall IW (2004) Longitudinal study of high intensity zones on MR of lumbar intervertebral discs. Clin Radiol 59(11):1002-1008
- Nowicki BH, Yu S, Reinartz J, Pintar F, Yoganandan N, Haughton VM (1990) Effect of axial loading on neural foramina and nerve roots in the lumbar spine. Radiology 176:433-437
- Willen J, Danielson B, Gaulitz A, Niklason T, Schonstrom N, Hansson T (1997) Dynamic effects on the lumbar spinal canal: axially loaded CT-myelography and MRI in patients with sciatica and/or neurogenic claudication. Spine 22:2968-2976
- 30. Weishaupt D, Schmid MR, Zanetti M, Boos N, Romanowski B, Kissling RO et al (2000) Positional MR imaging of the lumbar spine: does it demonstrate nerve root compromise not visible at conventional MR imaging? Radiology 215:247-253

# **Osteomyelitis and Septic Arthritis**

D. Forrester<sup>1</sup>, R.F. Kilcoyne<sup>2</sup>

<sup>1</sup>University of Southern California Keck School of Medicine, Los Angeles, CA, USA

<sup>2</sup> Department of Radiology, A-030, University of Colorado Health Sciences Center, CO, USA

## Introduction

Infections in bones and joints are usually considered from the point of view of the timing of their presentation – acute, subacute or chronic. The imaging presentation is related to this timing sequence. Most of the classic terms applied to osteomyelitis refer to chronic osteomyelitis, but the ability to make the diagnosis clinically at an earlier stage of disease is important. In some cases, imaging can help in this earlier diagnosis [1]. As we will see, advanced imaging techniques play a role in early diagnosis [2].

## **Features of Osteomyelitis**

### Acute Osteomyelitis

The initial clinical presentation of acute osteomyelitis will depend on the history and physical findings. Radiography is often negative in the early stages of infection. It may take two weeks for the radiographs to become positive. Treatment with antibiotics may be needed before radiographs become positive. Bone scintigraphy or magnetic resonance imaging (MRI) may confirm acute osteomyelitis and show the extent of its spread. Sequential radiographs will document the response to therapy. The lack of a clinical response may be an indication for biopsy in order to confirm the infecting organism or to rule out a tumor that is mimicking osteomyelitis, such as Ewing's sarcoma and lymphoma.

### Subacute Osteomyelitis

Brodie's abscess is a term applied to one form of subacute osteomyelitis. The radiographic signs are typical -adiscrete radiolucent lesion in the shaft of a long bone. The margins are usually sharply defined, indicating the slow progression of the infection. A typical feature is a linear tract extending from the medullary cavity to the cortex or through the cortex into the soft tissues (Fig. 1). A more



Fig. 1. Lateral radiograph of the tibia of a child shows a discrete radiolucent area in the medullary cavity with sharply defined margins. Extending superiorly is a linear lucent tract that has not yet reached the cortex. This linear tract is typical of Brodie's abscess

latent form of subacute or chronic infection is sclerosing osteomyelitis of Garré. The radiographic signs are usually nonspecific. There is a focal area of increased density in the medullary cavity. Because of pain, the lesion may need to be biopsied in order to rule out a slow-growing tumor, such as osteoid osteoma. The pathologist will find signs of chronic inflammation, but frequently no organism can be cultured.

### **Chronic Osteomyelitis**

The body reacts to chronic infection in bone by destroying bone and producing new bone. Classic terms have been applied to various clinical stages and appearances. *Periosteal cloaking* is the new bone surrounding an area of medullary infection in a long bone. A similar type of healing response in the periosteum in the case of fracture is called callus. *Sequestrum* is the term for an area of dead infected bone that has lost its blood supply. Because the surrounding area is undergoing bone resorption secondary to the inflammatory response, the dead bone appears whiter than the remainder (Fig. 2). *Involucrum* is healing bone surrounding a sequestrum or under elevated periosteum. It may be seen on radiographs as an area


Fig. 2. AP radiograph of the distal femur of a 15-year-old male shows findings of chronic osteomyelitis. The linear dense area in the femoral cortex (circle) is a sequestrum of dead bone

of bone resorption or radiolucency. Cloaca is a linear tract in bone, commonly seen with Brodie's abscess (mentioned above). Extension of the tract from the bone into the soft tissues or the skin is a sinus tract. A classification of chronic osteomyelitis can take into account clinical presentation and method of spread of infection. Hematogenous osteomyelitis develops as a result of a blood-borne infection settling into a focus of bone. This is common in children and intravenous drug abusers (Fig. 3). Another type of osteomyelitis is direct extension from a contiguous source of infection. An example of this would be open fractures that allow organisms to

communicate with the bone. Iatrogenic infections can occur as a result of surgical repair of fracture or by needle puncture into a bone or joint. A special form of chronic infection is chronic recurrent multifocal osteomyelitis. This peculiar disease may not be an infection at all. It is the childhood form of a non-infectious disease called SAPHO syndrome - a chronic sclerosing bone disease associated with skin lesions in adults [3]. The clavicle is most commonly affected.

#### **Advanced Imaging Features of Chronic Osteomyelitis**

MRI may be useful in determining the activity of chronic osteomyelitis [4]. Edematous changes of the bone marrow and surrounding soft tissues indicate ongoing infection. Furthermore, MRI may detect sequestra and sinus tracts. In the presence of periosteal new bone secondary to a soft-tissue ulcer or cellulitis, MRI is useful in evaluating the bone marrow to establish the absence of bone infection. In this situation, a bone scan may be misleading because of the uptake of the periosteal new bone. Advanced nuclear medicine imaging with SPECT may be a useful alternative [5].

Patients with a predisposition to infection and bone infarcts, such as sickle-cell patients and patients on steroids, frequently present with bone pain. MRI is a useful imaging modality to distinguish these two causes of pain. The pattern of marrow destruction is distinct from the appearance of an occult bone infarct. The central location of an infarct, its rectilinear delineation, absence of cellulitis in the surrounding soft tissue, and absence of sinus tract, distinguishes an infarct from osteomyelitis.



Fig. 3a-c. Images of the lumbar spine of a person who uses illicit drugs intravenously. **a** AP and **b** lateral radiographs show destruction of the L2 vertebral body and adjacent l2-3 end plate with kyphosis. c Sagittal T2 MR image shows the focal area of destruction at L2-3. The infection remains localized to this level and does not extend into the epidural space



Cellulitis and ulcers are common complications of diabetes. The radiologist is frequently asked to determine whether there is extension of infection to the adjacent bone or joint. MRI is the imaging modality of choice, giving more precise anatomic information than nuclear medicine scans (Fig. 4) [6]. A combination of T1 and STIR images detects bone-marrow edema and fluid in the joint. In the majority of clinical situations, gadolinium does not add to the standard imaging sequences. Attention must be paid to the position of the toes, aligning the image along the axis of the toe on the sagittal slices to facilitate interpretation of the bone-marrow signal.

Diabetic patients with cellulitis or foot ulcers and normal appearing bones on conventional radiography are classic candidates for MRI evaluation of the extent of infection. Even patients whose films show destructive changes of bone may benefit from an MRI evaluation if surgery is contemplated. The surgeon needs to define the proximal extent of the bone-marrow involvement in order to determine the site of amputation.

In the presence of neuropathic osteoarthropathy or fractures, the diagnosis of a superimposed infection by MRI is difficult [7]. Marrow edema is present within the bones of a neuropathic joint. In this situation, one must look carefully for evidence of destructive changes of the articular surfaces of the bones. If present, infection should be suspected. The neuropathic foot may also be investigated by a combination of Tc99-MDP and tagged white cells [8]. Comparison with plain films is useful in nearly all cases.

#### **Features of Septic Arthritis**

#### **Clinical Presentation and Methods of Spread**

The infected joint is a medical emergency [9, 10]. The earlier the diagnosis is made and the organism is detected, the greater the chance that the joint can be salvaged [11, 12]. Bacteria may enter a joint by several mechanisms [13]: hematogenous spread, spread from a contiguous infected site (soft tissue or osteomyelitis), direct implantation (penetrating injury, aspiration, arthrography) [14, 15, 16], and following arthroplasty.

Prime targets are the elderly, patients with chronic illness or immunosuppression [17], and those with preexisting joint disease. Both the host's ability to resist disease as well as the virulence of the organism are factors in the fate of the infected joint [18].

#### Pathophysiology of Septic Arthritis

An acute inflammatory response is initiated when bacteria enter the joint. The defense mechanism begins with the response by polymorphonuclear leucocytes, which release proteolytic enzymes, while lysozomes are released from the synovial membrane. Both of these enzymes contribute to the degradation of the proteoglycan matrix of the collagen and cartilage ground substance. Thus, the defense mechanism intended to protect the joint ultimately leads to its destruction.



medial side of the forefoot and the dislocation of the second metatarsal-phalangeal joint. b Coronal T2 MRI through the second metatarsal region shows two discrete soft-tissue masses in the plantar surface of the foot; these are abscesses. There is a skin ulcer extending to

one of the abscesses (arrow). c Sagittal fat-suppression T1-weighted MR image after the intravenous administration of gadolinium contrast agent shows an area of increased activity, corresponding to infection, in the soft tissues around the second metatarsal. One of the abscess cavities has low signal (arrow). Images courtesy of Dr. Jason Mehrling, Denver, Colorado, USA

#### **Organisms Related to Septic Arthritis**

*Neisseria gonorrhoeae* and *Staphylococcus* aureus are common sources of infection in healthy adults. Streptococcal arthritis usually is a result of hematogenous spread from skin or respiratory tract. Gram-negative bacteria (*Pseudomonas aeruginosa* and *Escherichia coli*) are associated with intravenous drug abuse or urinary tract infection. Fungal infection occurs in patients with decreased host defenses (HIV, chemotherapy). *Haemophilus influenzae* is seen in children from 6 months to 3 years of age.

#### **Clinical Findings**

The typical patient presents with acute onset of pain, swelling, warmth and erythema in a single joint. If the infection is due to hematogenous spread, there may be signs of systemic illness.

#### **Radiographic Findings**

The radiographic changes reflect the underlying pathophysiology: Synovial-membrane infection results in soft-tissue swelling and joint effusion. Hyperemia causes juxtaarticular demineralization. Proteolytic enzymes result in uniform destruction of the cartilage with uniform joint-space narrowing (Fig. 5). Pannus formation causes marginal erosions. The end result may be fibrous or bony ankylosis.



**Fig. 5.** This man had lacerations over the knuckles after hitting someone in a bar fight. The radiograph of this closed-fist injury was taken a week after the fight and shows destruction of the third metacarpal-phalangeal joint due to infection by mouth bacteria

#### Management

The goal is to prevent joint destruction. Therefore, early diagnosis and prompt administration of the proper antibiotic is the prime consideration by the clinician and radiologist. With typical clinical signs of infection and easy access to the joint fluid, the radiologist is generally not involved in the diagnostic workup of the patient with acute monoarticular arthritis. Advanced imaging techniques involving the radiologist are useful in the more difficult joints. Computed tomography or fluoroscopy is recommended for guidance of needle placement, with injection of contrast at the end of the procedure to confirm the intraarticular position of the needle. This is particularly useful in joints such as the hip, sacroiliac joint and shoulder.

If there is a question of monoarticular inflammatory arthritis, MRI may be useful to demonstrate the presence or absence of effusion in joints that are difficult to assess on physical examination (sternomanubrium, sacroiliac, facet joints). Establishing the presence of fluid in the proper clinical situation then mandates aspiration using CT guidance.

#### **Tuberculous Arthritis**

In the Los Angeles community, as well as the rest of the world, we are seeing a rise in tuberculosis (TB) in epidemic proportions. *Tuberculous arthritis and tuberculous spondylitis* must be considered in musculoskeletal infections [19]. The weight-bearing joints, i.e., the knee, hip, and sacroiliac joint, are affected more commonly than the joints of the upper extremity, but any joint, including the small joints of the hand, may be involved [20]. A diagnosis of TB should be considered in patients who are immunosuppressed or who are immigrants from third-world countries [21]. In this subset of patients, obtaining material for culture should include culture for acid-fast bacillus. Acid-fast stain alone may result in false-negative results.

Tuberculous exudate lacks the high concentration of proteolytic enzymes observed in pyogenic arthritis. Hence, there is often relative preservation of the cartilage associated with juxtaarticular demineralization and marginal erosions. These three findings are known as Phemister's triad. The absence of simultaneous jointspace narrowing, in the presence of destructive marginal erosions, should alert one to the possibility of a non-pyogenic process. Tuberculous arthritis and tuberculous spondylitis have a tendency to be associated with a cold abscess [22]. The abscess may predominate, giving a misleading clinical picture that this is a soft-tissue tumor. MRI may help in differentiating pyogenic and tuberculous skeletal infection [23].

#### Coccidioidomycosis

Coccidioidomycosis is endemic in Mexico and the southwestern United States. The fungus is disseminated in dust that is inhaled. The immunosuppressed popula-

tion as well as the darker-skinned races are most susceptible to this infection. A transient self-limited arthritis may accompany the respiratory infection. Only a small percentage of infected patients will develop musculoskeletal infection. A solitary osseous lesion or multiple geographic lytic lesions may be seen. The axial skeleton is the prime target, but the organism also has an affinity for the metaphyses of long bones and bony prominences. Unlike tuberculosis of the musculoskeletal system, the joints are less commonly infected than the bones. However, when present in joints, the typical presentation is that of monoarticular arthritis. The destructive changes are similar to those of other granulomatous infections. Diagnosis is made by culturing the aspirate or histologically viewing the organism on a biopsy specimen. MRI may be useful in selected cases to define the extent of infection [24].

#### Conclusions

The careful selection of imaging tests, taking into account the clinical presentation, physical appearance and the results of appropriate lab tests, can lead to prompt recognition of the infection and institution of therapy. In selected cases, advanced imaging techniques can be costeffective and aid treatment.

#### References

- Lazzarini L, Mader JT, Calhoun JH (2004) Osteomyelitis in long bones. J Bone Joint Surg 86-A:2305-2318
- Santiago Restrepo C, Gimenez CR, McCarthy K (2003) Imaging of osteomyelitis and musculoskeletal soft tissue infections: current concepts. Rheum Dis Clin North Am 29:89-109
- Earwaker JW, Cotten A (2003) SAPHO: syndrome or concept? Imaging findings. Skeletal Radiol 32:311-327
- Karchevsky M, Schweitzer ME, Morrison WB, Parellada JA (2004) MRI findings of septic arthritis and associated osteomyelitis in adults. Am J Roentgenol 182:119-122
- 5. Horger M, Eschmann SM, Pfannenberg C, Storek D, Dammann F, Vonthein R, Claussen CD, Bares R (2003) The

value of SPET/CT in chronic osteomyelitis. Eur J Nucl Med Mol Imaging 30:1665-1673

- Schweitzer ME, Morrison WB (2003) MR imaging of the diabetic foot. Radiol Clin North Am 42:61-71
- Aliabadi P, Nikpoor N, Alparslan L (2003) Imaging of neuropathic arthropathy. Semin Musculoskelet Radiol 7:217-226
- Love C, Din AS, Tomas MB, Kalapparambath TP, Palestro CJ (2003) Radionuclide bone imaging: an illustrative review. Radiographics 23:341-358
- Sack K (1997) Monoarthritis: differential diagnosis. Am J Med 102(1A):30S-34S
- Graif M, Schweitzer ME, Deely D et al (1999) The septic versus nonseptic inflamed joint: MRI characteristics. Skeletal Radiol 28:616-620
- 11. Goldenberg DL (1988) Septic Arthritis. Lancet 351:197-202
- Donatto KC (1998) Orthopedic management of septic arthritis. Rheum Dis Clin of North Am 24:275-86
- Pioro MH, Mandell BF (1997) Septic arthritis. Rheum Dis Clin North Am 123:239-259
- Von Essen R, Savolainen HA (1989) Bacterial infection following intra-articular injection. A brief review. Scan J Rheum 18:7-12
- Babcock HM, Matava MJ, Fraser V (2002) Postarthroscopy surgical site infections: review of the literature. Clin Infect Dis 34:65-71
- Resnik CS, Sawyer RW, Tisnado J (1987) Septic arthritis of the hip: a rare complication of angiography. Can Assoc Radiol J 4:299-301
- Tehranzadeh J, Ter-Organesyan RR, Steinbach LS (2004) Musculoskeletal disorders associated with HIV infection and AIDS: Part I: Infectious musculoskeletal conditions. Skeletal Radiol 33:249-260
- Dubost JJ, Fis I, Denis P et al (1993) Polyarticular septic arthritis. Medicine 72:296-310
- Yao DC, Sartoris DJ (1995) Musculoskeletal tuberculosis. Rad Clin North Am 33:679-689
- Ridley N, Shaikh MI, Remedios D, Mitchell R (1998) Radiology of skeletal tuberculosis. Orthopedics 21:1213-20
- Park Y-K, Park JS, Han C-S (2004) Tuberculosis manifesting as multifocal lytic cortical lesions in the femur. Skeletal Radiol 33:244-247
- Hugosson C, Nyman RS, Brismar J, Lrsson SG, Lindahl S, Lundstedt C (1996) Imaging of tuberculosis. V. Peripheral osteoarticular and soft-tissue tuberculosis. Acta Radiol 37:512-6
- De Vuyst D, Vanhoenacker F, Gielen J, Bernaerts A, De Schepper AM (2003) Imaging features of musculoskeletal tuberculosis. Eur Radiol 13:1809-1819
- Lund PJ, Chan KM, Unger EC, Galgiani TN, Pitt MJ (1996) Magnetic resonance imaging in coccidioidal arthritis. Skeletal Radiol 25:661-665

### **Peripheral Arthritis**

L.F. Rogers<sup>1</sup>, C.S. Resnik<sup>2</sup>

<sup>1</sup> Department of Radiology, University of Arizona Health Sciences Center, AZ, USA

<sup>2</sup> Depatment of Diagnostic Radiology, University of Maryland Medical System, MD, USA

#### Introduction

Radiographs are used in peripheral arthritis to confirm the clinical diagnosis of joint disease, determine the type of joint disease, and evaluate the extent of clinically known disease. The radiographic findings may be either consistent or inconsistent with the clinical diagnosis. If inconsistent, alternative diagnosis should be made on the basis of the radiographic appearance of the disease process.

Each joint disease has a relatively specific pattern of radiographic abnormalities. This pattern is based on the

radiographic characteristics at each individual joint, the distribution of joint involvement, and the presence or absence of other ancillary radiographic findings.

The distribution of joint involvement is extremely important. First, it should be determined whether the process is limited to one joint (monoarticular) or involves multiple joints (polyarticular). Each joint disease has a characteristic distribution of joint involvement (Fig. 1) and is more likely to involve certain joints than others, and to involve those joints either symmetrically (simultaneous involvement of similar joints of both extremities) or asymmetrically (involvement of a joint on one side



Fig. 1. Sites and distribution of common arthritides of the hand (A) and foot (B). The more common sites are encircled with *thick* lines and the less common sites with thin lines. Note the periosteal reaction or new-bone formaclassically tion identified in Reiter's disease. Note also the potential for "sausage digit" distribution in psoriasis. When joints are encircled in isolation, the distribution is random and may be isolated to any joint

without simultaneous involvement of the corresponding joint on the opposite side).

The specific radiographic characteristics of importance in establishing or confirming the diagnosis often are the following: (1) whether the joint space narrowing is symmetrical or asymmetrical; (2) whether soft-tissue swelling is present and whether it is symmetrical (indicating a joint effusion) or asymmetrical (indicating a periarticular mass); and the presence or absence of (3) periarticular osteoporosis, (4) periarticular erosions, and (5) spur formation.

Ancillary radiographic findings include the presence or absence of periosteal reaction of bones in the vicinity of the involved joint. Finally, the presence or absence of calcification within the joint cartilage (*chondrocalcinosis*) is to be noted.

The clinical findings of importance are the age and sex of the patient, a history of previous trauma, the clinical appearance of the joint or joints involved, the presence or absence of associated diseases (particularly skin disease, uveitis, and urethritis), and identifiable tophaceous deposits. Laboratory values of importance are the erythrocyte sedimentation rate; the presence or absence of serum rheumatoid factor; and the serum levels of uric acid.

#### **Rheumatold Arthritis**

Rheumatoid arthritis typically begins in the peripheral joints, usually the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the hand and the carpus. As the disease progresses, it affects more proximal joints, advancing toward the trunk in all extremities until finally almost every joint in the body is involved. The disease may become arrested at any stage.

Pathologically, rheumatoid arthritis begins as synovitis. In the early stages, there is edema and inflammation of the synovium and the subsynovial tissues. Joint effusion accompanies the synovial changes. As the disease advances, the synovium becomes greatly thickened, with enlargement of the synovial villi. This is followed by proliferation of fibrovascular connective tissue known as pannus. Pannus is responsible for the characteristic marginal erosions that first occur in the socalled bare areas between the peripheral edge of the joint cartilage and the insertion of the joint capsule. Ultimately, pannus grows over and destroys the surface of the articular cartilage.

#### **Roentgenographic Observations**

The initial manifestations are soft-tissue swelling, symmetrical narrowing of the joints, periarticular osteoporosis, and marginal erosions. Radiographic manifestations of the disease are present in 66% of patients 3 to 6 months after the onset of disease and in 85% of those affected for 1 year.

The distribution of joint involvement is characteristic. The disease begins in the PIP, MCP, and carpal joints with a more or less symmetrical distribution in the right and left extremities. In some cases, the joints of the hand and wrist are equally affected, but in others the destructive process may be much more severe in the hand than in the carpus. In still others, it may be more severe in the carpus than in the hand. In the foot, the metatarsophalangeal (MTP) joints, particularly the fourth and fifth, are often involved in the initial stage of the disease process. In fact, characteristic changes of erosion may be present in the heads of the fourth or fifth metatarsal when the radiographic changes of the hand are minimal or nondiagnostic. Therefore, it is important to examine not only the hands but also the feet in the initial evaluation of a patient with rheumatoid arthritis.

#### **Marginal Erosions**

Bony erosions occur as a result of the development of granulation tissue (pannus) at the peripheral margin of the joint cartilage. These appear as small foci of destruction along the margins of the articular ends of the bones. They may be very minute, but they represent one of the most significant roentgenographic observations of early disease. Magnification is helpful when searching for the smallest erosion. The most common sites are the radial sides of the heads of the first, second, and third metacarpals: the heads of the fourth and fifth metatarsals: and the ulnar styloid. Magnetic resonance imaging (MRI) is more sensitive than plain radiography for detection of early bone erosions. Characteristically, the distal interphalangeal (DIP) joints are spared. Erosions occur at the sites of tendinous attachments, such as the Achilles tendon on the calcaneus.

#### Joint Malalignment

Ulnar deviation of the phalanges with or without associated subluxation or dislocation is characteristic. The distal phalanx of the thumb is characteristically hyperextended, giving rise to the "hitchhiker thumb" deformity. The carpus is characteristically rotated towards the ulna.

#### Juvenile Rheumatoid Arthritis (Still's Disease)

In general, the younger the patient, the more likely the disease is monoarticular, particularly involving a large joint such as the knee, ankle, or wrist. The disease may be limited to a few major joints. If it begins in an older child, there is more likely to be symmetrical involvement of the smaller peripheral joints, as in an adult. There is interference with skeletal maturation, usually manifested as acceleration of maturation, with premature fusion of the ossification centers. The premature fusion leads to shortening of the digits. Involvement of the spine is much more common in children than in adults. The involvement may be manifested by atlantoaxial subluxation and erosions and eventually by bony ankylosis of the facet joints in the cervical spine.

## Rheumatoid Variants (Seronegative Spondyloarthropathies)

The term *rheumatoid variants* refers to inflammatory arthritides that differ immunologically, clinically, and radiographically from rheumatoid arthritis. The diseases are ankylosing spondylitis, psoriatic arthritis, Reiter's disease, and colitic arthritis. Afflicted persons usually have a negative rheumatoid factor, but a significant percentage have the HLA-B27 antigen. The diseases are more common in men and usually cause symptoms in the axial skeleton. This is in contrast to rheumatoid arthritis, which is more common in females and involves the distal appendicular skeleton. Radiographically, these diseases differ from rheumatoid arthritis in the absence or mild nature of periarticular osteoporosis or demineralization, the frequent occurrence of periostitis or periosteal new-bone formation, and the asymmetrical involvement of the peripheral skeleton.

#### **Psoriatic Arthritis**

Fewer than 10% of patients with psoriasis develop a peculiar form of arthritis, a smaller percentage develop classic rheumatoid arthritis, and an even smaller number develop some combination of the two. The extent of the arthritis does not correlate with the degree of skin disease. In some cases, the arthritis may even precede the skin manifestations by several years.

Psoriatic arthritis tends to involve the small joints of the hands and feet. The process is characteristically asymmetrical and is not associated with periarticular osteoporosis. The most characteristic involvement is in the DIP joints of the hands and toes, usually in association with psoriatic changes of the nails. At times, the asymmetrical involvement is confined to a single digit, sometimes referred to as a "sausage digit", with involvement of both IP joints and occasionally the MCP joint of one digit of one hand. Ankylosis of the IP joints is also common. Periostitis with periosteal reaction is frequent in the small bones of the hand and on the plantar surface of the calcaneus, as in other rheumatoid variants.

Sacroiliitis is common and resembles that seen in ankylosing spondylitis except that it is often asymmetrical; spondylitic changes are less common. The syndesmophytes in psoriatic spondylitis are typically broad, coarse, and asymmetrical. Vertebral squaring and apophyseal joint ankylos are also less common than in ankylosing spondylitis.

#### **Reiter's Syndrome**

Reiter's syndrome is characterized by urethritis, conjunctivitis, and mucocutaneous lesions in the oropharynx, tongue, glans penis, and skin, as well as arthritis. In general, the radiographic manifestations are similar to those of psoriatic arthritis, except that the axial skeleton is not as commonly involved and changes in the upper extremity are rare. The major joint involvement is the lower extremities, particularly the feet. The sacroiliac joint changes tend to be asymmetrical.

The most dramatic radiographic finding is usually periostitis, particularly the exuberant, fluffy, or whiskerlike periostitis at the site of tendon insertions, most frequently at the attachment of the plantar fascia, which forms a poorly defined spur on the plantar surface of the calcaneus. Periosteal reaction is also found in metatarsal shafts and on the surfaces of the tarsal bones in the distal tibia and fibula. The IP, MTP, and tarsal joints are most often affected.

#### **Colitic Arthritis**

Arthritis occurs in approximately 10% of patients with chronic inflammatory bowel disease, more commonly in patients with ulcerative colitis than in those with Crohn's disease. The most common manifestation is sacroiliitis, which is similar to but not as extensive as in ankylosing spondylitis and is usually symmetrical. Patients are rarely symptomatic, and the radiographic findings of sacroiliitis are often noted incidentally on abdominal radiographs obtained as part of a small bowel or colon examination. Peripheral arthritis, is uncommon.

#### Systemic Lupus Erythematosus

Arthralgia is a very common complaint in systemic lupus erythematosus (SLE), but radiographic findings occur in only one third of patients, and these are usually nonspecific changes of soft-tissue atrophy and osteoporosis. The most characteristic radiologic finding is an abnormality of joint alignment without articular erosions: Involvement of the IP joints results in either a "swan neck" or "boutonniere" deformity of the digit. Many patients are able to correct their deformities voluntarily.

#### **Degenerative Joint Disease (Osteoarthritis)**

Pathologically degenerative joint disease is characterized by degeneration and shredding of articular cartilage. It is not an inflammatory lesion, and therefore the term *arthritis* is a misnomer. Some prefer the use of the term *osteoarthrosis*, which removes reference to inflammation. It mainly affects the IP joints of the fingers, particularly the DIP joints, and the weight-bearing joints of the hips and knees. Degenerative joint disease occurs in two major forms, the primary form is a generalized disease, affecting all of the aforementioned joints, and the secondary form is limited to joints disrupted by previous intra-articular trauma or other joint disease. The roentgen signs and pathologic changes are similar in the two forms.

The principal radiographic features of osteoarthritis are asymmetrical joint-space narrowing, subchondral sclerosis of bone, marginal osteophytes, and subchondral cysts. Narrowing of the joint space in osteoarthritis is almost invariably uneven and more pronounced in that portion of the joint where weight-bearing stresses are greatest. In general, the greater the degree of narrowing, the more severe the associated findings of subchondral sclerosis and spur formation. Bony spurs or osteophytes may be found on the opposing surfaces of bone at the peripheral margins of the joint. Subchondral sclerosis or eburnation refers to the increase in density of the subchondral surface of bone. Subchondral cyst formation is much more pronounced in larger joints and is frequently more prominent on one side of the joint than on the other. The cysts, which have a sclerotic border, extend to the articular surface and may communicate with the joint. Calcified or ossified fragments of bone, termed loose bodies, may be identified within the joint but are particularly common in the knee. They represent calcified or ossified detached fragments of cartilage.

#### **Erosive Osteoarthritis**

Erosive osteoarthritis is an inflammatory form of osteoarthritis that occurs in postmenopausal women. It is usually limited to the IP joints of the hand. Clinically the joints are acutely inflamed; marginal erosions are prominent and are superimposed on the standard radiographic features of osteoarthritis. Erosions are often more pronounced at the PIP joints. The involved joints may eventually undergo bony ankylosis, which rarely occurs in the more common form of primary osteoarthritis.

#### **Diabetic Osteoarthropathy**

Diabetic osteoarthropathy is confined almost exclusively to the ankle and foot. Calcification of the smaller arteries of the foot is a frequent and important clue to the presence of underlying diabetes but may not always be evident. Fractures or fracture-dislocations of the tarsals or metatarsals are particularly common manifestations of diabetic neuropathic joints. Often such fractures or dislocations are incidental findings on radiographs obtained for the evaluation of infections of the foot or complaints of swelling without a history of trauma. Less commonly, the neuropathic process appears to be initiated by a traumatic event that results in a fracture or dislocation.

#### **Metabolic Joint Disease**

#### Gout

Gout is characterized by intermittent acute attacks of arthritis, an increase in the serum level of uric acid, and deposition of sodium urate in joints, bones, and periarticular tissues. Tophi, irregular, superficial, soft-tissue masses of varying size, eventually appear. Erosions are typically rather sharply defined and of variable size, asymmetrical, and are often defined by a sclerotic margin with an overhanging edge, which forms a characteristic hook or spur-like projection of cortical bone at the peripheral margin of the erosion.

Classically, the first MTP joint is the joint most often affected. The clinical expression of the disease in this location is known as *podagra*. Involvement of the metatarsotarsal and metacarpocarpal joints frequently occurs. Joint involvement is characteristically asymmetrical, both within any given joint and compared to the opposite side of the body.

Radiographic findings of gout do not occur until the disease has been present for as long as 6 to 8 years. Therefore, a negative roentgenogram does not rule out the possibility of gout. The principal radiographic features of the disease are periarticular marginal erosions, asymmetrical periarticular soft-tissue masses (tophi) with or without calcification, preservation of the joint space, and an absence of osteoporosis.

These soft-tissue masses appear at the margins of the joints of the foot and hand and in bursae, particularly the olecranon bursa, where they may be associated with underlying erosion of the olecranon. Characteristically, the bones maintain a normal density without evidence of osteoporosis and the joint space is often well maintained despite the presence of sizable erosions.

## Calcium Pyrophosphate Dihydrate Crystal Deposition Disease

*Chondrocalcinosis* is the presence of intra-articular calcium-containing salts within hyaline cartilage and fibrocartilage. Calcium within the fibrocartilage is characteristically somewhat irregular, as seen in the menisci of the knee or the triangular fibrocartilage of the wrist. The articular surface is composed of hyaline cartilage and, when calcified, appears as a fine, linear radiodensity closely paralleling the bony margins of the joint.

Deposition of calcium pyrophosphate dihydrate (CP-PD) crystals in the joint cartilage and periarticular tissues occurs in elderly persons and usually is manifested in the sixth and seventh decades by the radiographic demonstration of calcifications in the fibrocartilage and hyaline cartilage of the knees and wrists. Many affected persons are asymptomatic, but in others intermittent acute attacks of arthritic pain is associated with a joint effusion. The correct diagnosis is established by the identification of typical calcium pyrophosphate crystals in the synovial fluid.

The joints most commonly involved are the knee, the radiocarpal joints of the wrist, the MCP joints of the hand, the shoulder, and the hip. The joint changes that occur in this disorder, termed *pyrophosphate arthropathy*, resemble osteoarthritis, with joint-space narrowing, bone sclerorsis, osteophytes, and subchondral cyst formation. The unusual distribution of these findings and the presence of chondrocalcinosis allow a specific diagnosis to be made. Involvement of the MCP joints, particularly the second and third MCP joints, is characteristic of this disorder. Hemochromatosis also affects these joints in a similar fashion.

#### **Suggested Readings**

- Aliabadi P, Nikpoor N, Alparslan L (2003) Imaging of neuropathic arthropathy. Semin Musculoskelet Radiol 7(3):217-225
- Bennett DL (2004) Spondyloarthropathies: ankylosing spondylitis and psoriatic arthritis. Radiol Clin North Am 42(1):121-134
- 3. Bohndorf K, Imhof H, Pope TL (2001) Musculoskeletal Imaging: A concise multimodality approach. pp 292-377
- Buchmann RF (2004) Imaging of articular disorders in children. Radiol Clin North Am 42(1):151-168
- Greenspan A (2003) Erosive osteoarthritis. Semin Musculoskelet Radiol 7(2):155-159
- Gupta KB (2004) Radiographic evaluation of osteoarthritis. Radiol Clin North Am 42(1):11-41
- Klecker RJ, Weissman BN (2003) Imaging features of psoriatic arthritis and Reiter's syndrome. Semin Musculoskelet Radiol 7(2):115-126
- Monu JU (2004) Gout: a clinical and radiologic review. Radiol Clin North Am 42(1):169-184
- Steinbach LS (2004) Calcium pyrophosphate dihydrate and calcium hydroxyapatite crystal deposition diseases: imaging perspectives. Radiol Clin North Am 42(1):185-205
- Tehranzadeh J (2004) Advanced imaging of early rheumatoid arthritis. Radiol Clin North Am 42(1):89-107

### Special Aspects of Musculoskeletal Imaging in Children\*

D. Jaramillo<sup>1,a</sup>, G. Sebag<sup>2</sup>

<sup>1</sup> Division of Pediatric Radiology, Massachusetts General Hospital; Department of Radiology, Harvard Medical School, Boston, MA, USA
<sup>2</sup> Department of Pediatric Radiology, Hôpital Robert Debré; Faculté de Médecine Lariboisière-Saint-Louis, Université Paris, France

#### The Changing Skeleton of the Child

In children, the skeleton undergoes multiple changes with age. These age-related transformations determine the patterns of injury or disease and their imaging findings. The epiphysis and apophysis, entirely cartilaginous at birth, later become ossified. The unossified epiphysis can separate from the smooth metaphysis and, on radiographs, an apparent dislocation of the hip and shoulder can actually be a separation. The physis or growth plate, initially a flat disk between epiphysis and metaphysis, becomes progressively undulated after puberty and ultimately closes. The pattern of physeal injuries is thus more complex in older children and adolescents. The interfaces between bone and cartilage are particularly weak and prone to injury. The physis, for example, is weakest at the zone of provisional calcification, where the endochondral ossification occurs. The apophyses also tend to be avulsed at the base, where the apophyseal cartilage meets the parent bone. The thin, porous bony cortex of the newborn is transformed to dense lamellar bone beginning in the diaphysis; metaphyseal fractures usually occur at the point of transition between the two types of bone. The bones of children often bow rather than break, and the fractures frequently involve only one cortex. The injured radius and ulna often fracture incompletely, and the pelvis of a child is elastic and often breaks in a single place. The loosely attached periosteum of growing bones separates easily from the bone during a fracture; the intact periosteum is essential for the rapid healing and remodeling. The perichondrium, on the other hand, is tightly attached to the metaphyseal bone. In the metaphyseal fractures of battered children the perichondrium retains a rim of juxtaphyseal cortex which is seen as a bucket handle or a corner fracture on radiographs. The ligaments are perhaps the strongest element in the child's skeleton, and it is unusual to have ligamentous injuries before ten years of age. Fracture healing also changes with age. A femoral fracture heals in one week in the newborn, four weeks in the 5-year-old, eight weeks in the 10-year-old, three months in the adolescent, and more than four months in the adult.

## Normal Age-Related Variants and Related Diseases

#### Radiographs

Normal variants are often bilateral, but reassuring symmetry is not always present. It is useful to think of variants by site.

- Epiphyseal and apophyseal. Irregularity of the secondary center of ossification of the distal femur is found in approximately two-thirds of boys and 40% of girls (Fig. 1). It involves both condyles in 44% of cases. only the lateral condyle in 44%, and only the medial condyle in 12% [1]. Accessory centers of ossification are more conspicuous in the posterior femoral condyles. The tibial tubercle ossifies between 8 and 12 years in girls and 9 and 14 years in boys [2] and is normally irregular. Osgood-Schlatter disease is characterized by local pain and inflammation, and by imaging evidence of edema anterior to the tubercle and patellar tendon. The calcaneal apophyseal center ossifies in girls at 4-6 years of age, and in boys at 4-9 years of age, and is uneven, asymmetric, fragmented and sclerotic (Fig. 2). Normal calcaneal sclerosis decreases with disuse of the foot or after a month of not bearing weight [3]. Sever's disease (calcaneal apophysitis) can be diagnosed if there is soft tissue swelling on radiographs or cross-sectional images or increased scintigraphic activity [4].
- Physeal. A pseudofracture produced by one end of the physeal disc projecting over the other is easily recognized in the proximal humerus, but can be confused with a lateral condylar fracture in the distal humerus. A normal undulation of the medial distal tibia occurs at the site of normal closure; this is known as Poland's hump or Kump's bump and is located just above the medial talar hump (Fig. 2).

<sup>&</sup>lt;sup>a</sup> During the IDKD 2005, D. Jaramillo will be substituted by P.K. Kleinman.

<sup>\*</sup> This chapter originally appeared in: Von Schulthess GK, Zollikofer Ch L (2001) Musculoskeletal Diseases – Diagnostic Imaging and Interventional Techniques. Springer-Verlag Italia, Milan



**Fig. 2a, b.** Normal calcaneal ossification and distal tibial undulation in a 9-year-old boy. **a** On the lateral radiograph, there is sclerosis and irregularity of the apophysis, which is related to weight bearing (arrow). **b** On the frontal radiograph, the area of the medial distal tibial physis is undulated corresponding to Kump's bump (arrow)



Fig. 3. Four-year-old boy who had suffered a lawnmower injury involving the distal tibial physis 18 months ago. There is an irregularity in the metaphysis (*arrow*) corresponding to the healed injury which has been left behind by the physis

Metaphyseal. The juxtaphyseal metaphysis of weightbearing bones can be sclerotic between 2 and 6 years of age [5]. The metaphyseal band of lead intoxication, however, affects both weight-bearing and non-weightbearing bones (such as the fibula). A growth recovery line, or Harris line, follows a period of slower growth due to disease or trauma. Apparent metaphyseal sclerosis can result from resorption of the metaphyseal trabecula with preservation of the zone of provisional calcification. This can be seen in neonates under stress, and in children with leukemia or methotrexate osteopathy [6]. Tendinous insertions at metadiaphyseal junctions are prone to repeated minor avulsive injury. The cortex becomes irregular, particularly in the posterior distal femoral insertion of the medial head of the gastrocnemius muscle, where it can resemble a neoplasm on radiographs [7] or even on some coronal sections on magnetic resonance imaging (MRI) [8]. In difficult cases, a limited computed tomography (CT) exam can confirm the typical defect in the posterior cortex and the absence of soft tissue mass.

The metaphysis is also a reflection of the skeletal history of the child. As the child grows, lesions in the vicinity of the physis are left behind, falsely appearing to migrate towards the diaphysis (Fig. 3).

Round bones. The navicular is the last tarsal bone to ossify. There are normally two ossification centers, but multiple irregular, dense centers can develop, and fuse

by age 20 years. Aseptic necrosis of the navicular (Kohler's disease) affects older children, and is associated with pain [9].

#### MR Imaging

Age-related transformations of cartilage to bone, and of hematopoietic to fatty marrow, strongly influence the MRI appearance of the growing skeleton.

- Cartilage. Epiphyseal cartilage has intermediate signal intensity on T1-weighted images and low signal intensity on T2-weighted and STIR images. Epiphyseal cartilage is hypointense along weight-bearing regions and hyperintense in areas of active ossification [10]. Epiphyseal vascular canals enhance after the intravenous administration of gadolinium and help exclude epiphyseal ischemia [11]. The physis is of high signal intensity on most pulse sequences (Fig. 4). With physeal closure, the cartilage loses signal intensity and ultimately disappears [12].
- Marrow. Because of its high water content, normal hematopoietic marrow is of low signal intensity on T1-weighted images, intermediate signal intensity on conventional T2-weighted and STIR images, and high signal intensity on fast spin-echo T2-weighted and STIR images [13]. In each bone, conversion of hematopoietic to fatty marrow begins in the epiphyses and diaphysis, and then advances into metaphyses. In the extremities, conversion begins in the fingers and toes and ends in the proximal humeral and femoral metaphyses. Axial hematopoietic marrow persists throughout life.
- Menisci. Unlike meniscal tears, which are usually vertical in children [14], intrameniscal nutrient vessels are horizontal, central, originate from the capsular attachment, and do not extend to the articular surface [15].



Fig. 4. Two-year-old girl with an abscess in the soft tissues of the thigh. Sagittal post-gadolinium T1-weighted image shows enhancement of the physis (*double arrow*) and of the epiphyseal vascular canals (*single arrow*)

#### Scintigraphy

Tc-99m diphosphonate uptake is high in long bone physes and in physeal equivalents of the flat bones [16, 17]. This physiologic uptake decreases gradually with age but may persist even after the physes have fused radiographically. Skeletal structures that have not yet ossified have no Tc-99m diphosphonate uptake. For example, a "cold" femoral capital epiphyses during the first six months of life does not mean ischemia, and decreased activity in the tarsal navicular below the ages of 2 years in girls and 4 years in boys does not mean Kohler's disease [18, 19]. Uptake in the ischiopubic synchondroses between the ages of 4 and 12 years is commonly asymmetric [20, 21].

#### **Imaging Strategies**

In pediatric musculoskeletal imaging, the first imaging study is still plain radiography. The only exception is perhaps the use of ultrasonography during the first six month of life for evaluation of developmental dysplasia of the hip, where radiographs are of little value. Different imaging modalities have different strengths, and, for the most part, their information is complementary rather than competitive. It is important to know the relative strengths and indications of each modality in the evaluation of pediatric musculoskeletal problems.

#### **Computerized Tomography**

CT is a useful adjunct to plain radiography whenever multiplanar and three-dimensional (3D) reconstructions are considered (Fig. 5). In the context of trauma, CT is optimal for detection of subtle fractures (e.g. scaphoid injuries), for assessment of fragment separation (particularly in triplane and Tillaux fractures), and for evaluation of healing (such as in traumatic spondylolysis). In slipped capital femoral epiphysis, CT demonstrates the physeal irregularity, the degree of inferior and posterior displacement of the femoral head, and the retroversion of the contralateral femur. In acetabular fractures, 3D reconstructions demonstrate the relationships between fragments better. In infants with hip dislocation who have undergone reduction and placement of the hips in an abduction spica cast, CT can be used to assess the position of the femoral heads. If a low mAs technique is used, the total ovarian dose can be as low as 112 mrad (1.12 mGy) [22]. In adolescents and young adults with undetected hip dysplasia, CT with 3D reconstructions demonstrates the configuration and containment of the femoral head, acetabular architecture, and narrowing of the joint space. In the spine, vertebral abnormalities and fusions between vertebrae or ribs are easily demonstrated with CT.

Regarding *tarsal coalition*, the coronal plane is best for evaluation of talocalcaneal coalitions, whereas calcaneonavicular coalitions, which are usually evident on oblique radiographs, are best seen on sagittal reconstructions [23].



**Fig. 5a, b.** Frontal and posterior oblique 3D surface renderings of the spine of a 3-month-old girl with a severe defect of the bony thorax. The study was performed using a multi-detector CT, without need for sedation. **a** Frontal projection shows scoliosis and a defect of the ribs of the left side of the chest. **b** Oblique posterior reconstruction shows fusion of several ribs (*arrow*) which need to be distracted in order to correct the scoliosis

Both feet should be studied, as bilateral abnormalities can be seen in up to 81% of patients [24]. Multiple tarsal coalitions may occur in up to 20% of cases [25] and not 5%-10% as previously believed. Although accuracy for detecting tarsal coalitions is comparable for CT and MR imaging [26], CT allows easier evaluation of both feet, and it is less expensive, and more readily available. CT images demonstrate a complete osseous fusion if the coalition is bony, or irregularity of the articular surfaces of the anterior and middle facet if it is fibrous.

#### **MR Imaging**

MR imaging is the modality of choice for assessing spinal abnormalities. In infants, MR imaging evaluates abnormalities of vertebral segmentation, and the location of the conus medullaris (normally at L2 level, more caudal if the cord is tethered). In older children, MRI is optimal for evaluating protrusion or herniation of the discs, spinal stenosis, and nerve root compression. Infections and tumors involving the epidural and subarachnoid spaces are best demonstrated with gadolinium-enhanced imaging.

In cases of developmental hip dysplasia which do not respond to standard therapeutic measures, MR imaging depicts the position of the femoral head before and after reduction and detects obstacles to reduction (pulvinar, interposed psoas tendon, deformed labrum, or capsular infolding) [27-30]. Gadolinium-enhanced MR imaging may detect ischemia related to abduction during treatment for developmental dysplasia of the hip (DDH) [31, 32]. In the early presentation of Legg-Calvé-Perthes disease, and with femoral ischemia of other etiologies, MR imaging will demonstrate marrow edema [33] and lack of gadolinium enhancement of the femoral epiphysis (Fig. 6) [34]. MR imaging can also depict associated physeal and metaphyseal abnormalities [35] and the extent of marrow involvement [36, 37]. In more advanced disease, MR imaging shows the containment of the femoral head and the congruity of the articular surfaces [38].

MR imaging is crucial for evaluating spinal osteomyelitis, by depicting epidural abscess and extension of the infection into the paraspinal soft tissues. It is also very useful in pelvic osteomyelitis, where bony geometry is complex and soft tissue involvement is often the most important component of the infection; and in patients who do not respond after 48 hours of antibiotic therapy to exclude a subperiosteal or soft tissue abscess. MR imaging is useful in osteomyelitis involving the physis, where adequate mapping of the infection is important to minimize the risk of growth arrest [39, 40]. MR imaging of osteomyelitis should always include gadolinium enhancement to ascertain whether the infected volume contains drainable pus.

MR imaging helps in the evaluation of trauma to cartilaginous structures. It can detect: epiphyseal separation



**Fig. 6a, b.** Septic arthritis and femoral head ischemia in an 11-year-old boy who had osteomyelitis of the ischium. **a** Anterior sonograms of the hips show that in the right hip, the capsule (arrow) is elevated by an effusion of mixed echogenicity (\*). The normal left hip is shown for comparison. **b** Coronal gadolinium-enhanced imaging of the hips shows that there is decreased enhancement of the right femoral head (arrow) and enhancement of the hip synovium on that side

related to birth trauma or child abuse [41, 42]; extension of a lateral or medial condylar fracture into the unossified epiphysis of the elbow [43-47]; and focal areas of physeal widening and sometimes transphyseal bridging. Physeal widening occurs in the distal radius of young gymnasts [48] and in ambulatory meningomyelocele patients in whom impaired sensation and continued motion result in repeated physeal damage [49]. In patients with a suspected post-traumatic bony bridge, MR imaging can define the size and location of the bridge as well as the percentage of the physis affected by the abnormality [43, 50, 51]. A 3D fat-suppressed spoiled gradient-recalled echo sequence provides most, if not all, of the information required to assess growth arrest [52, 53]. Osteochondritis dissecans and meniscal and ligamentous injuries have a similar MR imaging appearance in children and adults [54].

The length of an intramedullary tumor is best depicted on T1-weighted images with a large field of view [55]. T1-weighted images also depict skip lesions and metastases or multifocal disease in the contralateral extremity [56]. In children it is particularly important to evaluate extension of tumor into the epiphysis, which occurs in roughly 80% of cases.

#### **Cross-sectional Measurements**

*Glenoid version* is the angle between the main axis of the scapula and the glenoid [57]. *Femoral anteversion* is determined by obtaining slices from the femoral head to the lesser trochanter, and slices through the distal femoral condyles. A line through the main axis of the femoral neck and another along the posterior surfaces of the distal

femoral condyles form the angle of femoral anteversion. The angle of anteversion is  $32^{\circ}$  at birth and  $16^{\circ}$  by age 16 [58]. *Tibial torsion* is determined by the angle between a line through the center of the epiphysis (representing the main axis of the proximal tibia), and a line connecting the distal tibial malleoli. External tibial torsion determined by physical examination is normally  $4^{\circ}$  at birth, and  $14^{\circ}$  at ten years of age [59, 60].

#### Sonography

Sonography is the main study in infants younger than 6 months with a question of hip dysplasia because it allows dynamic assessment of articular relationships and depicts the nonosseous structures [61]. It depicts: the hypoechoic cartilages of the proximal femoral epiphysis and acetabulum; the very echogenic bones of the proximal femoral metaphysis and acetabulum; and the echogenic fibrocartilaginous labrum. In Coventry, England, screening of more than 14 000 newborns detected a 6% incidence of sonographic abnormalities. Of these, nearly 80% were normal by 4 weeks and 90% by 8 weeks [62]. Uncritical acceptance of sonographic abnormalities in the first weeks of life can lead to overtreatment. In the United States, however, hip sonography is usually performed when the physical examination is abnormal or when there are risk factors [63]; these include a positive family history, breech delivery, oligohydramnios and conditions sometimes caused by uterine crowding, such as torticollis, clubfoot, or metatarsus adductus.

The coronal view, oriented like a frontal radiograph, shows acetabular morphology [64]. The angle between

the iliac wing and the bony acetabulum (the alpha angle) is approximately  $60^{\circ}$  in normal newborns [65]. The sonolucent cartilaginous acetabulum is more concave than the bony roof and it is in direct contact with the cartilaginous epiphysis. Ossification of the proximal femoral epiphysis is detected sonographically several weeks earlier than with radiographs [66]. The transverse view serves to examine hip motion and detect subluxation dynamically [67]. The femoro-acetabular relationships can be assessed during abduction and adduction and during the Barlow maneuver. The anterior axial view is used to detect adequacy of reduction. The two hips are easily compared. Under 3 months of age, the hip can appear slightly dysplastic due to immaturity, but any infant hip with an alpha angle under  $50^{\circ}$ , a beta angle over 70°, or subluxability on the dynamic examination is clearly abnormal. Before two weeks of age, because of hormonally induced ligamentous laxity up to 6 mm of posterior displacement of the femoral head with the Barlow maneuver is normal [67]. Sonography is of little value when hip dislocation is obvious clinically. The acetabular concavity cannot be adequately depicted when the femoral head is dislocated, and it is very difficult to align the femoral head with the midplane of the acetabulum. Infants with successfully treated dysplasia should be evaluated with a radiograph at 6 months of age, to verify that the acetabular abnormality has indeed resolved. Doppler sonography can show the vascularity of the femoral head of infants and newborns [68], but blood flow may be difficult to detect when the infant is moving significantly. Because only a few vessels are detected, it is difficult to differentiate normal perfusion from ischemia limited to a portion of the head.

Sonography is useful in the assessment of septic arthritis, as absence of a sonographically detected effusion speaks strongly against septic arthritis [69]. The evaluation of joint fluid is performed using an anterior approach, with the transducer placed in the groin along the femoral neck. An effusion is a relatively hypoechoic collection between the capsule and the femoral neck [70]. There is no relationship between the amount or echogenicity of the joint fluid and its likelihood of being infected (Fig. 6). On Doppler sonography, increased flow in the capsule is sensitive but not specific for infection [71]. Ultimately, if septic arthritis is suspected and fluid is detected, the hip should be tapped and the fluid analyzed.

#### Scintigraphy

In pediatrics, skeletal scintigraphy is the preferred study whenever the entire body is to be evaluated, such as for surveillance of metastatic disease, most notably from osteosarcoma, Ewing's sarcoma, and neuroblastoma. For the study of osteomyelitis, scintigraphy is better than MR imaging when acute osteomyelitis may be multifocal, as in newborns [72, 73], or poorly localized, as in infants presenting with a limp. Skeletal scintigraphy is also highly sensitive for evaluation of avascular necrosis, detection of skeletal metastases, and early identification of traumatic injuries, such as lower extremity injuries of toddlers and stress injuries of young athletes. In child abuse, skeletal scintigraphy complements the radiographic skeletal survey [74, 75] particularly when radiographic findings are negative or uncertain. Scintigraphy is very sensitive for rib fractures and diaphyseal fractures, but it often fails to detect linear skull fractures and certain metaphyseal injuries.

#### References

- 1. Caffey J, Madell SH, Royer C, Morales P (1958) Ossification of the distal femoral epiphysis. J Bone Joint Surg Am 40:647-654
- Ogden J (1984) Radiology of postnatal skeletal development: X. Patella and tibial tuberosity. Skeletal Radiol 11:246-247
- Shopfner C, Coin C (1966) Effect of weight-bearing on the appearance and development of the secondary calcaneal epiphysis. Radiology 86:201-206
- Lawson JP (1985) Symptomatic radiographic variants in extremities. Radiology 157:625-631
- Laor T, Jaramillo D (1993) Metaphyseal abnormalities in children: pathophysiology and radiologic appearance. AJR Am J Roentgenol 151:1029-1036
- Ecklund K, Laor T, Goorin AM, Connolly LP, Jaramillo D (1997) Methotrexate osteopathy in patients with osteosarcoma. Radiology 202(2):543-547
- 7. Keats T, Joyce J (1984) Metaphyseal cortical irregularities in children. Skeletal Radiol 12:112
- Yamazaki T, Maruoka S, Takahashi S, Saito H, Takese K, Nakamura M et al (1995) MR findings of avulsive cortical irregularities of the distal femur. Skeletal Radiol 24:43-46
- 9. Schmidt H, Freyschmidt J (1993) Köhler/Zimmer Borderlands of normal and early pathologic findings in skeletal radiography, 4th edn. Thiemel, Stuttgart
- Jaramillo D, Shapiro F (1998) Growth cartilage: normal appearance, variants and abnormalities. Magn Reson Imaging Clin N Am 6(3):455-471
- Barnewolt CE, Shapiro F, Jaramillo D (1997) Normal gadolinium-enhanced MR images of the developing appendicular skeleton: Part I. Cartilaginous epiphysis and physis. AJR Am J Roentgenol 169(1):183-189
- Chung T, Jaramillo D (1995) Normal maturing distal tibia and fibula: changes with age at MR imaging. Radiology 194:227-232
- Babyn PS, Ranson M, McCarville ME (1998) Normal bone marrow: signal characteristics and fatty conversion. Magn Reson Imaging Clin N Am 6(3):473-495
- Busch MT (1990) Meniscal injuries in children and adolescents. Clin Sports Med 9:661-680
- Al-Otaibi L, Siegel MJ (1998) The pediatric knee. Magn Reson Imaging Clin N Am 6(3):643-660
- 16. Connolly L, Treves S (1997) Pediatric skeletal scintigraphy with multimodality imaging correlations. Springer, Berlin Heidelberg New York
- 17. Hahn K, Fischer S, Gordon I (1993) Atlas of bone scintigraphy in the developing paediatric skeleton. Springer, Berlin Heidelberg New York
- Budinger TF (1998) MR safety: past, present, and future from a historical perspective. Magn Reson Imaging Clin N Am 6(4):701-714
- Connolly L, Treves S (1998) Pediatric skeletal scintigraphy. Springer, Berlin Heidelberg New York

- Cawley KA, Dvorak AD, Wilmot MD (1983) Normal anatomic variant scintigraphy of the ischiopubic synchondrosis. J Nucl Med 24:14-16
- Kloiber R, Pavlosky W, Portner O, Gartke K (1983) Bone scintigraphy of hip joint effusions in children. AJR Am J Roentgenol 140:995-999
- 22. Eggli KD, King SH, Boal DKB, Quoigue T (1994) Low-dose CT of developmental dysplasia of the hip after reduction: diagnostic accuracy and dosimetry. AJR Am J Roentgenol 163:1441-1443
- 23. Wechsler RJ, Schwertzer ME, Deely DM, Horn BD, Pizzutillo PD (1994) Tarsal coalition: depiction and characterization with CT and MR imaging. Radiology 193:449-452
- 24. Leonard MA (1974) The inheritance of tarsal coalition and its relationship to spastic flat foot. J Bone Joint Surg Br 56(3):520-526
- Clarke DM (1997) Multiple tarsal coalitions in the same foot. J Pediatr Orthop 17(6):777-780
- Emery KH, Bisset GS 3rd, Johnson ND, Nunan PJ (1998) Tarsal coalition: a blinded comparison of MRI and CT. Pediatr Radiol 28(8):612-616
- 27. Suzuki S, Kashiwagi N, Seto Y, Mukai S (1999) Location of the femoral head in developmental dysplasia of the hip: threedimensional evaluation by means of magnetic resonance image. J Pediatr Orthop 19(1):88-91
- McNally EG, Tasker A, Benson MK (1997) MRI after operative reduction for developmental dysplasia of the hip. J Bone Joint Surg Br 79(5):724-726
- 29. Kashiwagi N, Suzuki S, Kasahara Y, Seto Y (1996) Prediction of reduction in developmental dysplasia of the hip by magnetic resonance imaging. J Pediatr Orthop 16(2):254-258
- 30. Suzuki S (1995) Deformity of the pelvis in developmental dysplasia of the hip: three-dimensional evaluation by means of magnetic resonance image. J Pediatr Orthop 15(6):812-816
- Jaramillo D, Villegas-Medina OL, Doty DK, Dwek JR, Ransil BJ, Mulkern RV et al (1996) Gadolinium-enhanced MR imaging demonstrates abduction-caused hip ischemia and its reversal in piglets. AJR Am J Roentgenol 166:879-887
- 32. Jaramillo D, Villegas-Medina O, Laor T, Shapiro F, Millis MB (1998) Gadolinium-enhanced MR imaging of pediatric patients after reduction of dysplastic hips: assessment of femoral head position, factors impeding reduction, and femoral head ischemia. AJR Am J Roentgenol 170(6):1633-1637
- Bos CF, Bloem JL, Bloem RM (1991) Sequential magnetic resonance imaging in Perthes' disease. J Bone Joint Surg Br 73(2):219-224
- 34. Sebag G, Ducou Le Pointe H, Klein I, Maiza D, Mazda K, Bensahel H et al (1997) Dynamic gadolinium-enhanced subtraction MR imaging–a simple technique for the early diagnosis of Legg-Calve-Perthes disease: preliminary results. Pediatr Radiol 27(3):216-220
- 35. Jaramillo D, Kasser JR, Villegas-Medina OL, Gaary E, Zurakowski D (1995) Cartilaginous abnormalities and growth disturbances in Legg-Calvé-Perthes disease: evaluation with MR imaging. Radiology 197:767-773
- Ducou le Pointe H, Haddad S, Silberman B, Filipe G, Monroc M, Montagne JP (1994) Legg-Perthes-Calve disease: staging by MRI using gadolinium. Pediatr Radiol 24(2):88-91
- Sebag GH (1998) Disorders of the hip. Magn Reson Imaging Clin N Am 6(3):627-641
- Kaniklides C (1996) Diagnostic radiology in Legg-Calvé-Perthes disease. Uppsala University, Uppsala
- Gylys-Morin VM (1998) MR imaging of pediatric musculoskeletal inflammatory and infectious disorders. Magn Reson Imaging Clin N Am 6(3):537-560
- 40. Jaramillo D, Treves ST, Kasser JR, Harper M, Sundel R, Laor T (1995) Osteomyelitis and septic arthritis in children: appropriate use of imaging to guide therapy. AJR Am J Roentgenol 165:399-403

- Lazar RD, Waters PM, Jaramillo D (1998) The use of ultrasonography in the diagnosis of occult fracture of the radial neck. A case report. J Bone Joint Surg Am 80(9):1361-1364
- 42. Nimkin K, Kleinman PK, Teeger S, Spevak MR (1995) Distal humeral physeal injuries in child abuse: MR imaging and ultrasound findings. Pediatr Radiol 25:562-565
- 43. Jaramillo D, Hoffer F, Shapiro F, Rand F (1990) MR imaging of fractures of the growth plate. AJR Am J Roentgenol 155:1261-1265
- 44. Jaramillo D, Hoffer FA (1992) Cartilaginous epiphysis and growth plate: normal and abnormal MR imaging findings. AJR Am J Roentgenol 158:1105-1111
- Beltran J, Rosenberg ZS, Kawelblum M, Montes L, Bergman AG, Strongwater A (1994) Pediatric elbow fractures: MR evaluation. Skeletal Radiol 23:277-281
- Beltran J, Rosenberg ZS (1997) MR imaging of pediatric elbow fractures. MRI Clin North Am 5:567-578
- 47. Gordon AC, Friedman L, White PG (1997) Pictorial review: magnetic resonance imaging of the paediatric elbow. Clin Radiol 52(8):582-588
- Shih C, Chang C, Penn I, Tiu C, Chang T, Wu J (1995) Chronically stressed wrists in adolescent gymnasts: MR imaging appearance. Radiology 195:855-859
- Rodgers WB, Schwend RM, Jaramillo D, Kasser JR, Emans JB (1997) Chronic physeal fractures in myelodysplasia: magnetic resonance analysis, histologic description, treatment, and outcome. J Pediatr Orthop 17(5):615-621
- Rogers L, Poznanski A (1994) Imaging of epiphyseal injuries. Radiology 191:297-308
- Havranek P, Lizler J (1991) Magnetic resonance imaging in the evaluation of partial growth arrest after physeal injuries in children. J Bone Joint Surg Am 73:1234-1241
- Disler DG (1997) Fat-suppressed three-dimensional spoiled gradient-recalled MR imaging: assessment of articular and physeal hyaline cartilage. AJR Am J Roentgenol 169:1117-1124
- Borsa JJ, Peterson HA, Ehman RL (1996) MR imaging of physeal bars. Radiology 199:683-687
- Zobel MS, Borrello JA, Siegel MJ, Stewart NR (1994) Pediatric knee MR imaging: pattern of injuries in the immature skeleton. Radiology 190:397-401
- Fletcher BD (1991) Response of osteosarcoma and Ewing sarcoma to chemotherapy: imaging evaluation. AJR Am J Roentgenol 157(4):825-833
- Laor T, Chung T, Hoffer FA, Jaramillo D (1996) Musculoskeletal magnetic resonance imaging: how we do it. Pediatr Radiol 26(10):695-700
- Waters PM, Smith GR, Jaramillo D (1998) Glenohumeral deformity secondary to brachial plexus birth palsy. J Bone Joint Surg Am 80(5):668-677
- Ruby L, Mital MA, O'Connor J, Patel U (1979) Anteversion of the femoral neck. comparison of methods of measurements in patients. J Bone Joint Surg Am 61A:46-51
- Ritter MA, DeRosa GP, Babcock JL (1976) Tibial torsion? Clin Orthop 120:159-163
- Staheli LT, Engel GM (1972) Tibial torsion: a method of assessment and a survey of normal children. Clin Orthop 86:183
- Grissom L, Harcke H (1998) The pediatric hip. In: Rumack C, Wilson S, Charboneau J (eds) Diagnostic ultrasound. Mosby-Year Book, St. Louis, pp 1799-1814
- 62. Marks DS, Clegg J, al-Chalabi AN (1994) Routine ultrasound screening for neonatal hip instability. Can it abolish late-presenting congenital dislocation of the hip? J Bone Joint Surg Br 76(4):534-538
- Harcke HT (1994) Screening newborns for developmental dysplasia of the hip: role of sonography. AJR Am J Roentgenol 162:395-397
- 64. Graf R, Schuler P (1986) Sonography of the infant hip: an atlas. VCH Verlagsgesellschaftl, Weinheim
- 65. Graf R (1987) Guide to sonography of the infant hip. Thiemel, New York

- 66. Harcke HT, Lee MS, Sinning L, Clarke NMP, Borns PF, MacEwen GD (1986) Ossification center of the infant hip: sonographic and radiographic correlation. AJR Am J Roentgenol 147:317-321
- 67. Harcke HT, Grissom LE (1990) Performing dynamic sonography of the infant hip. AJR Am J Roentgenol 155:837-844
- 68. Bearcroft P, Berman L, Robinson A, Butler G (1996) Vascularity of the neonatal femoral head: in vivo demonstration with power Doppler US. Radiology 200:209-211
- Zawin JK, Hoffer FA, Rand FF, Teele RL (1993) Joint effusion in children with an irritable hip: US diagnosis and aspiration. Radiology 187(2):459-463
- 70. Miralles M, Gonzalez G, Pulpeiro JR, Millan JM, Gordillo I, Serrano C et al (1989) Sonography of the painful hip in chil-

dren: 500 consecutive cases. AJR Am J Roentgenol 152(3): 579-482

- Strouse PJ, DiPietro MA, Adler RS (1998) Pediatric hip effusions: evaluation with power Doppler sonography. Radiology 206(3):731-735
- Aigner RM, Fueger GF, Ritter G (1996) Results of three-phase bone scintigraphy and radiography in 20 cases of neonatal osteomyelitis. Nucl Med Commun 17(1):20-28
- 73. Bressler E, Conway J, Weiss S (1984) Neonatal osteomyelitis examined by bone scintigraphy. Radiology 152:685-688
- Nimkin K, Kleinman PK (1997) Imaging of child abuse. Pediatr Clin North Am 44(3):615-635
- 75. Conway JJ, Collins M, Tanz RR, Radkowski MA, Anandappa E, Hernandez R et al (1993) The role of bone scintigraphy in detecting child abuse. Semin Nucl Med 23(4):321-333

### Musculoskeletal Sonography

S. Bianchi<sup>1</sup>, S. Marcelis<sup>2</sup>

<sup>1</sup> Fondation et Clinique des Grangettes, Chêne-Bougeries, Switzerland

<sup>2</sup> Department of Radiology, Sint-Andriesziekenhuis Tielt, Tielt, Belgium

#### Introduction

The use of musculoskeletal ultrasound continues to expand, mainly because of technical improvements (highfrequency broadband transducers, refined focusing, and sensitive color and power Doppler technology) and growing interest on the part of musculoskeletal radiologists. Its low cost, non-invasiveness and portability, which, for example, allows examination athletes to be examined directly on the sport field, are additional qualities appreciated by patients. In addition, up to-date, high-level equipment allow detection of normal anatomic details and identification of a variety of pathologic conditions [1]. The possibility to perform a dynamic examination is a specific advantage of US over magnetic resonance imaging (MRI) and computed tomography (CT). The main disadvantages of US are limited assessment capability of internal structures of the joints, bone and bone marrow. However, introduction of the extended-field-of-view technology has allowed imaging of larger segments and has made their interpretation by the referring physician easier.

The objective of this article is to present the basic US aspect sof normal and pathologic muscles, tendons, and peripheral nerves followed by a review of the more commonly encountered abnormalities in different joints.

#### Muscles

#### **Ultrasound Anatomy of Muscles**

Ultrasound allows an accurate assessment of muscles. Muscle fibers are hypo-anechoic while fibro adipose septa (perimysium), which contain the nerves and vessels surrounded by fat, are hyperechoic. In longitudinal sonograms the perimysium appears as multiples hyperechoic lines while in transverse sonograms there are multiple spotty hyperechoic areas. The muscle fascia presents as a regular hyperechoic line of differing thickness surrounding the muscle. At musculotendinous junctions, US shows the concomitant decrease in the size of the muscle and increase in the size of the tendon. Muscle vessels are easily evaluated using the color Doppler technique, and a physiological increase in vascularization can be demonstrated during muscle exercise.

Dynamic examination is helpful in appreciating muscles changes during contraction. In isometric contraction, the muscles shorten and enlarge in their transverse diameters. In addition, they appear more hypo echoic due to the increase in the size of the muscle fibers and the relative decrease in the hyperechoic perimysium.

#### **Muscle Diseases**

Diseases of muscles can be divided into traumatic and atraumatic in origin.

#### **Traumatic Disorders**

Muscles trauma has increased in frequency due to the rapid expansion in amateur sport activities. US can efficiently locate tears, evaluate their size, differentiate between partial and complete lesions, successfully guide aspiration of hematomas, and allow follow-up and monitoring of healing. Dynamic scans performed during isometric contraction of the affected muscle can help in detecting smaller lesions. Firm pressure applied through the probe is invaluable in focusing the examination to the point of maximal tenderness, thus shortening the examination time and increasing the possibility to detect subtle injuries that otherwise can go unnoticed. Depending on the site, muscle traumas can be classified as affecting the fascia, muscle, or musculotendinous junction.

#### Lesions of the Fascia

The most common traumatic lesions of muscle fasciae are herniations (Fig. 1). Fascia rupture can involve its central portion or its attachment at the periosteal inser-



**Fig. 1.** Longitudinal sonogram of muscle herniation of the trapezium muscle. Image shows a focal interruption of the superficial muscle fascia (*arrowheads*) and local bulging (*asterisk*) corresponding to a muscle herniation



Fig. 2a, b. Tennis leg. a Longitudinal sonogram of the gastrocnemious medial head (GMH) in tennis leg. Note avulsion of the distal muscle septa (*black arrowheads*) from the disrupted distal aponuerosis (*empty arrowhead*) and the distal blood infarction (arrow). b More laterally, an anechoic fluid collection (*asterisk*), corresponding to a hematoma, is seen interposed between the GMH and the soleus. Arrowheads Muscle aponuerosis

tion. Before starting the US examination, it is important to locate the hernia by inspection in order to focus the examination and reduce the scanning time. Dynamic scanning obtained with the patient standing, supine or squatting efficiently facilitates hernia detection by showing an increase in muscle bulging through the fascia defect. Moreover, real-time examination during application of different amounts of pressure through the US transducer can demonstrate the possibility to reduce larger lesions [2].

#### Lesions of the Muscles

Traumatic muscles lesions can be due to direct local muscle trauma (external mechanism) or to maximal powerful contractions (internal mechanism). The formers are usually observed in contact sports, such as rugby, and involve mainly the quadriceps muscle, which becomes squeezed against the femoral shaft. The US aspect is that of an ill defined, irregular area located inside the muscle belly due to fiber tears, local infiltration of blood and the formation of a hematoma. Muscle ruptures due to maximal contractions are much more rare. Posttraumatic muscle calcifications (myositis ossificans) can follow both types of trauma and present at US as multiple foci of hyperechoic lesions with posterior shadowing. Although, in the proper clinical setting, the US appearance of myositis ossificans is quite typical, standard radiographs are always required to confirm the diagnosis.

#### Lesions of the Musculotendinous Junction

Clinical experience as well as experimental studies [3] have shown that, during maximal forceful activation of muscles, the first site to be injured is the musculotendinous junction. Muscles of the lower extremity are more frequently affected (Fig 2). The rectus femoris [4], medial head of the gastrocnemious [5] and biceps femoris are involved frequently since they cross two joints and have a high percentage of type II muscle fibers, which are well suited to rapid forceful activity. Moreover, the possibility of a strain is increased by the fact that they contract in an eccentric manner (i.e. they contract while passively stretched). At US, the retracted muscle fibers show a heterogeneous hypo-hyperechoic appearance due to the rupture of muscle fibers and blood infarction. Typically, the fibro-adipose septa, which in longitudinal images are seen inserting into the distal tendon or aponeurosis, are retracted proximally to a variable degree depending on the extent of the trauma. An anechoic fluid collection related to a hematoma is interposed between the retracted muscle and the tendon in larger lesions. Good results have recently been reported using US-guided evacuation of the hematoma followed by application of an elastic bandage. This approach allows more rapid cicatrization of the tear and an earlier return to sports activities. US can also detect local complications of tears such as venous thrombosis.

#### **Non-traumatic Disorders**

Non-traumatic disorders are quite uncommon in daily US practice. Muscle tumors are rare. The most common neoplasias are intramuscular lipomas, which appear as hyperechoic expansible lesions located inside the muscle (intramuscular lipoma) or in the fascial plane among muscles (intermuscular lipomas). Color Doppler shows absent or weak internal flow signals related to the low vascularity of the tumor. The size of intramuscular masses cannot be easily assessed by US when the diameters are larger that the size of the probe, although extended field of view technology can be of some help. Moreove,r it is sometimes difficult to exactly define the borders of a tumor. For these reasons, MRI examination with contrast enhancement is almost always required for assessment of muscles masses, particularly in the preoperative setting.

#### Tendons

#### **Ultrasound Anatomy of Tendons**

Tendons transmit the forces generated in muscles to bones. They are formed by parallel collagen bundles enfolded by the endotendineum and peritendineum. Longitudinal US demonstrates normal tendons as hyperechoic bands of variable thickness characterized by an internal arrangement composed of fine, packed, parallel echoes with a fibrillar pattern [6]. Such echoes are not related to the collagen bundles but to the interfaces between them and the endotendineum septa [6]. Transverse sonograms show tendons as circular ovoid structures with an internal dotted appearance. It is important to emphasize that the typical US pattern of tendons is evident only when the US beam is perpendicular to them. Any obliquity of the beam results in artifactual tendon hypoechogenicity, which can simulate a pathologic condition. An accurate technique of examination is therefore essential to avoid diagnostic mistakes.

From the anatomic and biomechanical point of view, tendons can be divided into two main groups. The first group includes tendons that present a straight course and are not prone to friction against other anatomic structures. These tendons are surrounded by paratenon, a loose areolar and adipose tissue envelope adherent to the tendon. Tendons of the second group reflect against bones surfaces or retinacula and are surrounded by a synovial sheath, which contains a thin amount of synovial fluid that facilitates frictionless movements and prevents tendon damage. At US, the paratenon appears as a hyperechoic tissue in continuity with subcutaneous fat. The synovial sheath can be appreciated only when the examination is performed with high-resolution equipment and presents as a thin hypoechoic rim surrounding the tendons, related to the synovial fluid contained in the sheath.

#### **Tendons Diseases**

#### Tendinosis and Ruptures

Tendinosis is a degenerative disease that result mainly from chronic local microtraumas. In tendinosis, US shows an enlarged tendon with internal irregularities of the normal internal structure, focal hypoechoic areas, and hyperechoic regions with posterior shadowing [7]. Histological examination showed that intratendinous hypoechoic areas correlate with fibromyxoid degeneration while hyperechoic images correlate with calcifications [8].

In partial tear, there is a localized disruption of some tendon fibers. Since this is almost always associated with changes related to tendinosis, the US distinction between tendon degeneration and small tears is often not feasible. Nevertheless, high-frequency US can easily differentiate partial from complete tears and help in clinical decision-making, particularly in acute cases in which local edema and pain limit a proper physical examination. In full-thickness rupture, a complete disruption of the tendon causes a retraction of the proximal torn edge due to the muscle action. In recent lesions, a hypoechoic hematoma fills the tendon gap whereas granulation tissue can be demonstrated in chronic cases. One of the main applications of US in evaluation of complete tears is detection of the retraction site of the proximal tendon end, which can help in choosing the extent of the surgical incision.

A distinctive type of tendon tear, so called longitudinal fissuration, can be observed in the ankle tendons, particularly the tibialis posterior and peroneal tendons, as a result of repetitive subbluxation of these tendon against the malleoli. US images show a hypoechoic cleft partially or completely dividing the tendon into two or more bands. An associated effusion located inside the tendon sheath is usually present and facilitates recognition of fissures [9].

#### **Inflammatory Conditions**

US can be used for diagnosing inflammatory conditions affecting tendons of both type 1 and 2. In tendons of the first group, changes are mainly observed at the level of the peritendon (peritendinitis). US demonstrates a hypoechoic thickening of the peritendon usually associated with surface irregularities of the outer portion of the tendon that appear hypoechoic and do not have a fibrillar appearance. Color Doppler demonstrates the local inflammatory hyperemia as flow signals into the tendon and in the surrounding tissues.

The hallmark of tenosynovitis in tendons of the second group is the presence of an effusion in the tendon's sheath. The most common causes of tenosynovitis are trauma, foreign bodies, infection and arthritis. In posttraumatic tenosynovitis, which can be suspected on the basis of the patient history, the fluid collection is usually anechoic. Increased echogenicity of the effusion andof the foreign body within the synovial space suggests an infective tenosynovitis, but it must be stressed that a definite diagnosis cannot be made only on the basis of US findings but relies on fluid aspiration, which can be performed under US guidance, and culture. In tenosynovitis secondary to systemic arthritis, the synovial membrane of the tendon sheath appears hypertrophied and presents at US as hypoechoic villous projections floating inside the effusion. In the most severe cases, the synovial pannus can eventually completely fill the synovial space. Since the hypertrophied synovium can damage the tendons and lead to pathological ruptures, accurate US assessment of tendon borders and echo texture is imperative. In hypertrophic tenosynovitis, color Doppler can help in distinguishing the hypoechoic pannus from the effusion based on the presence or absence of flow signals.

#### **Tendon Dislocation**

Dislocation can occur only in tendons of the second group and results from retinacular tears. The most frequent dislocations affect the long head of the biceps tendon at the shoulder [10] and the peroneal tendons [11] at the ankle. Due to its tomographic capability, US is well suited to detect tendon displacement. Transverse images optimally show the relation of the tendons with the osteofibrous tunnels that usually house them. Secondary changes, such as tendon sheath effusion due to inflammation, are also well demonstrated. Dynamic examination performed during different movements of the arm or foot may detect intermittent subluxation.

#### **Tendon Tumors**

Ganglia and the giant-cell tumor of the tendon sheath are the most common tendon masses. Ganglia are peritendinous cystic lesions containing mucoid, viscid fluid that usually are found in the hand and foot. They presents at US as multiloculated, well-defined, cystic anechoic masses. Rarely, they grow inside the tendon and appear as hypoechoic internal masses that follow the tendon during dynamic scanning. Giant-cell tumor of the tendon sheath presents as a painless, slowly growing mass located in close relationship with a tendon. The hand and foot are more commonly affected. US depicts giant-cell tumor as a hypoechoic mass with sharp borders located adjacent to the tendon. Internal flow signals can be detected using color Doppler. Fibrous and clear-cell sarcomas are rare.

#### Nerves

#### **Ultrasound Anatomy of Nerves**

Nerves are formed of nervous fibers grouped in fascicles. The nerve and the fascicles are surrounded by connective tissue, respectively the epyneurium and the perineurium. The US appearance of nerves, examined in vitro, reflects their anatomy [12]. Longitudinal sonograms show several hypoechoic parallel linear areas (nerve fascicles) separated by hyperechoic bands (connective tissue), forming a fascicular pattern. On transverse scans, the nerve fascicles is a hypoechoic rounded structures embedded in a hyperechoic background [12, 13].

Most peripheral nerves can be identified by US not only on the basis of their appearance but also because of their anatomic location. In doubtful cases, minor movements on dynamic examination performed during muscle activation can help in differentiating them from tendons.

#### **Nerves Diseases**

#### Traumatic Lesions

Nerves lesions can result from chronic repetitive microtraumas or a single acute trauma. Recurrent microtraumas are mainly observed in nerves entrapments syndromes, which typically affect nerves that course in unextensible osteofibrous tunnels. US is an effective imaging method to confirm clinical suspicion of entrapment neuropathy and to plan appropriate treatment, since it can depict nerve changes and the cause of the compression. The main nerve findings in chronic entrapments are: localized flattening at the level of compression and proximal bulbous enlargement, hypoechogenicity with loss of fascicular echo texture, enhanced flow signals on color Doppler.

Different causes of compression can be demonstrated by US. In carpal tunnel syndrome, tenosynovitis of the flexor tendons presents as an area of hypoechogenicity overlaying the tendons, ganglia as focal anechoic masses without internal flow signals, and accessory muscle as a peculiar muscle architecture and typical behavior during dynamic examination. In cubital tunnel syndrome, elbow osteophytes appear as hyperechoic lesion arising from the joint margins.

In nerves injures secondary to acute traumas, US can detect the level of the nerve section. This has practical value in planning operative treatment in patients with multiple traumas at different levels. The defect in the nerve appears as a local discontinuity in the nerve fascicles. Partial and complete tears can be differentiated in superficial nerve using a high-resolution probe. Bulbous neuromas, which present as localized hypoechoic enlargements of the nerve ends, are helpful in detecting the location of the tear.

#### Nerve Tumors

Most nerve tumors are benign schwannomas and neurofibromas. Schwannomas are encapsulated, well-circumscribed lesions that can be easily treated surgically, while neurofibromas spread within the fascicles and are difficult to remove (Fig. 3). The US diagnosis of a nerve tumor is based on detection of a mass along the course of a nerve in association with clinical signs. Typically, both tumors present as hypoechoic lesions. A definite differ-



**Fig. 3a, b.** Schwannoma. **a** Longitudinal sonogram of the anterolateral aspect of the leg. Note a solid mass (*asterisk*) connected with the deep peroneal nerve (*arrowheads*) corresponding to a schwannoma. The size, borders, internal structure and relation to the adjacent nerve can be well depicted by US. **b** Corresponding T1-weighted post-Gd image

entiation between schwannomas and neurofibromas is difficult to obtain on the basis of US findings. The value of US in this field is to differentiate compression due to extrinsic masses from a nerve tumor. Once a tumor is detected, US allows its careful location and accurate measurement of its longitudinal diameter, which is a crucial factor affecting the choice between end-to-end nerve suture and graft interposition.

#### Shoulder Sonography

The sensitivity of US in the detection of full-thickness tears (FTT) of the rotator cuff (RC) ranges from 94 to100%, for the detection of partial-thickness tears (PTT) from 93 to 96%, with a specificity of 94% for both [14, 15].

US signs of FTT [16, 17] are: (1) complete absence of the RC, and (2) an anechoic cleft in the cuff (Fig. 3). These injuries may result in: a naked tuberosity, herniation of deltoid muscle in the tendon gap, and a communication of the subdeltoid bursa with the joint space. Associated US signs of FTT [18, 17] are: (1) joint effusion, (2) effusion in the subdeltoid bursa, (3) surface irregularities of the greater tuberosity, and (4) focal cartilage interface sign.

An irregularity in the cortical of the greater tuberosity and joint fluid are important signs of FTT of the supraspinatus tendon [17].

PTT appear as anechoic to hypoechoic clefts with irregular hyperechoic borders, or as flattening of the bursal surface of the RC.

Degenerative changes in tendinosis are, in general, hypoechoic [17, 19], or hyperechoic [19].

In calcified tendonitis, US localizes and quantifies the calcifications, which appear as hyperechoic foci that may produce shadowing. Associated hypoechoic tendon thickening and positive Doppler examination reflect inflammation.

In impingement syndrome, US can demonstrate thickening of the subacromial-subdeltoid bursa, which accumulates in front of the acromion during elevation or abduction. Less frequently, a comparative study will show a difference of >2 mm in RC thickness due to tendonitis.

A small effusion, surrounding the biceps tendon may accompany any of the above-mentioned findings. In stage 2 or 3 of impingement, a PTT or FTT is found.

A fracture of the greater tuberosity may lead to a secondary type of impingement. Dynamic examination can also demonstrate anterior and posterior shoulder impingement

Effusion in the biceps tendon sheath reflects pathology elsewhere in the joint in 90% of cases. In inflammation, the biceps tendon is tender, enlarged, heterogeneous, surrounded by an effusion and may present longitudinal splits. When the bicipital groove is empty, the tendon may be ruptured, with variable retraction, or it may be dislocated (almost invariably associated with a tear of the subscapularis tendon). US is more accurate than plain film in detecting fractures of the greater or lesser tuberosity, Hill-Sachs deformities, grade 1 luxations of the AC joint, and bone erosions[20].

In experienced hands, US depicts linear hypoechoic labral tears and associated fractures in anterior shoulder instability [21]. US also demonstrates paraglenoid cyst, which can mimic RCT by compressing the suprascapular nerve at the suprascapular or spinoglenoid notch [22].

#### **Elbow Sonography**

A standardized examination technique using high frequency linear transducers and a comparative approach will detect several types of lesions [23, 24], such as small effusions in the coronoid fossa and annular recess and posterolateral joint space when the elbow is extended, or, in the olecranon fossa when the elbow is flexed. Depending on the etiology, the echogenicity of the effusion varies. US also detects intraarticular loose bodies, fractures (radial head) and osteocartilaginous lesions [25]. Power Doppler can be used for the detection and follow-up of inflammatory pathology (e.g., synovitis in rheumatoid arthritis). Tears of the ulnar collateral ligament appear as a focal discontinuity or a non-visualization, partial tears as (focal) thickening, decreased echogenicity and surrounding edema [26].

In epicondylitis (lateral or medial), a hypoechoic tendon thickening (Fig. 4) can be complicated by fissures,



**Fig. 4.** Full-thickness tear of the supraspinatus tendon, transverse plane A hypoechoic cleft filled with fluid is seen in the supraspinatus tendon. A focal cartilage interface sign is present (*arrow*)



**Fig. 5.** Tennis elbow, coronal plane. US demonstrates a hypoechoic area of tendinosis (*arrows*) at the common extensor insertion, in which a partial tear (*asterisk*) is noted

partial tears, calcifications, or synovitis [27, 28]. Complete tendon disruption represents a FTT. Cortical irregularity or spur formation can be detected at the epicondyle [28]. Intratendinous neo-angiogenesis or peritendinous hyperemia can be demonstrated using power Doppler evaluation.

Typical signs of a distal biceps tendon rupture are: a retracted distal biceps tendon causing acoustic shadowing, and a triangular-shaped blood-filled cavity at the musculotendinous junction [29]. Ultrasound can also demonstrate non-retracted tears and PTT with abnormal undulation of tendon fibers [23]. A thickened heterogeneous tendon is present in tendinosis; a fluid-filled bicipito-radial bursa can also be demonstrated.

The ulnar nerve measures 2-3 mm and should be evaluated comparatively and dynamically during flexing of the elbow [30]. In cubital tunnel syndrome, the ulnar nerve is thickened, hypoechoic and can be subluxed.

US can reveal underlying masses, large joint effusions, synovial proliferations, bony protuberances, and an anconeus epitrochlearis muscle.

Olecranon bursitis can be caused by friction (irregular wall thickening, hypoechoic fluid and echogenic fibrous clots), gout (hyperechoic nodular crystal depositions $\pm$  acoustic shadowing), or infection (intermediate echogenic fluid, surrounding edema, positive power Doppler, foreign body).

#### Hand and Wrist

Small-size US probes, utilizing frequencies ranging from 10 to 17 MHz, allow accurate assessment the superficial tissues of the hand and wrist [31]. The combination of standard radiographs with US works well in the evaluation of a large spectrum of disorders, although several conditions cannot be diagnosed by US and require MR or MR-arthrography for proper evaluation.

#### Traumas

Foreign bodies appear on US as hyperechoic structures associated with posterior shadowing (bone and vegetable splinters) or comet-tail artifact (glass or metallic fragment) (Fig. 6). The main advantage of US is the possibility to detect radiolucent fragments, which are undetectable on standard radiographs, and to assess their relationship with anatomic structures. US can diagnose rupture of the ulnar collateral ligament of the thumb and tears of the annular pulleys of the fingers. Partial tendon rupture appears as an area of localized swelling and decreased echogenicity inside the tendon. A complete tear is diagnosed when the tendon cannot be appreciated at the level of the injury and the swollen end is detected proximally. Microtraumatic tendon diseases, including De Quervain disease [33] and trigger finger [34], are due to repetitive movements that induce friction at the level of the osteofibrous tunnel (Fig. 7). US can easily detect tendon swelling, echo texture changes, and synovial sheath effusion, and eventually guide a local steroid iniection.



**Fig. 6.** Foreign body. Sonogram of the dorsal aspect of the wrist shows a hyperechoic foreign body (*white arrowhead*) surrounded by a hypoechoic inflammatory halo (*empty arrowheads*). Surgical exploration revealed a wood splinter



**Fig. 7a, b.** De Quervain tenosynovitis. Longitudinal sonogram of the first extensor compartment of the wrist. Longitudinal (**a**) and transverse (**b**) images show the thickened, hypoechoic retinaculum (*arrowheads*) surrounding the extensor tendons (*asterisk*)

#### Arthritis and Tenosynovitis

US allows diagnosis and follow-up of inflammatory disorders affecting the hand and wrist [32]. At early stages, when osseous erosions are not detected by standard radiographs, it demonstrates paraarticular edema as well as joint- and tendon-sheath effusions. Hypertrophy of the synovial membrane (pannus) producing marginal erosions can also be detected (Fig. 8). Fibrous pannus can be differentiated from active vascular hypertrophy using color Doppler. US aids in guiding a diagnostic joint puncture and allows proper intraarticular injection of steroids.

#### **Entrapment Neuropathies**

Entrapment neuropathies of the wrist concern the median nerve at the carpal tunnel [35] and the ulnar nerve at the Guyon tunnel. In both locations, US can show hypoechoic swelling of the involved nerve and loss of its fascicular pattern. The cause of the compression (tenosynovitis, ganglia, amyloid deposits) can also be detected by US. In carpal tunnel syndrome, US allows: (1) confirmation of the diagnosis when invasive nerve conduction studies are not accepted by the patient, (2) aid in planning surgery by demonstration of anatomic variants, such as a bifid median nerve or the presence of median artery, and by detection of expansible masses that cannot be successfully treated by endoscopy.

#### **Soft-Tissue Tumors**

As discussed above, the most common masses of the hand and wrist are ganglia [36] and giant-cell tumor of the tendon sheath. Ganglia are depicted as well-demarcated, anechoic masses with regular borders without internal flow signals at color Doppler (Fig. 9). In older lesions, internal septa and fibrosis explain the hypoechoic appearance. US-guided aspiration and local steroid injection can be performed in selected cases. Giant-cell tumors of the tendon sheath appear on US as paraarticular or paratendinous, solid, hypoechoic well-marginated masses that can present internal signals on color Doppler. They may also cause pressure erosions on the cortical bone of the phalanges. Although the US findings are not specific, US is invaluable in accurate evaluation of tumor size, location and relationship to surrounding structures, as well as in the early diagnosis of local recurrences.

#### **Hip Sonography**

Ultrasound detects different types of joint effusions in the hip when an anterior approach is used. The effusion can be demonstrated between the hyperechoic linings of the iliofemoral ligament and the femoral neck (transient synovitis, septic arthritis, rheumatoid arthritis, osteoarthritis, osteonecrosis).



**Fig. 8a-c.** Rheumatoid arthritis. Longitudinal (**a**) and transverse (**b**) color Doppler images obtained over the dorsal aspect of the wrist. **c** Corresponding T1-weighted post-Gd image. US shows the pannus as a hypoechoic area containing multiple flow signals (*white arrowheads*). An erosion (*empty arrowheads*) can be noted on the dorsal aspect of the capitate. Note the excellent US-MRI correlation



glion (*asterisk*) as an anechoic mass with sharp borders located close to the A2 annular pulley (*arrowheads*). The flexor tendons appear normal (*arrows*). P1 Proximal phalanx

In loosening of a hip prosthesis, the capsule to bone distance which is normally less than 3.2 mm, increases, and extracapsular collections will appear in cases of infection [37].

US can detect different hypoechoic bursae around the hip: the trochanteric bursa, the ischiogluteal bursa, and the iliopsoas bursa, of which the latest may communicate with the joint [38]. The cause is most often mechanical, less frequent inflammatory, and rarely infectious or tumoral. The complete spectrum of changes associated with tendinosis may occur at the insertion of the gluteus medius and minimus tendons [39].

The diagnosis of hamstring or adductor insertion tendonitis requires a comparison of the thickness and echo texture of the involved structures (Fig. 10).

When calcific tendonitis (rectus femoris, gluteal muscles) (Fig. 11) is diagnosed by US, a confirmation by plain film or CT is mandatory.

US can demonstrate the extraarticular origin of a painful snapping hip by a dynamic evaluation of the iliopsoas, gluteal and tensor fascia lata tendons [40]. A snapping iliopsoas tendon can produce a typical streak artifact when tissue harmonic imaging is used [41].

Different types of hernias in the groin region can also be differentiated and diagnosed by US [42].

In sports injuries, US detects hematoma and discontinuity in tendon or muscle tears of the hamstrings, adductor, and rectus femoris muscles, or, apophyseal avulsions in patients 14-25 years of age.

In chronic or repetitive lesions, muscular fibrosis and calcifications are found.

The US evaluation of hip dysplasia allows evaluation of the cartilage components of the femoral head and acetabulum in multiple planes, both at rest and with movement [43, 44].



**Fig. 10.** Tendinosis hamstring's insertion. Right and left comparative study of the hamstring's insertion in a transverse plane at the ischial tuberosity. The right hamstring's insertion appears markedly thickened compared to the left



**Fig. 11.** Calcific tendonitis rectus femoris insertion, sagittal plane. Hyperechoic crystal deposition (*arrow*) proximal in the rectus femoris tendon surrounded by edema

#### **Knee Sonography**

Ultrasound can detect small effusions of various echogenicity (depending on the etiology), loose bodies, and synovial proliferations (Fig. 12), which, in contrast to fluid, will not be displaced when pressure is exerted [45].

In tendinosis of the proximal patellar tendon (jumper's knee), the spectrum of focal hypoechoic tendon enlargement (areas of fibromyxoid degeneration), fissures, partial tears, focal hypervascularization, and calcifications can be monitored by US, and can easily be differentiated from peritendonitis or bursitis [46, 47]. Microavulsions of cartilage in Osgood-Schlatter or Sinding Larson Johansson disease are seen as hyperehoic calcified foci accompanied by hypoechoic focal tendon thickening and, occasionally, mild bursal effusion.

US differentiates quadriceps tendon lesions from injuries to the distal quadriceps muscle bellies.

In iliotibial band friction syndrome, hypoechoic thickening and fluid collection in the soft tissues between the lateral femoral condyle and the ilotibial tract should be looked for in a comparative study completed by a dynamic evaluation [48].

Different types of bursitis, chronic, metabolic, infectious, and hemorrhagic, generally have a distinct clinical and sonographic presentation. Acute inflammation of synovial- (bursa, joint space) or peritendinous tissue can be detected and monitored by power Doppler. When a hemorrhagic prepatellar bursitis is detected, a rupture of the



**Fig. 12.** Effusion and synovitis in a Baker's cyst, sagittal plane. Anechoic fluid in a Baker's cyst with hyperechoic thickened synovial wall (chronic synovitis). The cyst lies superficial to the medial gastrocnemius muscle and has a rounded inferior border (no rupture)

quadriceps tendon should be excluded. A ruptured Baker's cyst mimics a deep thrombophlebitis, and is characterized by a pointed (not a rounded) inferior border, accompanied by subcutaneous edema and fluid surrounding the muscles of the calf [25]. Chronic traumatic bursitis presents as hyperechoic thickened walls and a variable echogenic content. Hyperechoic foci embedded in a hypoechoic inflammatory substance is a typical presentation of bursitis in chronic gout at the extensor site of the knees and elbows [29].

Ultrasound can show the muscular, vascular, neurogenic, or cystic nature of a popliteal mass [49].

The US examination of the meniscus may reveal meniscal expulsion, cyst formation, amputation, tear (Fig. 13), central degeneration, or meniscocapsular separation. US is useful in evaluating menisci in patients below 6 and above 50 years of age. Tears appear as hypoechoic clefts reaching the surface of the meniscus [49]. Cysts may have a variable echogenicity and can expand at a considerable distance from the joint space connected to the ruptured

meniscus by a long pedicle [50]. Crystal deposition appears as hyperechoic deposits in the menisci.

The broad (15 mm) trilaminar medial collateral ligament and the cordlike lateral collateral ligament will be interrupted and surrounded by a hematoma when torn, or will show a hypoechoic focal thickening at the site of rupture [51]. The ligament is surrounded by hypoechoic edema when elongated [52]. A torn posterior cruciale ligament appears hypoechoic and diffusely thickened; the anterior cruciale ligament is evaluated by a comparatively posterior approach to the intercondylar region in a transverse plane and appears markedly swollen when torn [53]. US can also demonstrate focal lesions of the retinaculae.

Nerve-sheath ganglia of the peroneal nerve may arise either in the nerve sheath or from the proximal tibiofibular joint and appear as spindle-shaped cysts [54].

#### Ankle and Foot Sonography

In tendinosis, a focal or diffuse tendon enlargement and a hypoechoic appearance is noted; calcifications are a sign of chronic disease [55]. Neovascularization is associated with pain, while tendon inhomogeneity is correlated with an unfavorable outcome [56].

In tenosynovitis, an abnormal amount of fluid is noted in the tendon sheath (but: less than 3 mm of fluid can be seen at the dependent portions of the peroneal tendons, anterior tibiotalar joint and retrocalcaneal bursa, and up to 4 mm around the posterior tibial tendon) [57, 58].

Tendon tears may appear as longitudinal splits (especially peroneal tendons), partial transverse tears, or complete ruptures. US signs of FTT of the Achilles tendon are: undetectable tendon at injury site, tendon retraction, refraction shadowing arising from the retracted tendon ends (Fig. 14), a gap filled with hematoma, and herniating surrounding tissue [59].



**Fig. 13.** Tear medial meniscus, coronal plane. A hypoechoic cleft reaches the surface of the meniscus



**Fig. 14.** Full-thickness tear of the Achilles tendon, sagittal plane. The retracted torn end of the Achilles tendon (*arrows*) produces refraction artifacts. A chronic hematoma is seen in the gap (*star*)

Mobilization confirms complete rupture and demonstrates the presence of opposing torn ends.

Dynamic evaluation can demonstrate tendon instability, especially for the peroneal tendons [60], and can demonstrate tendon impingement secondary to osteophytes, fracture fragments, orthopedic hardware, or a prominent peroneal tubercle [61].

Bursitis of inflammatory or mechanical origin at the lateral or medial malleolus, sole of the foot, superficial to the Achilles tendon, or in a retrocalcaneal position can be distinguished from other cyst-like formations, such as arthrosynovial or ganglion cyst (frequently septated at the ankle and foot [62]), or from tumors, such as lipoma, or accessory muscles.

The evaluation of the joint space may reveal effusion, loose bodies and different degrees of ligamentous injury (Fig. 15) [58].

Morton neuroma appear as hypoechoic nodules in the intermetatarsal web space (Fig. 16) [63], elongated along the major axis of metatarsals and in continuity with the digital nerve [64].

In plantar fasciitis, the fascia is thickened (>4 mm) and becomes hypoechoic [65].

Fibromatosis of the plantar fascia appears as hypoechoic fusiform avascular nodules without acoustic enhancement [66].

The US signs of degenerative or inflammatory joint diseases of the foot and ankle are similar to those of the hands.



Fig. 15. Torn anterior talofibular ligament (*arrowhead*), joint effusion (*arrows*) extending anterior to the talus, transverse plane



Fig. 16. Morton neuroma, plantar approach, coronal plane. A hypoechoic nodule is seen in the intermetatarsal space

A partial torn ligament shows a focal hypoechoic thickening, while in acute complete rupture the torn ends are surrounded by hematoma.

US can detect occult bone lesions (erosions, stress fractures) and the presence of foreign bodies, which are linear, hyperechoic, and sometimes surrounded by a hypoechoic inflammatory reaction [67].

#### References

- Jacobson JA, van Holsbeeck MT (1998) Musculoskeletal ultrasonography. Orth Clin North Am 29:135
- Bianchi S, Abdelwahab IF, Mazzola CG et al (1995) Sonographic examination of muscle herniation. J Ultrasound Med May 14(5):357-60
- 3. Garrett WE Jr (1996) Muscle strain injuries. Am J Sports Med 24(6 Suppl):S2-8
- Bianchi S, Zwass A, Abdelwahab IF et al (1994) Diagnosis of tears of the quadriceps tendon of the knee: value of sonography. AJR 162:1137
- Bianchi S, Martinoli C, Abdelwahab IF et al (1998) Sonographic evaluation of tears of the gastrocnemius medial head ("tennis leg"). J Ultrasound Med 17:157
- Martinoli C, Derchi LE, Pastorino C et al (1993) Analysis of echotexture of tendons with US. Radiology 186:839
- Kalebo P, Allenmark C, Peterson L et al (1992) Diagnostic value of ultrasonography in partial ruptures of the Achilles tendon. Am J Sports Med 20:378
- Khan KM, Bonar F, Desmond PM et al (1996) Patellar tendinosis (jumpers knee): findings at histopathologic examination, US and MR imaging. Radiology 200:821
- 9. Diaz GC, van Holsbeeck M, Jacobson JA (1998) Longitudinal split of the peroneus longus and peroneus brevis tendons with disruption of the superior peroneal retinaculum. J Ultrasound Med Aug 17(8):525-529
- Prato N, Derchi LE, Martinoli C (1996) Sonographic diagnosis of biceps tendon dislocation. Clinical Radiology 51:737
- Magnano GM, Occhi M, Di Stadio M et al (1998) High-resolution US of non-traumatic recurrent dislocation of the peroneal tendons: a case report. Pediatr Radiol 28:476
- 12. Silvestri E, Martinoli C, Derchi LE et al (1995) Echotexture of peripheral nerves: correlation between US and histologic findings and criteria to differentiate tendons. Radiology 197:291
- Martinoli C, Bianchi S, Derchi LE (1999) Tendon and nerve sonography. Radiol Clin North Am Jul 37(4):691-711
- Wiener SN, Seitz WH (1993) Sonography of the shoulder in patients with tears of the rotator cuff: accuracy and value for selecting surgical options. AJR160:103-107
- Van Holsbeeck MT, Kolowich PA, Eyler WR et al (1995) US depiction of partial-thickness tear of the rotator cuff. Radiology 197:443-446
- Middleton WD, Teefey SA, Yamaguchi K (1998) Sonography of the shoulder. Seminars in musculoskeletal radiology 2:211-221
- 17. Jacobson J, Lancaster S, Prasad A et al (2004) Full-thickness and partial-thickness supraspinatus tendon tears: value of US signs in diagnosis. Radiology 230:234-242
- Hollister MS, Mack LA, Patten RM et al (1995) Association of sonographically detected subacromial/subdeltoid bursal effusion and intraarticular fluid with rotator cuff tear. AJR 165:605-608
- Bachmann GF, Melzer C, Heinrichs CM et al (1997) Diagnosis of rotator cuff lesions: comparison of US and MRI on 38 joint specimens. Eur Radiol 7:192-197
- Peetrons P, Chhem R (2000) Atlas d'Echographie du Systeme Locomoteur Tome 1, Le membre superieur. Sauramps Medical, Montpelier, France

- 21. Hammar M, Wintzell G, Äström K et al (2001) Role of US in the preoperative evaluation of patients with anterior shoulder instability. Radiology 219:29-34
- Martinoli C, Bianchi S, Gandolfo N et al (2000) Ultrasound of nerve entrapments in osteofibrous tunnels. Radiographics 20:199-217
- 23. Finlay K, Ferri M, Friedman L (2004) Ultrasound of the elbow. Skeletal Radiol 33:63-79
- 24. Martinoli C, Bianchi S, Giovagnorio F et al (2001) Ultrasound of the elbow. Skeletal Radiol 30: 605-614
- 25. Van Holsbeeck M, Introcaso JH (1991) Musculoskeletal ultrasound. Mosby Year Book, St. Louis, Missouri
- Miller T, Adler R, Friedman L (2004) Sonography of injury of the ulnar collateral ligament of the elbow. Skeletal Radiology 33:386-391
- 27. Vanderscheuren G, Prasad A, Van Holsbeeck M (1998) Ultrasound of the elbow. Seminars in musculoskeletal Radiology 2:223-235
- 28. Connell D, Burke F, Coombs P et al (2001) Sonographic examination of lateral epicondylitis. AJR 176:777-782
- Marcelis S, Daenen B, Ferrara MA, edited by RF Dondelinger (1996) Peripheral Musculoskeletal Ultrasound Atlas. Thieme, New York
- Jacobson JA, Jebson PJL, Jeffers AW et al (2001) Ulnar nerve dislocation and snapping triceps syndrome: diagnosis with dynamic sonography- report of three cases. Radiology 220:601-605
- Bianchi S, Martinoli C, Abdelwahab IF (1999) High-frequency ultrasound examination of the wrist and hand Skeletal Radiol Mar 28(3):121-129
- 32. Ribbens C, Andre B, Marcelis S et al (2003) Rheumatoid hand joint synovitis: gray-scale and power Doppler US quantifications following anti-tumor necrosis factor-alpha treatment: pilot study. Radiology Nov 229(2):562-569
- Giovagnorio F, Andreoli C, De Cicco ML (1997) Ultrasonographic evaluation of de Quervain's disease. J Ultrasound Med 16:685
- Serafini G, Derchi LE, Quadri P et al (1996) High resolution sonography of the flexor tendons in trigger fingers. J Ultrasound Med 15:213
- 35. Buchberger W, Judmaier W, Birbamer G et al (1992) Carpal tunnel syndrome: diagnosis with high-resolution sonography. AJR 159:793
- Bianchi S, Abdelwahab IF, Zwass A et al (1993) Sonographic findings in examination of digital ganglia: retrospective study. Clin Radiol 48:45
- Van Holsbeeck MT, Eyler WR, Suerman LS et al (1994) Detection of infection in loosened hip prosthesis: eficacy of sonography. AJR 163:381-384
- Bard H. Morvan G (2001) Les bursopathies de la racine du member inferieur. In: Rodineau J, Saillant G: Actualités sur les tendinopathies et les bursopathies du membre inférieur. Masson, Paris, 27-36
- Connell D, Bass C, Sykes C et al (2003) Sonographic evaluation of gluteus medius and minimus tendinopathy. Eur Radiol 13:1339-1347
- 40. Pelsser V, Cardinal E, Hobden R et al (2001) Extraarticular snapping hip: sonographic findings AJR 176:67-73
- 41. Cardinal E, Bureau N, Lafortune M et al (2002) The streak artifact using tissue harmonic imaging: a new sign of snapping tendons Abstr. Radiology 225:603
- Van den Berg JC, De Valois JC, Go PMNYH et al (2000) Radiological anatomy of the groin region Eur Radiol 10:661-670
- 43. Harcke HT (1995) The role of ultrasound in diagnosis and

management of developmental dysplasia of the hip. Pediatr Radiol 25:225-227

- Graf R (1984) Classification of hip joint dysplasia by means of sonography. Arch Orthop Trauma Surg 102:248-255
- 45. Richardson ML, Selby B, Montana MA et al (1988) Ultrasonography of the knee. Radiol Clin North AM 26:63-75
- Bouffard J.A, Dhanju J (1998) Ultrasonography of the knee. Seminars in musculoskeletal radiology 2:245-270
- Khan KM, Bonar F, Desmond PM et al (1996) Patellar tendinosis (jumper's knee): findings at histopathologic examination, US and MR imaging Radiology 200:821-827
- Bonaldi VM, Chem RK, Drolet R et al (1998) Iliotibial band friction syndrome: sonographic findings. J. Ultrasound Med 17(4):257-260
- 49. Van Holsbeeck M.T, Introcaso J (2001) Musculoskeletal Ultrasound, 2nd edn. Mosby, St. Louis, Missouri
- 50. Bianchi S, Martinoli C, Zamorani MP et al (2002) Ultrasound of the joints. Eur Radiol 12:56-61
- De Maeseneer M, Lenchik L, Starok M et al (1998) Normal and abnormal meniscocapsular structures: MR imaging and sonography in cadavers. AJR 171:969-976
- Lee JI, Son GIS, Yung YB et al (1996) Medial collateral ligament injuries of the knee: Ultrasonographic findings. J ultrasound Med 15:621-625
- 53. Ptasznik R, Feller J, Bartlet J et al. The value of sonography in the diagnosis of traumatic rupture of the anterior cruciate ligament of the knee. AJR 164:1461-1463
- Murphy MD, Smith WS, Smith SE et al (1999) Imaging of musculoskeletal neurogenic tumors: radiologic-pathologic correlations. Radiographics 19:1253-1280
- Fornage BD (1986) Achilles tendon: ultrasound examination. Radiology 159:769-774
- Zanetti M, Metzdorf A, Kundert H et al (2003) Achilles tendons: clinical relevance of neovascularisation diagnosed with power doppler. Radiology 227:556-560
- 57. Fessell DP, Vanderscheuren GM, Jacobson JA et al (1998) Ankle US: technique, anatomy and pathology. Radiographics 18:325-340
- Nazarian LN, Rawool NM, Martin CE et al (1995) Synovial fluid in the hindfoot and ankle: detection of amount and distribution with US. Radiology 197:275-278
- Hartgerink P, Fessell D, Jacobson J et al (2001) Full-thickness Achilles tendon tears: sonographic accuracy and characterization in 26 cases with surgical correlation. Radiology 220:406-412
- Ceulemans R, van Holsbeeck MT (1997) Ultrasonography, In: De Schepper AM (ed) Imaging of soft tissue tumors. Springer-Verlag, Heidelberg, pp 3-18
- Shetty M, Fessell D, Femino J et al (2002) Sonography of ankle tendon impingement with surgical correlation. AJR 179:949-953
- 62. Ortega R, Fessell D, Jacobson J et al (2002) Sonography of ankle ganglia with pathologic correlation in 10 pediatric and adult patients. AJR 178:1445-1449
- Pollak RA, Bellacosa RA, Dornbluth NC et al (1992) Sonographic analysis of Morton's neuroma. J Foot Surg 31:534-537
- Quinn TJ, Jacobson JA, Craig JG et al (2000) Sonography of Morton's neuromas. AJR 174:1723-1728
- 65. Gibbon W, Long G (1997) Plantar Fasciitis: Ultrasound Evaluation. Radiology 203:290
- Griffith J, Wong T, Wong S et al (2002) Sonography of plantar fibromatosis. AJR 179:1167-1172
- Jacobson JA, Powell A, Craig JG et al (1998) Wooden foreign bodies in soft tissue: Detection at US. Radiology 206:45-48

## PEDIATRIC SATELLITE COURSE "KANGAROO"





### The Spectrum of Non-accidental Injury and Its Imitators in Children\*

#### P.K. Kleinman

Department of Radiology, Children's Hospital, Boston, MA, USA

#### Introduction

Skeletal injuries are the most common findings noted on imaging studies in cases of child abuse. In infants, they result from shaking and other forms of manual assault (Fig. 1). In contrast to central nervous system and other visceral injuries, they are rarely life threatening. However, documentation of skeletal trauma is often central to the diagnosis of abuse. In infants, certain lesions are sufficiently characteristic to point strongly to the diagnosis of inflicted trauma (Table 1). Other fractures are less specific for abuse, but when correlated with other imaging findings and clinical information, their presence may add strong support for the diagnosis.

In the 50 years since Caffey's original description, radiologists have become familiar with the imaging features of commonly encountered inflicted skeletal injuries



Fig. 1. The shaken infant. (Illustrated by Laura Perry, M.D., based on description by assailants. Reproduced with permission from [23]) [1]. In recent years, increasing attention has been given to those conditions that may simulate inflicted injury. A variety of normal variants, naturally occurring diseases, and accidental skeletal injuries may be confused with the findings of child abuse. Sophisticated attorneys charged with the defense against allegations of abuse are often well versed in the differential diagnostic imaging specialists involved with cases of alleged abuse conduct their studies in a thorough and conscientious fashion that will provide the greatest likelihood of a correct diagnosis that can be sustained in a highly adversarial legal arena.

#### **Classic Metaphyseal Lesion**

The corner fracture and bucket handle lesions described in 1957 by Caffey are frequent findings in young abused infants [2]. Pathologically, the fracture

Table 1. Specificity of radiologic findings (From [3] with permission)

High specificity<sup>a</sup> Classic metaphyseal lesions Rib fractures, especially posterior Scapular fractures Spinous process fractures Sternal fractures Moderate specificity Multiple fractures, especially bilateral Fractures of different ages **Epiphyseal** separations Vertebral body fractures and subluxations Digital fractures Complex skull fractures Common but low specificity Subperiosteal new bone formation Clavicular fractures Long bone shaft fractures Linear skull fractures

<sup>a</sup> Highest specificity applies in infants

<sup>\*</sup> This chapter originally appeared in: von Schulthess GK, Zollikofer Ch L (2001) Musculoskeletal Diseases - Diagnostic Imaging and Interventional Techniques. Springer-Verlag Italia, Milan

extends in a planar fashion through the primary spongiosa. Centrally, the fracture abuts the chondro-osseous junction, and peripherally, the fracture veers from the physis to undercut a larger peripheral segment encompassing the subperiosteal bone collar. The fracture may extend partially or completely across the metaphysis (Fig. 2). The fractures are most common in the distal femur, proximal and distal tibia, and proximal humeri and are much less common at the elbow, wrist, and proximal femur (Figs. 3 and 4). The frac-



**Fig. 2a-d.** Corner fracture and bucket-handle patterns of the classic metaphyseal lesion (CML). Fractures (arrows) extend adjacent to the chondroosseous junction and then veer toward the diaphysis to undercut the large peripheral segment that encompasses the subperiosteal bone collar. **a**, **c** When the physis is viewed tangentially, the CML appears as a corner fracture pattern. **b**, **d** When a view is obtained with beam angulation, a bucket-handle pattern results. **a**, **b** Diffuse injury. **c**, **d** Localized injury. (Reproduced with permission from [3])



**Fig. 3a, b.** *Distal tibial CML.* **a** AP view of the distal tibia in a 5-month-old abused infant. A bucket-handle pattern is noted. **b** The lateral view shows a corner fracture pattern (*arrow*). (Reproduced with permission from [3])



**Fig. 4a, b.** *Proximal tibial CML, showing the value of serial examinations.* **a** AP view of the proximal right tibia in a 3-week-old abused infant. Radiograph is normal. **b** The AP view two weeks later demonstrates a bucket-handle pattern injury with subperiosteal new bone formation evident laterally (arrows). (Reproduced with permission from [3]

tures occur with torsion and traction of the extremities as the infant is grabbed by the arm or leg. The fractures may also occur with the sudden acceleration and deceleration of the extremities as the infant is shaken violently while grabbed by the thorax. When present in an otherwise normal infant, these findings point strongly to inflicted injury [3]. However, a variety of differential considerations for the classic metaphyseal lesion (CML) exist.

#### Rickets

Metaphyseal irregularity, cupping, physeal widening and bony demineralization are the hallmarks of rickets, however, on occasion discrete osseous fragments resembling corner fractures may be identified in the absence of more dramatic signs of rickets. The diagnosis may be particularly difficult if the metabolic disturbance is partially treated because demineralization may be modest and the density of the zone of provisional calcification may be relatively normal.

The small premature infant may demonstrate metaphyseal fractures indistinguishable from the CML, and infants with rickets undergoing vigorous passive range of motion exercises may develop these lesions [3].

#### **Birth Injury**

Caffey noted that metaphyseal injuries identical to those occurring with abuse can result from birth injury [1]. Vigorous extraction from a breech or armling presentation can result in forces similar to those occurring in abusive situations [4]. The tractional and torsional forces can produce metaphyseal lesions, particularly in the lower extremities [5]. The injuries can be overlooked at birth and may be identified within the first few weeks of life. These fractures are uncommon in the modern obstetrical era and can be readily excluded by a detailed birth history.

#### **Inherited Bone Dysplasias**

Although metaphyseal irregularity and fragmentation are seen in a variety of skeletal dysplasias, the presence of an underlying disease is usually apparent on clinical and/or radiologic grounds. Certain bone dysplasias, however, may manifest only modest osseous changes in early infancy, and the bony metaphyseal fragments in these cases may raise strong concerns of inflicted injury. Metaphyseal chondrodysplasia, Schmid type, may present in an infant of normal stature with metaphyseal fragments indistinguishable from abuse [6]. Similar findings have been described in spondylometaphyseal dysplasia, corner fracture type [6]. A thorough skeletal survey will generally point to the diagnosis. A followup skeletal survey in several weeks will show no change in the metaphyseal fragments in contrast to features of healing noted with the CML.

#### **Osteogenesis Imperfecta**

Most cases of osteogenesis imperfecta are accompanied by blue sclera, frank bony demineralization and other typical clinical and radiologic features (Type I). Rarely blue sclera may be absent (Type IV). Most long bone fractures involve the shafts or metadiaphyseal regions [6]. However, on rare occasions, small metaphyseal fragments with a corner fracture pattern may be encountered [3]. The presence of demineralization and other radiologic features of osteogenesis imperfecta confirm the diagnosis. Paterson and colleagues have described a group of children with metaphyseal lesions as well as other osseous injuries characteristic of abuse [7]. They coined the term "temporary brittle bone disease" to explain these injuries. There work has been widely criticized, and the lack of rigorous scientific methodology in their publications makes it impossible to draw any meaningful conclusions from their work [8, 9]. More recently Miller has proposed that "temporary brittle bone disease" is due to diminished intrauterine movement [10, 11]. His work has been strongly criticized on methodologic grounds [12].

#### **Developmental Variants**

The subperiosteal bone collar, an osseous ring that surrounds the primary spongiosa of the metaphysis and to a variable extent the physis, can produce a variety of imaging appearances which may simulate metaphyseal fractures [3, 13]. The subperiosteal collar can result in an abrupt step-off of the metaphyseal cortex as it approaches the physis. The subperiosteal bone collar may extend beyond the metaphysis forming a discrete linear mineralized spur at the periphery of the physis. These findings are most common at the knees and wrists.

#### **Rib Fractures**

Rib fractures are the most common fractures noted in infants dying with inflicted injury. Fractures can occur anywhere along the rib arc, but are most common near the costovertebral articulations. These fractures, as well as fractures near the costochondral junction are the most difficult to identify radiographically (Fig. 5). Fractures at the costovertebral junctions will become more visible on follow-up studies at two weeks; fractures at the costochondral junctions tend to heal with little subperiosteal new bone and tend to become less distinct with time. Most fractures occur with thoracic compression (Fig. 1). Evidence supports that excessive leverage of the ribs over the transverse processes with anteroposterior compression of the chest results in fractures of the rib head and neck (Fig. 6) [3].



Fig. 5a, b. Acute posterior rib fractures in a 6-week-old abused infant. Value of follow-up study. a An initial study demonstrates possible fractures of the right fourth, fifth, and sixth rib necks (*arrows*). b A follow-up study approximately 10 days later confirms the fracture on the right and reveals callus formation around the fractures of the left sixth and seventh rib necks. Note that callus appears predominantly below the rib margins (*arrows*). (Reproduced with permission from [3])



**Fig. 6.** Mechanism of injury. With anteroposterior compression of the chest, there is excessive leverage of the posterior ribs over the fulcrum of the transverse processes. This places tension along the inner aspects of the rib head and neck regions, resulting in fractures at these sites (*arrows*). This mechanism is also consistent with the morphologic patterns of injury occurring at other sites along the rib arcs and at the costochondral junction (*arrows*). (Reproduced with permission from [3])

#### **Birth Injury**

Rib fractures with birth injury are rare, but several reports suggest that obstetrical rib fractures may be more common than generally believed [14-18]. Typically, the fractures occur posterolaterally in large infants delivered by vacuum extraction and/or with shoulder dystocia. The absence of this history weighs strongly against birth injury. The absence of radiographic signs of healing by ten days of age on high quality radiographs helps to exclude obstetrical injury.

#### **Cardiopulmonary Resuscitation**

Although cardiopulmonary resuscitation (CPR) occasionally results in rib fractures in older children, documented cases of CPR induced fractures in infants are rare. Large studies including infants undergoing long periods of CPR administered by inexperienced individuals fail to reveal radiographic or autopsy evidence of rib fracture [3]. When fractures are noted with CPR, they are typically situated anterolaterally. The excessive levering forces occurring with inflicted rib fractures do not appear to occur with customary CPR [19].

#### **Accidental Rib Fractures**

Rib fractures are rarely noted in normal infants and young children sustaining mild to moderate traumatic injury. Accidental rib fractures near the costovertebral articulations have been described in infants involved in motor vehicle accidents when severe anteroposterior compression of the chest has occurred, presumably associated with levering forces similar to those occurring in abusive assaults [19].

#### **Skull Fractures**

Linear skull fractures are common accidental injuries in young infants. Clinical and laboratory studies indicate that a fall from several feet can result in a nondiastatic linear fracture in a young infant. In these cases, the diagnosis of abuse must rest on other imaging and clinical findings.

#### **Long Bone Fractures**

Although long bone fractures are commonly identified in abused children, and are the most common fracture beyond one year of age, they must be viewed in conjunction with other clinical and imaging findings. The early literature suggested that an oblique or spiral fracture pattern was a strong indicator of abuse, but it is now clear that the pattern of shaft fracture has little correlation with the presence or absence of abuse. Patient age appears to be the most important factor in predicting whether a shaft fracture is accidental or inflicted. Most femoral fractures in children under one year of age are inflicted. The percentage of fractures due to abuse declines dramatically in toddlers and older children. It is now well recognized that the running child may twist and fall, generating sufficient torsional forces to result in a spiral femoral shaft fracture. Femoral fractures may occur in infants who fall downstairs while being held by a caretaker.

As with the femur, most humeral fractures in infants are inflicted. This association diminishes beyond one year of age, particularly with regard to supracondylar fractures that are usually accidental. Rarely, a humeral shaft fracture can occur if an infant is held by one arm and forcefully turned from a prone to a supine position, trapping the other arm beneath the baby (Fig. 7) [20].

The toddler's fracture, an oblique or spiral fracture of the mid/distal tibial shaft is a common accidental injury in infants who have begun to bear weight [21]. As is generally the case with abuse, specific trauma is minimal or

Fig. 7. Humeral shaft fracture, possibly accidental. An AP view of the left humerus in a 5-month-old boy demonstrates an oblique fracture of the diaphysis (*arrows*). The father said he attempted to roll the child over in the crib and heard a "pop", followed by inability of the infant to move his left arm. There was evidence of previous neglect, but a skeletal survey and a Department of Social Service investigation considered that abuse was unlikely. (Reproduced with permission from [3])

absent. Although toddler type fractures do occur with abuse, an isolated nondisplaced oblique or spiral fracture of the distal tibia in a weight-bearing infant or toddler has a strong correlation with accidental injury.

#### **Imaging Approach**

The American College of Radiology has recently published standards for the performance of skeletal surveys for suspected abuse [22]. The protocol is outlined in Table 2. Images should be obtained with a high-detail film-screen system with a spatial resolution of at least 10 line pairs per millimeter and a speed of no greater than 200. When rib fractures are suspected, oblique views are advisable. When extremity fractures are identified, at least two projections should be obtained. When head trauma is suspected, a full skull series should be performed. A follow-up examination in two weeks may increase the diagnostic yield (Figs. 4 and 5). As digital radiography replaces film-screen imaging, care should be taken to insure that the diagnostic performance of the digital system is comparable to a high-detail film-screen combination.

#### Conclusions

The fundamental role of diagnostic imaging in cases of suspected abuse is much the same as with other medical conditions. The diagnostic process is characterized by gathering facts through appropriate imaging studies, integrating these findings with clinical and laboratory data, consulting with colleagues, and formulating a diagnosis based on one's knowledge and expertise. This process is predicated on a thorough understanding of the varied manifestations of child abuse and its imitators on modern diagnostic imaging studies. Imaging strategies for suspected abuse are therefore formulated to minimize the risk of a missed diagnosis. On the other hand, overzealous efforts by professionals who are ill prepared to differentiate child abuse from other conditions can have a profoundly negative impact on children and their families [3].

Table 2. The skeletal survey (From [3] with permission)

AP skullAP humeriLateral skullAP forearmsLateral cervical spineOblique handsAP thoraxAP femursLateral thoraxAP tibiasAP pelvisAP feetLateral lumbar spineAP feet

AP, anteroposterior



#### References

- Caffey J (1946) Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. AJR Am J Roentgenol 56:163-173
- Caffey J (1957) Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features. Br J Radiol 30:225-238
- 3. Kleinman PK (1998) Diagnostic imaging of child abuse, 2nd edn. Mosby, St. Louis
- Ekengren K, Bergdahl S, Ekstrom G (1978) Birth injuries to the epiphyseal cartilage. Acta Radiol Diagn 19:197-204
- 5. Snedecor ST, Wilson HB (1949) Some obstetrical injuries to the long bones. J Bone Joint Surg Am 31:378-384
- Taybi H, Lachman R (1996) Radiology of syndromes, metabolic disorders and skeletal dysplasias, 4th edn. Mosby-Year Book, St. Louis
- Paterson CR, Burns J, McAllion SJ (1993) Osteogenesis imperfecta: the distinction from child abuse and the recognition of a variant form. Am J Med Genet 45:187-192
- Chapman S, Hall CM (1997) Non-accidental injury or brittle bones. Pediatr Radiol 27:106-110
- Ablin DS, Sane SM (1997) Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. Pediatr Radiol 27:111-113
- Miller ME (1999) Temporary brittle bone disease: a true entity? Semin Perinatol 23(2):174-182
- Miller ME, Hangartner TN (1999) Temporary brittle bone disease: Association with decreased fatal movement and osteopenia. Calcif Tisue Int 64:137-143

#### **Suggested Readings**

- ACR (2001) Practice Guideline for Skeletal Surveys in Children, Res 31. In American College of Radiology: ACR Standards. Edited by Am Co Radiology. Reston, VA, pp 85
- Belfer RA, Klein BL, Orr L (2001) Use of the skeletal survey in the evaluation of child maltreatment. Am J Emerg Med 19:122-124
- Bulloch B, Schubert CJ, Brophy PD et al (2000) Cause and clinical caracteristics of rib fractures in infants. Pediatrics 105:E48
- Chalumeau M, Foix-L'Helias L, Scheinmann P et al (2002) Rib fractures after chest physiotherapy for bronchiolitis or penumonia in infants. Pediatr Radiol 32:644-647
- Gabos PG, Tuten HR, Leet A et al (1998) Fracture-dislocation of the lumbar spine in an abused child. Pediatrics 101:473-477
- Grayev A, Boal D, Wallach D et al (2001) Metaphyseal fractures mimicking abuse during treatment for clubfoot. Pediatr Radiol 31:559-563
- Gunther WM, Symes SA, Berryman HE (2000) Characteristics of child abuse by anteroposterior manual compression versus cardiopulmonary resuscitation: case reports. American Journal of Forensic Medicine & Pathology 21:5-10
- Hechter S, Huyer D, Manson D (2002) Sternal fractures as a manifestation of abusive injury in children. Pediatr Radiol 32:902-906
- Kleinman PK, O'Connor B, Nimkin K et al (2002) Detection of rib fractures in an abused infant using digital radiography: a laboratory study. Pediatr Radiol 32:896-901

- 12. Spivack BS (2000) Contributing Editor's note. Child Abuse Quarterly Medical Update 8:20
- Laval-Jeantet M, Balmain N, Juster M, Bernard J (1968) Les rapports de la virole perichondrale et du cartilage en croissance normale et pathologique. Ann Radiol 11:327-335.
- Gresham EL (1975) Birth trauma. Pediatr Clin North Am 22:317-328
- 15. Rizzolo PJ, Coleman PR (1989) Neonatal rib fracture: birth trauma or child abuse? J Fam Pract 29:561-563
- Barry PW, Hocking MD (1993) Infant rib fracture-birth trauma or non-accidental injury, Letter to the editor. Arch Dis Child 68:250
- Hartmann RW (1997) Radiological case of the month. Rib fractures produced by birth trauma. Arch Pediatr Adolesc Med 151:947-948
- Thomas PS (1997) Rib fractures in infancy. Ann Radiol 20:115-122
- Kleinman PK, Schlesinger AE (1997) Mechanical factors associated with posterior rib fractures: laboratory and case studies. Pediatr Radiol 27:87-91
- Hymel KP, Jenny C (1996) Abusive spiral fractures of the humerus: a videotaped exception. Arch Pediatr Adolesc Med 150:226-228
- Dunbar JW, Owen HF, Nogrady MG, McLease R (1964) Obscure tibial fractures of infants -- The toddler's fracture. J Can Assoc Radiol 15:136-144
- American College of Radiology (1997) ACR standards. Standards for skeletal surveys in children, Res. 22. ACR, Reston, p 23
- Kleinman PK (1990) Diagnostic imaging of infant abuse. AJR Am J Roentgenol 155:703-712
- Kleinman PL, Kleinman PK, Savageau JA (2004) Suspected Infart Abuse: Radiographic Skeletal Survey Practices in Pediatric Health Care Facilities. Radiology 233:477-485
- Krugman R, Bays J, Chadwick D et al (1993) American Academy of Pediatrics Commitee Child Abuse and Neglect: Shaken baby syndrome: inficted cerebral trauma. Pediatrics 92:872-875
- Kuhn J, Slovis T, Haller J (2003) Caffey's Pediatric Diagnostic Imaging, 10th edition. St. Louis, CV Mosby, pp 2672
- Kwon DS, Spevak MR, Fletcher K, Kleinman PK (2002) Physiologic subperiosteal new bone formation: prevalence, distribution, and thickness in neonates and infants. AJR. American Journal of Roentgenology 179(4):985-988, 2002
- Mandelstam SA, Cook D, Fitzgerald M et al (2003) Complementary use of radiological skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. Arch Dis Child 88:387-390; discussion 387-390
- McGraw EP, Pless JE, Pennington DJ et al (2002) Postmortem radiography after unexpected death in neonates, infants, and children: should imaging be routine? AJR Am J Roentgenol 178:1517-1521
- Ng CS, Hall CM (1998) Costochondral junction fractures and intra-abdominal trauma in non-accidental injury (child abuse). Pediatric Radiology 28:671-676
- Starling SP, Heller RM, Jenny C (2002) Pelvic fractures in infants as a sign of physical abuse. Child Abuse Negl 26:475-480



# **Contrast Enhancement of the Growing Skeleton: Rationale and Optimization in Pediatric MRI**

#### G. Sebag

Department of Pediatric Radiology, Faculté de Médecine Lariboisière-Saint-Louis, Université Paris VII - Hôpital R. Debré-Assistance Publique-Hôpitaux de Paris, Paris, France

The indications for contrast-enhanced magnetic resonance imaging (MRI) in the pediatric skeletal system are rapidly evolving and increasing [1-14]. MRI after gadolinium administration is unique in children in that it allows evaluation of the vascularity of growing osteocartilaginous structures and their maturational patterns during normal development. With high-speed, highstrength gradients and faster post-processing systems becoming more widely avalaible, dynamic gadoliniumenhanced subtracted (DGS) MRI can yield routinel information on vascularization, local blood volume, and perfusion, both qualitatively and quantitatively. This article discusses the technical considerations for optimizing MRI protocols and reviews the contrast-enhancement patterns of the different states of normal skeletal development. Recognizing the pattern of normal enhancement will serve as a reference in the analysis of disease processes, such as ischemia, necrosis, inflammation, edema, revascularization, and neovascularization [1-14].

#### **Technical Considerations**

#### **Intravenous delivery**

The usual dose of gadolinium for pediatric musculoskeletal applications is 0.1 mmol/kg body weight. Intravenous access is achieved prior to sedation or immediately before obtaining postcontrast sequences in non-sedated children. The use of Emla cream is very effective for anesthetizing the injection site. In a dynamic gadolinium-enhanced MR study is to be carried out, contrast-filled extension tubing allows scanning before, during and after bolus injection without interruption.

#### **Pulse sequences**

The imaging protocol should always include a precontrast T1-weighted sequence followed by a series of postcontrast T1-weighted images. If the suspected lesion is hyperintense and/or surrounded by hyperintense fat signal (fatty marrow, epiphyseal marrow, subcutaneous fat, fat pad) gadolinium-enhanced MRI actually may overlook lesion enhancement or decrease visualization of the lesion. In these cases, subtraction and fat suppression techniques, such as chemical shift, or selective pre-saturation are helpful. Short TI inversion recovery (STIR) is not recommended for discriminating between fat and paramagnetically relaxed water because both may be suppressed. Fast T1 weighted gradient echo and fast spin echo sequences with rapid sequential image acquisition (5-20 s) allow dynamic imaging of the first pass of gadolinium after bolus injection.

#### **Data Post-Processing**

Automatic measurement of enhancement rates and slopes provides additional information on regional blood perfusion and vascularity (Figs. 1, 2). The results are displayed on either parametric enhancement maps and/or time-intensity curves in a region of interest. The



Fig. 1. Legg-Calve-Perthes disease. Epiphyseal revascularization of the lateral and medial pillar


Fig. 2. Red versus yellow marrow. Relative enhancement curves



Fig. 3. Physis: Maximum relative enhancement versus age

following parameters can be measured and displayed: absolute and relative enhancement, maximum relative enhancement, time of arrival, time to peak, wash-in rate, wash-out rate, brevity of enhancement, area under the curve (Fig. 2).

# Gadolinium Enhancement Characteristics of the Developing Skeleton

Recognition of the maturational pattern for a given state of development is mandatory in order to rule out pathologic processes such as ischemia, necrosis, inflammation, edema and revascularization. Furthermore, it will assist in determining the optimal timing of data acquisition with respect to contrast administration. Anatomic and Doppler studies have shown that nutrition is provided to the cartilaginous epiphysis and physis by intracartilaginous vascular canals [1-7, 15]. Vascularity is abundant in quickly growing regions of the body; thus, the number and distribution of these canals change with maturation [5, 10, 11].

In the physis and the acrophysis (growth cartilage of the ossification center), enhancement is very intense. The epiphyseal vessels provide nutrition to the growth zone of the physis, accounting for enhancement through diffusion in this region [5, 10], and are also responsible for enhancement of the chondro-osseous junction of the acrophysis (Fig. 2). The metaphyseal vessels are responsible for enhancement of the chondro-osseous junction of the physis. The center of the physis is avascular beyond the first year of life. This pattern is a good indication of normal endochondral ossification and is well demonstrated on imaging.

We studied the normal maximum relative enhancement (MRE) and wash-in rate (WIR) in the proximal femur in a series of 37 children ages 39 to 178 months. In this series, the physis and acrophysis showed the highest peak enhancement and enhancement rate (Figs. 3-6).



Fig. 4. Physis: wash-in rate versus age



Fig. 5. Acrophysis: maximum relative enhancement versus age



Fig. 6. Acrophysis: wash-in rate versus age

The physeal and acrophyseal peak enhancements correlated significantly with the maximum enhancement rate. In addition, the physeal MRE (107%) and WIR (10/s) were significantly greater than the acrophyseal MRE (41%) and WIR (5/s) (Table 1).

The physeal and acrophyseal peak enhancement and enhancement rates decreased significantly with increasing age (Figs. 3-6.) Significant interindividual variation in the physeal degree of enhancement has been noted, possibly reflecting variation in enchondral growth and ossification (Figs. 1, 4). The vascular supply in hematopoietic red marrow is typically rich, sinusoidal and arborized, whereas fatty marrow has a much sparser vascular supply [3]. Greater enhancement is seen within red marrow than for fatty marrow (Figs. 1, 2) [3, 5, 16]. Normal marrow enhancement decreases with advancing age, paralleling the fatty marrow conversion and presumably its changing vascularity [6]. Significant interindividual variation in the degree of marrow enhancement has also been noted, possibly reflecting the variation in the fat content. These enhancement patterns aid in the interpretation of suspicious marrow lesions and in the depiction of abnormal marrow vascularity [15]. It is important to realize that gadolinium enhancement can decrease the contrast between normal red and fatty marrow on postcontrast T1-weighted images without subtraction or fat-suppression techniques [3].

 Table 1. Maximum relative enhancement and wash-in rate of the proximal femur

	Maximum enhancement relative (%)	Wash-in rate (per s)
Physis	107	10
Acrophysis	41	5
Femoral head marrow	4	3
Femoral neck marrow		3

#### **Clinical Applications**

#### **Evaluating Pediatric Bone Tumors**

Erlemann et al. [7] reported that dynamic MR studies with rapid sequential image acquisition after bolus administration can be used to differentiate benign from malignant lesions. In their study, 84.1% of malignant tumors exhibited slope values higher than 30% per min; 72% of benign tumors showed slopes lower than 30% per min. However, both benign and malignant tumors demonstrated some overlap using this differential criterion, resulting in an accuracy of approximately 80% with this technique [7].

In the series of Verstraete et al. (18, 19, 20), the firstpass slope value correlated well with tissue vascularization and perfusion but not with the histopathologic type of lesion. Highly vascularized or well-perfused benign lesions, (e.g. aneurysmal bone cyst, eosinophilic granuloma, giant cell tumor, osteoid osteoma, acute osteomyelitis, subcutaneous abscess, and myositis ossifcians) will present with slope values similar to those of malignant tumors. However, the high spatial resolution of the first-pass or slope images allows easy detection of viable tumor tissue and differentiation from necrosis and inflammation. This is important not only in planning the biopsy site but also in evaluation of the response to induction chemotherapy.

#### Evaluating ischemia and necrosis

In Legg-Calve-Perthes (LCP) disease, an important prognostic factor is the balance between the necrotic process and revascularization, especially in the lateral pillar [21]. MRI depicts the extent of necrosis more clearly than pinhole scintigraphy and changes are seen earlier than on plain films. The advantage of DGS MRI is to clearly show revascularization patterns thought to be directly related to the prognosis [13, 17]. This was first described by Tsao et al on serial bone scintigraphy [20]. Two principal mechanisms of revascularization may occur in LCP disease. The early appearance of a lateral pillar is indicative of uncomplicated revascularization of the femoral head. The lateral pillar plays a key role, both through its distinctive pattern of revascularization and its mechanical unique property. Sparing of the lateral pillar owing to early recanalization of the pre-existing epiphyseal vessels is associated with a good prognosis (Fig. 2).

The second mechanism is a slower rate of revascularization and healing. Involvement and collapse of the lateral pillar secondary to extensive necrosis and late transphyseal revascularization result in deformity and loss of containment, associated with a poor prognosis. Scintigraphic activity extends centrally from the base and lacks a lateral column pattern. New vessels coming from the metaphyseal side and disrupting the normal architecture of the growth cartilage can lead to early physeal closure. A third pathway, called the regression process, involves interrupted recanalization, because of the occurrence of a complication and therefore a change to a neovascular pathway. Subsequently, the percentage of lateral pillar involvement should be evaluated prospectively in the early evolutionary period in order to allow appropriate management. With DGS MRI, we were able to demonstrate reperfusion patterns similar to those obtained on bone scintigraphy. DGS MR depiction of reperfusion was very conspicuous due to early increased and persistent enhancement within the revascularized zones, compared to the normal hip enhancement. This increased gadolinium uptake may have been due to a greater vascularity, vasodilatation, increased capillary permeability and diffusion in the repair process (Fig. 1). All of the scintigraphic uptakes were demonstrated on MRI. Discrepancies corresponded to additional information obtained with dynamic MR, due to better spatial resolution and multiplanar slices. It may also be that MR depicts revascularization within metabolically inactive osseous areas. Medial pillar uptake was less frequently depicted on scintigraphy, probably because the medial column can be obscured by the adjacent uptake of the acetabulum [13]. The lateral pillar that was not detected was partially collapsed.

DGS MRI also allowed accurate visualization of transphyseal revascularization. This basal pattern of reperfusion was more often depicted in the anterior area, which is known to be the site of subchondral fracture and a more compromised vascular area. Transphyseal perfusion seems to be a predictor of growth arrest.

#### **Evaluating Articular Structures**

Accurate evaluation of the status of the articular cartilage, joint fluid, and synovium is crucial and requires appropriate contrast-enhancement protocols, especially in inflammatory diseases.

#### Synovium

Inflamed synovium is thickened and hypervascular and may be indistinguishable from articular cartilage and joint effusion. Gadolinium-enhanced imaging is thus needed to depict the extent and distribution of abnormal synovium, especially if quantification for serial assessment of disease severity and treatment response is required [8, 12].

The synovial intima lacks a tight junction or basement membrane and thus allows rapid diffusion of gadolinium compounds into the joint fluid. Therefore, static imaging must be undertaken immediately after gadolinium administration, or dynamic techniques must be initiated. Contrast-enhanced, fat-suppressed T1weighted 3D gradient echo techniques are most effective, allowing an optimal differentiation from adjacent fat pads [12].

Synovial inflammation can be quantified and monitored as time-activity curves of enhancement or volume of enhancing pannus by processing the imaging data. The clinical applications include staging, guiding, and monitoring local treatment of children with juvenile chronic arthritis and hemophilic arthropathy [14, 23].

#### Joint Fluid

An effective arthrographic effect can be achieved with intravenously administered gadolinium. In the knee, maximal intraarticular diffusion and fluid enhancement is obtained 30 min to 1 h after intravenous injection [24]. Exercise and mobilization significantly increase this effect and distribute contrast material uniformly throughout the joint.

This arthrographic effect improves the evaluation of cartilage defects, meniscal tears, intraarticular osteochondral body, and osteochondritis dissecans [24]. Its main limitation is the considerable range of enhancement from one individual to another and the limited distension of the articular capsule.

# Conclusions

Optimization of gadolinium-enhanced musculoskeletal MRI warrants special attention to the timing of data acquisition with respect to contrast administration as well as to selecting the appropriate sequences and postprocessing techniques for a given child, for a given anatomical region and for a given clinical problem. Recent advances in contrast-enhancement provide new information, both qualitative and quantitative, on the endochondral growth process and on the mechanisms of neovascularization and revascularization. All of these elements are important in dictating appropriate management.

# References

- Babyn PS, Kim HK, Gahunia HK, Lemaire C, Salter RB, Fornasier V, Pritzker KP (1998) MRI of the cartilaginous epiphysis of the femoral head in the piglet hip ischemic damage. J Magn Reson Imaging 8:717-723
- Babyn PS, Kim HK, Lemaire C, Gahunia HK, Cross A, DeNanassy J, Pritzker KP (1996) High-resolution magnetic resonance imaging of normal porcine cartilage epiphyseal maturation. J Magn Reson Imaging 6:172-179
- Babyn PS, Ranson M, Mc Carville ME (1998) Normal bone marrow: signal characteristics and fatty conversion. Magn Reson Imaging Clin N Am 6:473-495
- Barnewolt CE, Chung T (1998) Techniques, coils, pulse sequences and contrast enhancement in pediatric musculoskeletal MR imaging, Magn Reson Imaging Clin N Am 6:441-453
- Barnewolt CE, Shapiro F, Jaramillo D (1997) Normal Gadolinium-enhanced MR images of the developing appendicular skeleton: part I Cartilaginous epiphysis and physis. Am J Roentgenol 169:183-189
- Dwek JR, Shapiro F, Laor T, Barnewolt CR, Jaramillo D (1997) Normal Gadolinium-enhanced MR images of the developing appendicular skeleton: Part 2. Epiphyseal and metaphyseal marrow. Am J Roentgenol 169:191-196
- Erlemann R, Reiser MF, Peters PE et al (1989) Musculoskeletal neoplasms: static and dynamic Gd-DTPA-enhanced MR imaging. Radiology 171:767-773

- Herve-Somma CM, Sebag GH, Prieur AM, Bonnerot V, Lallemand DP (1992) Juvenile rheumatoid arthritis of the knee: MR evaluation with Gd-Dota. Radiology 182:93-98
- Jaramillo D, Shapiro F (1998) Musculoskeletal trauma in children. Magn Reson Imaging Clin N Am 6:521-536
- Jaramillo D, Shapiro F (1998) Growth cartilage: normal appearance, variants and abnormalities, Magn Reson Imaging Clin N Am 6:455-471
- Jaramillo D, Villegas-Medina O, Laor T, Shapiro F, Millis MB (1998) Gadolinium-enhanced MR imaging of pediatric patients after reduction dysplastic hips: assessment of femoral head position, factors impeding and femoral head ischemia, Am J Roentgenol. 170:1633-1637
- 12. Lamer S, Sebag GH (2000) MRI and ultrasound in children with juvenile chronic arthritis. Eur J Radiol 33:85-93
- Lamer S, Dorgeret S, Khairouni S, Brillet PY, Bacheville E, Bloch J, Penneçot GF, Hassan M, Sebag G (2002) Femoral head vascularisation in Legg-Calvé-Perthes disease: comparison of dynamic gadolinium-enhanced subtraction MRI with bone scintigraphy. Pediatr Radiol 32:580-585
- 14. Polisson RP, Schoenberg OI, Fischman A et al (1995) Use of magnetic resonance imaging and positron emission tomography in the assessment of synovial volume and glucose metabolism in patients with rheumatoid arthritis. Arthritis Rheum 38:819
- Sebag GH, Pinzuti V, Argyropoulou M, Elmaleh M (1997) Doppler ultrasonography in the study of the vascularization of the femoral head in the newborns. J Radiol 78:289-292
- 16. Sebag GH, Dubois J, Tabet M, Bonato A, Lallemand D (1993)

Pediatric spinal bone marrow: assessment of normal age-related change MRI appearance. Pediatr Radiol 23:515-518

- 17. Sebag GH, Ducou le Pointe H, Klein I, Maiza D, Mazda K, Bensahel H, Hassan M (1997) Dynamic Gadolinium-enhanced subtraction MR imaging a simple technique in the early diagnosis of Legg-Calve-Perthes disease: preliminary results. Pediatr Radiol 27:216-220
- Vanel D, Vertraete KL, Shapeero LG (1997). Primary tumors of the musculoskeletal system. Radiol Clin North Am 35:213-237
- Verstraete KL, Dierick A, De Deene Y et al (1994) Firstpass images of musculoskeletal lesions: a new and useful diagnostic application of dynamic contrast-enhanced MRI. Magn Reson Imaging 12:687-690
- Verstraete KL, Van der Woude HJ, Hogendoorn PR et al (1996) Dynamic contrast-enhanced MR imaging of musculoskeletal tumors: basic principles and clinical applications. J Magn Reson Imaging 6:311-321
- Sebag GH (1998) Disorders of the hip. Magn Imaging Clin N Am 6:627-631
- Tsao AK, Dias LS, Conway JJ et al (1997). The prognostic value and significance of bone scintigraphy in LCP. J Pediatr Orthop 17:230-239
- Sebag GH, Laor T, Quignodon JF (2004) Imaging in children. In: Maddison PJ, Isenberg DA, Woo P, Glass DN (eds) Oxford textbook of rheumatology, third edition. Oxford University Press, New York, pp 534-52
- Winalski CS, Aliabadi P, Wright JR et al (1993) Enhancement of joint fluid with intravenously administered gadopentetate demeglumine: technique, rationale and implications. Radiology 187:179



# Imaging the Osseous and Soft Tissue Tumors in the Child

A. Geoffray

Fondation Lenval, Nice, France

Pediatric osseous and soft tissue tumors require imaging at each step: at diagnosis to approach the nature of the tumor and evaluate local and general extension, during treatment to follow-up the response, and after the end of treatment to search for recurrence. Imaging techniques must be adequately chosen according to each different type of tumor.

# **Bone Tumors**

Bone tumors in children may be benign or malignant, primitive or secondary. The original cell may be bone, cartilage, fibrous, vascular, or unknown tissue (Table 1).

Table 1.	Origins	of osseous	tumors and	pseudo-tumors
----------	---------	------------	------------	---------------

Origin	Benign	Malignant
Bone	Osteoid osteoma, osteoma, osteoblastoma	Osteogenic osteosarcoma
Cartilage	Osteochondroma, chondroma, chondroblastoma, chondromyxoid fibroma	Chondrosarcoma
Fibrous tissue	Cortical defect, non ossifying fibroma, periosteal desmoid, fibrous dysplasia	Fibrosarcoma
Hematologic	Eosinophilic granuloma	Metastases, lymphoma
Unknown	Giant cell tumor, bone cyst, aneurysmal bone cyst	Ewing sarcoma
Vascular	Hemangioma	Epithelioid hemangioendothelioma
Others	Dermoid or epidermoid cyst	Chordoma, adamantinoma

#### **Plain Films**

Although there have been significant improvements in CT, MR, and nuclear medicine during the past years, conventional radiography still remains the first step in the analysis of a bone tumor. Radiographs may be requested because of clinical symptoms, such as pain or tumefaction. The tumor may also be an incidental finding on a radiograph performed for another reason.

Two orthogonal views are taken. The analysis should follow a systematic approach:

- 1. Type of bone involved; long bone or flat bone, spine or girdle.
- 2. Situation within the bone long axis: epiphysis, metaphysis, diaphysis or several: articular involvement, for example an epiphyseal lesion in a child is most likely a chondroblastoma: in the axial plane: medullary, cortical, juxta-cortical; Table 2 summarizes possible etiologies according to the axial situation.
- 3. Size: more than 6 cm suggests malignancy
- 4. Appearance of the bone reaction: lytic, condensing or mixed.
- A condensing reaction may result from osteoblastic stimulation, osseous necrosis, osteogenic tumoral matrix, or peripheral reaction to osteolysis.
- Osteolysis can be described as:
  - a. Geographic: in which there is a relatively large, well defined hole or a few confluent holes with sharply

Table 2. Etiology according to axial position within the bone

Central	Bone cyst, enchondroma, osteoblastoma, Ewing
Lateral	Giant cell tumor, chondromyxoid fibroma, aneurismal bone cyst, osteosarcoma, osteoblastoma
Cortical	Cortical defect, osteoid osteoma, aneurismal bone cyst, osteosarcoma, Ewing, osteoblastoma
Juxta-cortical or paraosteal	Osteochondroma, chondroma, aneurismal bone cyst, osteosarcoma, Ewing



well circumscribed area (Lodwick type IA) at the tibial metaphyso-diaphyseal junction, typical of non-ossifying fibroma. No other imaging should be performed

Fig. 1. Plain film of

the knee in a teenager after trauma. Lytic





**Fig. 2a, b.** A 7-year-old with knee pain. **a** Plain films: lytic area with sharp edge (Lodwick type Ib) in the superior tibial epiphysis suggestive of benign lesion. The epiphyseal location suggests chondroblastoma. **b** CT confirms the radiological appearance and demonstrates some calcifications within the tumoral matrix

defined edges (Lodwick type 1a) (Fig. 1), or well defined margins but no sclerosis (type 1b) (Fig. 2a) or ill defined margins (type 1c).

- b. Moth-eaten (type II) (Fig. 3a): defined as multiple holes of moderate size that tend to coalesce.
- c. Permeative (type III): multiple tiny holes, principally in cortical bone.

The type of osteolysis gives an approach clue as to the aetiology (Table 3).



**Fig. 3a, b.** 8-year-old girl with pain in humerus. **a** Plain film: Osteolysis type II, moth-eaten appearance, in superior humeral diaphysis extending to the metaphysis, suggestive of an aggressive process. **b** MRI, coronal T1 sequence showing low signal within the medullary cavity. A diagnosis of osteosarcoma was made at biopsy

Table 3.	Etiology	according	to type	of bone	e reaction
----------	----------	-----------	---------	---------	------------

Geographic osteolysis Type Ia and b	Simple bone cyst, aneurismal bone cyst, cortical defect, fibrous dysplasia, chondroma, chondroblastoma (1a), Chondromyxoid fibroma, Eosinophilic granuloma, Giant cell tumor (1b) Circumscribed osteomyelitis
Geographic osteolysis Type Ic, II, III	Malignant tumors, eosinophilic granuloma Osteomyelitis
Condensing lesions	Osteoid osteoma, osteoma, osteosarcoma, bone infarct, osteomyelitis, consoliding fracture
Mixed, lytic and condensing reaction	Malignant tumors, osteomyelitis

- 5. Cortical layer appearance: ruptured or blown out if the tumor is aggressive, thickened in a slowly growing tumor.
- 6. A periosteal reaction is due to reactive osteogenesis when disease reaches the periosteum. Its appearance depends on the rapidity of the abnormal process. It is spiculated, and perpendicular to the cortex in aggressive, rapidly growing tumors; it is lamellar, parallel to the cortex, and thick in slowly progressing disease.
- 7. A soft tissue mass may be due to extra-osseous development of some tumors, such as osteochondromas or paraosteal sarcoma, or associated with the bone tumor, as in Ewing sarcoma, or other tumors. In this case, it is considered aggressive.
- 8. Bone remodelling or complication such as fracture.

This systematic approach leads to a distinction between aggressive and non aggressive lesions. In most cases, diagnosis is suggested according to age, tumor location, and radiological appearance of the tumor.

Benign, non aggressive lesions, such as cortical defect, non ossifying fibroma, periosteal desmoid, fibrous dysplasia, chondroma, osteochondroma, simple bone cyst, and vertebral angioma, should be recognized on plain films, thereby avoiding biopsy.

When an aggressive lesion is diagnosed, further imaging, mainly magnetic resonance imaging (MRI), is mandatory before any biopsy.

In some cases, when the diagnosis is questionable because of the particular location of the tumor (pelvis, spine), or when further analysis of the tumoral matrix is necessary, CT may be the proper examination.

# Ultrasound

Ultrasound is not useful in evaluating bone tumors, although it may aid in visualizing the soft tissue mass.

# **Computerized Tomography**

This approach has been supplanted by MRI in the evaluation of malignant tumors but remains of interest to demonstrate a subtle cortical lesion or thin periosteal bone formation. It can also be used to analyze the tumoral matrix (Fig. 2b) and search for calcifications, to evaluate density (fat, fluid within cysts, fluid-fluid level suggestive but not specific of aneurysmal bone cyst) or particular locations, such as pelvis and spine. CT, mostly indicated for analysis of the bone itself, should be performed without contrast. However, if MR cannot be obtained, CT may replace it and contrast will delineate the soft tissue mass.

With new multidetectors machines, thin slices are obtained; reformatted images are possible and often informative.

Another indication for CT is evaluation of lung metastases if the tumor has proven to be malignant.

# **Magnetic Resonance Imaging**

Magnetic resonance imaging has supplanted other techniques in the evaluation of malignant tumors. It should be performed before any biopsy because bleeding secondary to biopsy modifies the images. The protocol for an MR examination is now well established and should include (Table 4):

- 1. An initial sequence using a coil, which allows visualization of the whole bone, including both articulations above and below the tumor. This may be completed with a spin echo T1 (Fig. 3b) or STIR sequence along the long axis of the bone (double obliquity is often necessary). This allows the detection of skip lesions (small metastases within the bone marrow but not in continuity with the primary tumor) and joint involvement.
- 2. The other sequences (spin echo T1 and T2) may then be performed with a surface coil to achieve better resolution, with parameters such as thickness and slices orientation being adapted to tumor location. The tumor appears hypointense on T1 weighted sequences, allowing clear delineation between infiltrated marrow and normal bright fatty marrow. Skip metastases are also demonstrated. T2 weighted images are useful to delineate a soft tissue mass, which appears hyperintense compare to adjacent muscles. Neurovascular structures, surrounded by a fatty rim, are recognized on both T1 and T2 sequences.
- 3. Intravenous administration of gadolinium increases the signal intensity of pathological tissues, delineates cystic or necrotic areas, and distinguish peritumoral edema from tumoral tissue. Fat saturation must be used.
- 4. MR angiography is sometime necessary to better assess the vessels.
- 5. Dynamic contrast-enhanced MRI has been used to appreciate tumor viability. A very short T1 sequence is performed before and repeated after gadolinium rapid infusion and subtracted images are obtained; enhanced areas represent viable tumor. This method may be used also during follow-up, allowing evaluation of the tumoral response to treatment.

MR is usually not necessary in the case of benign tumors but if requested then one must be aware of the pitfalls. One example is the possible over appreciation of marrow edema in the case of an osteoid osteoma, which should not be mistaken for intramedullary involvement from a malignant tumor. Edema has ill defined margins and is faintly hypointense on T1 weighted images compared to clearly hypointense, sharply delineated tumoral

Table 4. Protocol for MRI of a probably malignant tumor

- SE T1 or STIR along the long axis of the bone showing both extremities
- SE T1- and T2-weighted sequences using a surface coil
- Fat-saturated T1 post-gadolinium, 3 planes

marrow infiltration. Another point is the absence of a soft tissue mass in the case of benign tumor. As with CT, MR may assess the diagnosis of specific tumors, such as aneurysmal bone cyst when showing fluid-fluid levels.

Whole body MR has recently been used to evaluate metastatic bone disease. Coronal SE T1 or STIR sequences have detected more lesions than Tc99MDP scintigraphy in some cases.

#### **PET Scan**

Although a new technique and not yet easily available, is probably very efficient in metastatic evaluation.

#### Follow-Up Under Treatment

On plain film, the tumoral matrix calcifies during chemotherapy, soft-tissue mass should reduce and may also calcify. The best way to appreciate tumor reduction is MRI: comparative measurements are taken of the intra medullary component and soft-tissue mass. However, MRI does not always reflect tumoral status (percentage of necrotic tumor after chemotherapy), which is obtained by pathological analysis of the specimen.

# **Soft-Tissue Tumors**

Soft tissue tumors are frequent in children, and are mostly benign. Malignant tumors are rare, often misdiagnosed at the beginning and inadequately treated by surgery. Diagnosis relies on pathology with immunohistochemical analysis. Cytogenetics and molecular biology are very

**Table 5.** Enzinger and Weiss classification (simplified)

helpful in diagnosing sarcomas, but may only be performed on fresh specimen. Part of the biopsy should be kept frozen in all cases for further analysis.

The reference classification by Enzinger and Weiss is summarized in Table 5. The most frequently occurring benign tumors in children are vascular tumors. Amongst the malignant tumors, rhabdomyosarcomas represent 60%.

#### Imaging

Conventional radiography may show calcifications or fat within the soft tissues, and bone modifications.

Ultrasonography is very convenient as a first approach and may be sufficient in pseudotumoral lesions (adenitis, cysts, hematomas), benign tumors (hemangiomas, fibromatosis colli), or vascular malformations. Doppler, using parameters adapted to slow flows, demonstrates avascular or cystic lesions, and solid tumors vascularization.

MRI is often necessary and should be performed before any biopsy. The coil is adapted to the lesion size and location, a cutaneous mark may help. SE T1 and T2, STIR sequences are the most informative, whereas gradient echo sequences are often artifactual but may help to demonstrate hemosiderin. Fat saturation after gadolinium improves the contrast and should be performed in all cases. Dynamic MR shows areas of solid tumor and guides the biopsy. It may also help during follow-up to demonstrate residual tumor or local recurrences. Diffusion and spectroscopy have been used recently but are still being evaluated.

There is no specificity and malignant lesions may appear totally cystic.

	Benign	Malignant
Fibrous tissues	Fibroma, fibromatosis fibrous histiocytoma, xanthogranuloma	Congenital infantile fibrosarcoma, myofibro- blastic tumors, malignant histiocytofibroma
Fatty tissues	Lipoma, lipoblastoma	liposarcoma
Muscular tissues	Leiomyoma Rhabdomyoma	Leiomyosarcoma, rhabdomyosarcoma, alveolar RMS, embryonal RMS, undifferentiated RMS
Vascular masses	Infantile hemangioma, vascular malformation, lymphangioma	Angiosarcoma, malignant hemangiopericytoma, Kaposi
Nervous tumors	Neurofibroma, plexiform neurofibroma, schwanoma, melanotic progonoma, granular cell tumor	Malignant tumors of peripheral nerves (MPNST) PNET
Synovial tumors	Giant cell tenosynovial tumor	Synovialosarcoma, malignant giant cell tenosynovial tumor
Extra-osseous bone and cartilaginous tumors	Ossifying myositis, extraskeletal chondroma,	Chondrosarcoma, osteosarcoma
Germinal tumors	Mature teratoma	Malignant germ cell tumor
Unclassable	Pilomatrixoma, myxoma	Alveolar sarcoma, rhabdoid tumor
Metastasis		Neuroblastoma, leukemia, malignant melanoma
Pseudo-tumors	Abscess, hematoma, , synovial cyst, popliteal cyst, meningocele, granuloma	

RMS, rhabdomyosarcoma; MPNST, malignant peripheral nerve sheath tumor; PNET, primary neuro-echodermal tumor

CT indications are now limited to very specific situations, such as demonstrating calcifications in cases of possible ossifying myositis. In this situation, diagnosis relies more on imaging than on the pathologic appearance, which may be misleading. The other indications for CT are thoracic or abdominal parietal lesions, where artifacts are significant on MRI.

The indications for PET scan have not been established yet.

As for bone tumors, imaging must take the following into account:

- 1. Topography
- 2. Size: large lesions being most likely malignant.
- 3. Tissue characteristics: fluid seen in pseudotumoral lesions, vascular, or necrotic malignant tumors; calcifications as in neuroblastomas, dermoid, or subcutaneous pilomatrixomas, phleboliths in venous malformations, peripheral calcifications of myositis ossificans; fat in lipomas or lipoblastomas. Hyposignal T2 area suggests a benign lesion, such as fibrous tumor, neurofibromas, fibromatosis.
- 4. Vascularization: multiple vessels with signal void on MR suggest infantile hemangiomas. On US, precise criteria are mandatory: more than 5 vessels per cm2, velocity over 2 kHz; these allow differentiation from other highly vascularized tumors, such as myofibromatosis or malignant tumors.

However, it is not always possible to recognize a malignant tumor. On US, structure or homogeneity is non specific. A vascularization pattern on power Doppler that includes anarchic vessels and trifurcations, suggests malignancy. Total absence of flux suggests a benign lesion.

On MRI, there are no specific morphological or signal criteria, but some are suggestive of malignancy, such as a size >5 or 6 cm, absence of hyposignal on T2, inhomogeneous appearance on T1, peripheral enhancement, and extension to bone or neurovascular structures. Sharp margins or peritumoral edema are not reliable criteria.

After clinical and imaging evaluation, a decision of follow up only may be made if suspicion of a benign lesion is high. In all other cases, percutaneous or surgical biopsy (not total excision) should be requested. Treatment is then decided upon when the precise nature of the tumor is known.

# **Suggested Readings**

- Teo HE, Peh WC (2004) The role of imaging in the staging and treatment planning of primary malignant bone tumors in children. Eur Radiol 14(3):465-475
- Miller SL, Hoffer FA (2001) Malignant and benign bone tumors. Radiol Clin North Am 39(4):673-699
- De Schepper AM, De Beuckeleer L, Vandevenne J, Somville J (2000) Magnetic resonance imaging of soft tissue tumors. Eur Radiol 10:213-223
- Enzinger FM, Weiss SW (1995) Soft tissue tumors. 3rd edn. Mosby, St. Louis
- Dubois J, Garel L (1999) Imaging and therapeutic approach of hemangiomas and vascular malformations in the pediatric age group. Pediatr Radiol 29, 879-893
- Shapeero LG, Vanel D, Verstraete KL, Bloem JL (2000) Fast magnetic resonance imaging with histologic correlation. Radiographics 20, 1007-1019



# Imaging the Child's Inflammatory and Infectious Musculoskeletal Pathology

# S.G.F. Robben

Department of Radiology, University Hospital Maastricht, AZ, Maastricht, The Netherlands

# Introduction

The purpose of this article is to emphasize the role of imaging in the diagnosis of various diseases in childhood, including cellulitis, subcutaneous abscess, necrotizing fasciitis, pyomyositis, infectious bursitis and arthritis, osteomyelitis, foreign bodies, infectious lymphadenitis, and non-infectious inflammatory disease, such as juvenile chronic arthritis. Scintigaphy and cross-sectional imaging techniques, such as ultrasonography (US), computed tomography (CT) and magnetic resonance imaging (MRI) have dramatically improved the ability to evaluate infectious and non-infectious inflammatory disease. Selection of the optimal techniques for each individual patient is essential, and factors such as cost, radiation dose and need for sedation should all be considered. US is an important initial modality for evaluation of musculoskeletal infections in children because it is rapid, non-ionizing, and very sensitive for (infectious) fluid collections and joint effusions. Moreover, the images are not degraded by metallic- or motion artefacts (as with CT and MRI). US also offers the possibility of fine-needle aspiration to confirm the infectious nature of a fluid collection without unnecessary contamination of adjacent anatomical compartments. US should be combined with radiography because the two imaging techniques are complimentary. Thus, along with conventional radiography, US is a very valuable modality for early diagnosis and follow-up of musculoskeletal infections in children.

Scintigraphy (three-phase bone scan with technetium-99m) has a high sensitivity for bone disease but a low specificty. Combining bone scintigraphy with gallium-67 and indium-111 can improve diagnostic performance [1].

MRI and CT are not screening methods but are very useful in detailing osseous and soft-tissue changes. CT allows good definition of cortical bone changes and deep soft-tissue abcesses that cannot be seen with US. MRI is superior in diagnosing soft-tissue abnormalities, bonemarrow changes, cartilage destruction and involvement of the growth plate by an infectious process. The major drawback of MRI is the need for sedation children 6 years or less.

# Cellulitis

Cellulitis is defined as an infection of the skin and subcutaneous tissues, with a predilection for the extremities in children [2]. *Staphylococcus aureus* and *Streptococcus pyogenes* account for the majority of the infections. Depending on the type of infection and the patient's immune system, cellulitis may either spread or remain localized to form an abscess. Patients present with soft-tissue swelling, erythema and fever.

Conventional radiographs often show non-specific soft-tissue swelling. The US appearance resembles edema of the subcutaneous fat, showing swelling, increased echogenicity of the subcutaneous fat with decreased acoustic transmission, blurring of tissue planes, progressing to hypoechoic strands between hyperechoic fatty lobules (Fig. 1). This appearance is non-specific and cannot be distinguished from non-infectious causes of soft-tissue edema [3]. Increased vascularity at color or power Doppler US suggests an infectious cause [4].

Cellulitis is usually a clinical diagnosis and the additional value of imaging techniques lies in the detection of underlying abscesses. Therefore, when the initial US examination shows only cellulitis, but the symptoms persist, the US examination should be repeated, because the characteristic US features of an abscess and liquefaction may become more apparent in time [5]. Moreover, cellulitis, especially in the vicinity of bone,



Fig. 1. Non-specific edema of subcutaneous fat (between *arrow-heads*). Hypoechoic strands surround small islands of hyperechoic fat

can be simulated by early stages of osteomyelitis. Once a suspicious fluid collection in the soft tissues is identified, US-guided puncture should be performed for diagnostic purposes.

CT findings of cellulitis include skin thickening, increased attenuation of the subcutaneous fat to more than –90 HE, loss of distinction between tissue planes, heterogeneous enhancement and normal deep fascial and muscle compartments [6, 7].

MR imaging shows an ill-defined linear or domeshaped area of low T1 and high T2 signal intensity with an interspersed, network-like appearance of the subcutaneous fat. Underlying bones and muscles are normal [6].

# **Necrotizing Fasciitis**

Necrotizing fasciitis is a rare, rapidly progressive, and often fatal infection of the subcutaneous tissues, fascia, and surrounding soft-tissue structures. Early diagnosis is mandatory because the disease may have a fatal course if adequate therapy (extensive surgical debridement and antibiotics) is not instituted promptly. Causative organisms are *S. aureus* and group A streptococcus. Although necrotizing fasciitis in its early phase can mimic cellulitis, imaging can be helpful in the diagnosis.

The sonographic features of necrotizing fasciitis are: (a) fascial thickening and accumulation of fluid, (b) cloudy fluid or loculated abscess in the fascial plane, allowing US\_guided diagnostic aspiration, (c) subcutaneous soft-tissue swelling, and (d) eventually, gas in soft tissues [4, 8].

CT demonstrates decreased attenuation of the superficial fascia compatible with necrosis and relative sparing of the muscular compartment. Additionally, gas may be seen dissecting along fascial planes and deeper fluid collections appear [7].

The MR image of necrotizing fasciitis resembles that of infectious cellulitis to a certain extent; however, there is also involvement of the deep fascia and intramuscular spaces. Abnormal gadolinium enhancement is caused by contrast extravasation from damaged capillaries in areas of necrosis. The depth of the soft-tissue involvement does not seem to be a reliable parameter to differentiate between cellulitis and necrotizing fasciitis [6, 9].

# **Soft-Tissue Abscess**

An abscess is defined as a collection of necrotic tissue, neutrophils, inflammatory cells, and bacteria walled off by highly vascular connective tissue [7]. Most abscesses are caused by *S. aureus*. Superficial abscesses begin as cellulitis but subsequently liquefy to form a localized pus collection. Deep abscesses, such as subperiosteal abscesses and pyomyositis, are discussed below.

Conventional radiographs may show non-specific softtissue swelling and, occasionally, gas in the soft tissues.

The ultrasonographic appearance of abscesses is highly variable: they may present with or without mass effect and the liquefied contents may be anechoic, hypoechoic, hyperechoic, and even isoechoic to surrounding tissues. The margins may be relatively sharp, blend in with the surrounding cellulitis, or outlined by an echogenic rim [5, 10]. Because of the variable ultrasonographic appearance, many diseases may simulate abscesses: seromas, hematomas, ganglion, synovial cysts and cystic tumors (anechoic contents). Hematomas and solid masses show hypoechoic contents: solid and necrotic tumors, hyperechoic contents: and cellulitis or edema, isoechoic contents [3, 5]. To confirm the liquid nature of a non-anechoic mass, the presence of "ultrasonographic fluctuation" should be looked for. This sign implies the motion of particles induced by gentle pressure of either the transducer or the finger of the sonographer [5, 10, 11]. Especially, small sinus tracts can be detected with this technique.

Another important tool in the characterization soft-tissue masses is color or power Doppler US [3, 4, 10, 12-14]. An abscess shows absence of flow within its contents and hyperemia in its direct surroundings. Therefore, Doppler US helps to discriminate inflammatory and infectious fluid collections from non-inflammatory collections, but it is not possible to discriminate between infectious and non-infectious inflammatory fluid collections [14]. Finally, US is extremely valuable in detecting fluid collections and abscesses around orthopedic hardware (Fig. 2) [4]. The value of CT and MRI is limited by the presence of artifacts, especially next to orthopedic devices. Also, isotope studies may show non-specific findings due to recent surgery.

CT demonstrates a focal fluid collection (HU>20) whose density varies with the relative amounts of proteinaceous fluid, debris, and necrosis. The central cavity is surrounded by a thick irregular rim that enhances after



**Fig. 2a, b.** An 8-year-old boy with progressive pain and fever after proximal femur osteotomy. Clinical inspection showed minor swelling but normal aspect of the skin. **a** Lucency around proximal part of the orthopedic hardware and extreme varus deformity of femoral neck, suggesting loosening. **b** Turbid fluid collection (between callipers) along orthopedic material (*arrows*), sealed from subcutaneous compartment by the fascia of the tensor lata muscle. Culture showed *S. aureus* 

contrast injection. These imaging findings can be better seen with MR [15]; the low signal intensity of the fluid on T1 and its high signal intensity on T2 contrast nicely with the enhancing rim. The presence of an enhancing rim on post-gadolinium images has a high sensitivity and specificity for the diagnosis of soft-tissue abscess. The abscess fluid itself does not enhance.

#### Osteomyelitis

Osteomyelitis is defined as an infection of the bone marrow. The most common organism is S. aureus, although, less frequently, Streptococcus, Escherichia coli and Pseudomonas aeruginosa are also cultured from blood or aspirates [7]. The incidence of *Haemophilus influenzae* osteomyelitis has decreased dramatically since the introduction of HIB vaccination [16]. The manifestation of osteomyelitis in children is age-dependent. In infants, diaphyseal vessels penetrate the growth plate to reach the epiphysis, facilitating epiphyseal and joint infections in this age group [17].

In older children, the growth plate constitutes a barrier for the diaphyseal vessels. Vessels at the metaphysis terminate in slow-flow venous sinusoidal lakes, predisposing the metaphysis as the starting point for acute hematogenous osteomyelitis.

The increased pressure within the medullary cavity causes the infection to spread via the Haversian and Volkmann's canals into the subperiosteal space. Because the periosteum is less firmly attached to the cortex in infants and children than in adults, elevation will be more pronounced in childhood osteomyelitis. In contrast, sequestration is rare in neonatal osteomyelitis [7].

Conventional radiography is usually the initial modality demonstrating deep soft-tissue swelling in early disease. Bone destruction and periosteal reaction become obvious only 7-10 days after the onset of disease. Nonetheless, conventional radiography is a screening method that may suggest the diagnosis, exclude other pathology, and can be correlated with other imaging findings.

Recently, several reports have recommend the use of US for the early diagnosis of osteomyelitis, especially in children [18-22]. The spectrum of initial ultrasonographic changes comprises deep edema, thickening of the periosteum, intra-articular fluid collection, and subperiosteal abscess formation (elevation of the periosteum by more than 2 mm) [19]. These findings precede radiographic changes by several days [21-24]. In addition, US can detect cortical defects in the course of the disease (Figs. 3, 4). The detection of subperiosteal abscesses is especially important because in these patients ultrasonographically guided aspiration or surgical drainage has to be considered, whereas patient with osteomyelitis without abscesses can be treated with antibiotics only. In animal studies subperiosteal abscesses are visualized ultrasonographically as a band of decreased echogenicity bordered by a line of increased echogenicity representing subperiosteal fluid and periosteal reaction, respectively [24, 25]. In children, sub-



A 13-year-old girl presented with complaints of periods of low-grade fever and a progressive swelling of the proximal

humerus, 4 years after treatment for acute osteomyelitis of the same humerus. A metaphyseal defect is present, as shown with ultrasonography (a) and radiography (b). c At the site of the swelling a subcutaneous abscess (arrows) is visible, containing a sequestrated bony fragment (arrowhead). d The abscess (A) is in continuity with the cortical defect (D) via a long fistula (marked with dots)





Fig. 4a, b. Chronic osteomyelitis. A 9-year-old girl with a 7-month history of arthralgia presented with a 3week history of a swelling at the sternoclavicular joint on the left. a Ultrasonography of left proximal clavicle. Although the cortex (vertical arrows) appears to be intact, there are echoes from the medulla (curved arrows), suggesting subtle permeative

changes of the cortex facilitating the passage of sound waves into the bone marrow. b Coronal T2 weighted MR image with fat suppression. The proximal left clavicle (arrows) shows increased signal intensity of medulla, cortex and surrounding soft tissues. Note normal right clavicle. A diagnosis of cat-scratch osteitis was made by the detection of Bartonella henselae DNA by PCR analysis of bone aspirate. Active infection was confirmed with serological tests

periosteal abscesses are spindle-shaped fluid collections along the cortex of a bone, either with increased or decreased echogenicity. In equivocal cases, the use of color Doppler US is mandatory. Pus collections with increased or decreased echogenicity will present as avascular periosteal masses with peripheral hyperemia [26]. However, it should be noted that color Doppler flow is not detectable earlier than 4 days after the onset of symptoms [26].

CT demonstrates osseous abnormalities earlier then conventional radiographs, but at the expense of a higher radiation dose. It is superior to MR imaging for visualizing bony destruction, gas in the bone, and a bony sequestration.

Like CT. MRI is not a screening method but is invaluable in demonstrating the intra- and extraosseous extension of osteomyelitis. Predictors of early osteomyelitis are ill-defined, low T1 and high T2 signal intensity; poorly defined soft-tissue planes; lack of cortical thickening; and poor interface between normal and abnormal marrow. In chronic osteomyelitis, there is a good differentiation between diseased marrow and soft-tissue abnormalities [1]. Sensitivity and specificity of gadolinium-enhanced MR imaging for osteomyelitis is reported at 97 and 92%, respectively [15].

# **Pyomyositis**

Pyomyositis is a suppurative bacterial infection in striated muscle. It is rare because striated muscle is relatively resistant to bacterial infection and is encountered most frequently in tropical regions. All striated muscles of the skeleton can be involved, but there is a predilection for muscles in the thigh and pelvis [27]. Contributing factors are trauma, diabetes mellitus, chronic steroid use, connective tissue disorders, and immunosuppression (HIV, malnutrition and chemotherapy) [3, 7]. Children are affected in one third of cases, both in tropical and non-tropical regions [27-29]. The most frequent causative organism is S. aureus. Pyomyositis can be difficult to diagnose because initially the infection is confined to the muscular compartment, causing myalgia, general malaise, and fever. It is often difficult for the child to localize the pain, particularly when pyomyositis involves the hips or pelvis. Also, the lack of awareness about the disease, especially in non-tropical settings, may cause diagnostic delay.

Ultrasonographic findings depend on the stage of the disease [11, 30]. Stage 1 (phlegmonous) shows localized muscle edema and distortion of the filamentous planes with ill-defined areas of decreased echogenicity. Stage 2 (suppurative) shows liquefaction corresponding with abscess formation. The echogenicity of the pus may be either increased, decreased, or equal to that of the surrounding tissues. Doppler ZS and gentle compression with the transducer to visualize the motion of particles can be useful in equivocal cases (see sections on osteomyelitis and soft-tissue abscesses). The presence of gas within an inflamed muscle is very suggestive of abscess formation caused by anaerobic organisms (Fig. 5) [31].





fever and pain of the right thigh, 6 month after surgery for an adenocarcinoma of the left kidney. a Ultrasonography shows reflections in quadriceps muscle with bright acoustic shadowing (arrows), suspect for intramuscular gas. F Femur. Conventional radiograph (b) and CT (c) confirm the presence of intramuscular gas. Blood cultures revealed gram-negative bacteria

The detection of an abscess in myositis is important because it requires drainage for complete resolution whereas stage 1 disease can be treated with antibiotic therapy alone. US has the advantage over other modalities that it can be used to guide percutaneous aspiration and drainage.

CT demonstrates enlargement and heterogeneous attenuation of the affected muscle with a focal fluid collection. Associated findings of cellulitis are always seen, with soft-tissue inflammatory stranding and skin thickening and loss of delineation between tissue planes.

MR imaging can identify abscesses and co-existing areas of osteomyelitis and septic arthritis. In stage 1 disease, heterogeneous T2 signal intensity is present in the enlarged muscle. In stage 2 disease, abscess formation is seen as a focus of T2 high signal intensity and T1 low signal intensity. Gadolinium demonstrates peripheral rim enhancement.

#### Septic arthritis

The hip joint is the most frequent location of septic arthritis in childhood, with the knee, shoulder and elbow also being common sites [7, 32]. Early diagnosis is mandatory to prevent cartilage destruction, joint deformity, growth disturbance and eventually premature arthrosis. Most commonly, it is caused by hematogeneous seeding or, less frequently, by extension into the joint space from osteomyelitis. Etiologic organisms are S. au*reus* (most common), group A streptococcus, and S. *pneumoniae*. In neonates, group B streptococcus and *E. coli* are important causes whereas *Neisseria* gonorrhoeae can occur in adolescents [7]. The incidence of *H. in-fluenzae* has declined since large vaccination programs were introduced [16]. The presenting sympoms are fever, non-weight bearing, erythrocyte sedimentation rate >40, and a peripheral white blood count of >12,000. If all these symptoms are present, the likelihood of septic arthritis is 99% [33]. Unfortunately, many children do not show such an obvious clinical picture; thus, imaging techniques are important tools to give additional information of the suspected joint.

Conventional radiographs may be normal or demonstrate joint-space widening with adjacent soft-tissue swelling. However, sensitivity and specificity for septic arthritis are low.

US is very sensitive for the detection of joint effusion, especially in the pediatric population,. Small amounts of fluid (up to 1 ml) can be detected. However, the specificity for the diagnosis is poor [34]. The absence of joint effusion virtually excludes septic arthritis [35], although Gordon et al. recently described two patients with septic arthritis of the hip with symptoms <24-h-old that showed no effusion on the initial ultrasonographic examination [36]. Neither the size, nor the echogenicity of the effusion can distinguish infectious from non-infectious effusion [35, 37-39] (Fig. 6). Physiological synovial fluid in asymptomatic joints can be visualized during specific maneuvers (endorotation of the hip) and appear as multiple small reflections, even more numerous than in pathological effusions (Fig. 7).

In adults, power Doppler US helps to differentiate noninflammatory fluid collections from those that are inflammatory and infectious, because the latter shows an increased adjacent soft-tissue perfusion. However, it does not reliably distinguish inflammatory collections of infectious and non-infectious origin, because both infectious and non-infectious inflammatory fluid collections show the same degree of hyperemia [14]. In children with hip-joint effusion, Strouse et al. showed that increased flow has a sensitivity of 27% and a specificity of 100% for the diagnosis of septic arthritis [40]. Therefore, power Doppler US does not allow exclusion of septic arthritis of the hip. Despite the technical innovations in US, joint aspiration is still necessary in equivocal cases [34].

CT is less sensitive for the detection of joint effusion but may identify areas of adjacent osteomyelitis. MR is very sensitive for the detection of small amounts of fluid. Initially, MR imaging reveals distention of the joint capsule by non-specific T2 high-signal-intensity fluid. In later stages, the joint effusion tends to have a more intermediate signal intensity and seems heterogeneous. Moreover, MR can demonstrate cartilage destruction and adjacent cellulitis. [41]. Combined with gadolinium injection and fat suppression techniques, a sensitivity of 100% and specificity of 77% can be accomplished [42].







**Fig. 6a-c.** Three patients with non-infectious hip joint effusion. **a** A 6-year-old boy with transient synovitis of 1-day duration. The effusion is clear. **b** A 7-year-old girl with transient synovitis of 7-days duration. Ultrasound shows small particles (*arrow*) can be seen floating in the effusion. **c** A 3-year-old girl with postviral reactive arthritis of 20-days duration; ultrasound shows cloudy effusion



Fig. 7a-c. Normal synovial fluid in asymptomatic children. **a** Small amount of synovial fluid containing many bright reflections in the superior articular recess of the hip joint during forced endorotation. **b** An in-vitro study shows normal synovial fluid (diluted with saline) in a test tube. The fluid shows identical reflections as the samples in **a** 

# **Infectious Bursitis**

Infectious bursitis in childhood is rare. Two series in the past 25 years, each consisting of ten children, have specifically addressed infectious bursitis [43, 44]. As in adults, local trauma is the most common risk factor in childhood [44, 45]. Causative organisms are S. aureus in the majority of patients, or group A Streptococcus. The prepatellar bursa is most commonly affected; less frequently the olecranon bursa is involved. The affected bursa is distended because of fluid accumulation and/or synovial thickening. The fluid may be clear or turbid, with or without septations. [3, 4, 10]. As in infectious arthritis, it is not possible to differentiate between infectious and non-infectious inflammatory bursitis (post-traumatic and rheumatoid bursitis). In order to confirm the diagnosis, aspiration of fluid is necessary. The advantage of ultrasonographically guided aspiration is two-fold: (a) the needle tip can be guided away from synovial hypertrophy and towards small fluid collections within the bursa; (b) when infectious bursitis is confused with infectious arthritis, US will identify the bursa as the causative structure, preventing contamination of a normal joint when the needle tip is inadvertently placed into the joint space after passing the infected bursa. Once diagnosed, rapid recovery is usually seen after adequate treatment.

On CT, an infected bursa resembles an abscess. The central fluid collection may appear heterogeneous due to debris or hemorrhage. With MR imaging, the fluid in the bursa is T1 low and T2 high signal intensity, although large amount of debris will cause an increase of the T1 signal intensity. Adjacent soft tissues may demonstrate feathery edema, and after intravenous gadolinium the



margins of the bursa will show enhancement. Occasionally, adjacent bone shows some edema on fatsuppressed images.

#### **Foreign Body**

Foreign bodies can be responsible for chronic recurrent infection resistant to antibiotic therapy. US has a sensitivity of 83% for foreign bodies (93% for wooden foreign bodies and 73% for plastic foreign bodies). Specificity is rather low (59%) [46]. The ultrasonographic aspect depends on the material involved and the reaction of the surrounding tissues to the foreign body. Metal and glass cause posterior reverberation artifacts whereas wood results in posterior acoustic shadowing [47]. Secondary infection are responsible for a suppurative or granulomatous reaction around the foreign body, facilitating its detection (Fig. 8).

# Conclusion

Diagnosis of musculoskeletal infection in children is difficult and challenging. Imaging studies play an important role. Conventional radiography and ultrasonography are the initial imaging modalities. The most important additional value of US in diagnosing pediatric musculoskeletal infections over other imaging modalities is the capability to detect fluid and the possibility to perform ultrasonographically guided aspiration. However, the value of gray-scale and Doppler imaging should not be underestimated: US provides important information on localization, architecture, relation to surrounding tissues, vascularity, and the behavior of a lesion over time. Each of these items separately contributes to the definitive diag-





**Fig. 8a, b.** Wooden splinter in the subcutaneous tissues of the heel. Transverse (**a**) and sagittal (**b**) images. The acoustic shadowing is caused by the foreign body itself, the surrounding hypoechoic halo is probably caused by chronic inflammatory tissue

nosis. This diagnostic capacity adds to the other, unrivalled advantages of US such as portability, availability, speed, and patient comfort. An important limitation for visualizing deeper structures, such as pelvic, paravertebral and mediastinal lesions, is the restricted acoustic window, limited penetration depth, and lack of penetration through bone and air. In these cases, CT and MR imaging are valuable additional techniques to evaluate the suspected anatomical region.

#### References

- Oudjhane K, Azouz EM (2001) Imaging of osteomyelitis in children. Radiol Clin North Am 39(2):251-266
- Choa H, Lin S, Huang Y, Lin T (2000) Sonographic evaluation of cellulitis in children. J Ultrasound Med 19:743-749
- Struk DW, Munk PL, Lee MJ, Ho SG, Worsley DF. (2001) Imaging of soft tissue infections. Radiol Clin North Am 39(2):277-303

- Cardinal E, Bureau N, Aubin B, Chhem R (2001) Role of ultrasound in musculoskeletal infection. Radiol Clin North Am 39(2):191-201
- Loyer EM, DuBrow RA, David CL, Coan JD, Eftekhari F (1996) Imaging of superficial soft-tissue infections: sonographic findings in cases of cellulitis and abscess. AJR 166:149-152
- Rahmouni A, Chosidow O, Mathieu D, Gueorguieva E, Jazaerli N, Radier C et al (1994) MR imaging in acute infectious cellulitis. Radiology 192(2):493-496
- Kothari NA, Pelchovitz DJ, Meyer JS. (2001) Imaging of musculoskeletal infections. Radiol Clin North Am 39(4):653-671
- Chao HC, Kong MS, Lin TY (1999) Diagnosis of necrotizing fasciitis in children. J Ultrasound Med 18(4):277-281
- Loh NN, Ch'en IY, Cheung LP, Li KC (1997) Deep fascial hyperintensity in soft-tissue abnormalities as revealed by T2-weighted MR imaging. AJR Am J Roentgenol 168(5):1301-1304
- Bureau N, RY C, Cardinal E (1999) Musculoskeletal infections: US manifestations. Radiographics 19:1585-1592
- Loyer EM, Kaur H, David CL, DuBrow R, Eftekhari FM (1995) Importance of dynamic assessment of the soft tissues in the sonographic diagnosis of echogenic superficial abscesses. J Ultrasound Med 14(9):669-671
- Sofka CM, Collins AJ, Adler RS (2001) Use of ultrasonographic guidance in interventional musculoskeletal procedures. J Ultrasound Med 20:21-26
- Newman JS, Adler RS, Bude RO, Rubin JM (1994) Detection of soft-tissue hyperemia: value of power Doppler sonography. AJR Am J Roentgenol 163(2):385-389
- Breidahl WH, Newman JS, Taljanovic MS, Adler RS (1996) Power Doppler sonography in the assessment of musculoskeletal fluid collections. AJR Am J Roentgenol 166(6):1443-1446
- Mazur JM, Ross G, Cummings J, Hahn GA, Jr., McCluskey WP (1995) Usefulness of magnetic resonance imaging for the diagnosis of acute musculoskeletal infections in children. J Pediatr Orthop 15(2):144-147
- Bowerman SG, Green NE, Mencio GA (1997) Decline of bone and joint infections attributable to Haemophilus influenzae type b. Clin Orthop (341):128-33
- Asmar BI (1992) Osteomyelitis in the neonate. Infect Dis Clin North Am 6(1):117-132
- Bar-Ziv J, Barki Y, Maroko A, Mares AJ (1985) Rib osteomyelitis in children. Early radiologic and ultrasonic findings. Pediatr Radiol 15(5):315-318
- Howard CB, Einhorn M, Dagan R, Nyska M (1993) Ultrasound in diagnosis and management of acute haematogenous osteomyelitis in children. J Bone Joint Surg Br 75(1):79-82
- Kaiser S, Rosenborg M (1994) Early detection of subperiosteal abscesses by ultrasonography. A means for further successful treatment in pediatric osteomyelitis. Pediatr Radiol 24(5):336-339
- Abernethy LJ, Lee YC, Cole WG (1993) Ultrasound localization of subperiosteal abscesses in children with late-acute osteomyelitis. J Pediatr Orthop 13(6):766-768
- Riebel TW, Nasir R, Nazarenko O. (1996) The value of sonography in the detection of osteomyelitis. Pediatr Radiol 26(4):291-297
- Mah ET, LeQuesne GW, Gent RJ, Paterson DC (1994) Ultrasonic features of acute osteomyelitis in children. J Bone Joint Surg Br 76(6):969-974
- 24. Cheon JE, Chung HW, Hong SH, Lee W, Lee KH, Kim CJ et al (2001) Sonography of acute osteomyelitis in rabbits with pathologic correlation. Acad Radiol 8(3):243-249
- Abiri MM, DeAngelis GA, Kirpekar M, Abou AN, Ablow RC (1992) Ultrasonic detection of osteomyelitis. Pathologic correlation in an animal model. Invest Radiol 27(2):111-113
- Chao HC, Lin SJ, Huang YC, Lin TY (1999) Color Doppler ultrasonographic evaluation of osteomyelitis in children. J Ultrasound Med 18(11):729-734) quiz 735-736

- 27. Spiegel DA, Meyer JS, Dormans JP, Flynn JM, Drummond DS (1999) Pyomyositis in children and adolescents: report of 12 cases and review of the literature. J Pediatr Orthop 19(2):143-150
- Christin L, Sarosi GA (1992) Pyomyositis in North America: case reports and review. Clin Infect Dis 15(4):668-677
- Chiedozi LC (1979) Pyomyositis. Review of 205 cases in 112 patients. Am J Surg 137(2):255-259
- Belli L, Reggiori A, Cocozza E, Riboldi L (1992) Ultrasound in tropical pyomyositis. Skeletal Radiol 21(2):107-109
- Brook I. Pyomyositis in children, caused by anaerobic bacteria. J Pediatr Surg (1996) 31(3):394-396
- Lim-Dunham JE, Ben-Ami TE, Yousefzadeh DK. (1995) Septic arthritis of the elbow in children: the role of sonography. Pediatr Radiol 25(7):556-559
- 33. Kocher MS, Zurakowski D, Kasser JR (1999) Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. J Bone Joint Surg Am 81(12):1662-1670
- van Holsbeeck M, Introcaso JH (1992) Musculoskeletal ultrasonography. Radiol Clin North Am 30(5):907-925
- Zawin JK, Hoffer FA, Rand FF, Teele RL (1993) Joint effusion in children with an irritable hip: US diagnosis and aspiration. Radiology 187(2):459-463
- 36. Gordon JE, Huang M, Dobbs M, Luhmann SJ, Szymanski DA, Schoenecker PL (2002) Causes of false-negative ultrasound scans in the diagnosis of septic arthritis of the hip in children. J Pediatr Orthop 22(3):312-316
- Dorr U, Zieger M, Hauke H (1988) Ultrasonography of the painful hip. Prospective studies in 204 patients. Pediatr Radiol 19(1):36-40

- Robben SG, Lequin MH, Diepstraten AF, den Hollander JC, Entius CA, Meradji M (1999) Anterior joint capsule of the normal hip and in children with transient synovitis: US study with anatomic and histologic correlation. Radiology 210(2):499-507
- Marchal GJ, Van Holsbeeck MT, Raes M, Favril AA, Verbeken EE, Casteels-Vandaele M et al (1987) Transient synovitis of the hip in children: role of US. Radiology 162(3):825-828
- Strouse PJ, DiPietro MA, Adler RS (1998) Pediatric hip effusions: evaluation with power Doppler sonography. Radiology 206(3):731-735
- Greenspan A, Tehranzadeh J (2001) Imaging of infectious arthritis. Radiol Clin North Am 39:267-276
- Hopkins KL, Li KC, Bergman G (1995) Gadolinium-DTPAenhanced magnetic resonance imaging of musculoskeletal infectious processes. Skeletal Radiol 24(5):325-330
- Paisley JW (1982) Septic bursitis in childhood. J Pediatr Orthop 2(1):57-61
- Harwell JI, Fisher D (2001) Pediatric septic bursitis: case report of retrocalcaneal infection and review of the literature. Clin Infect Dis 32(6):E102-104
- 45. Garcia-Porrua C, Gonzalez-Gay MA, Ibanez D, Garcia-Pais MJ (1999) The clinical spectrum of severe septic bursitis in northwestern Spain: a 10 year study. J Rheumatol 26(3):663-667
- Hill R, Conron R, Greissinger P, Heller M (1997) Ultrasound for the detection of foreign bodies in human tissue. Ann Emerg Med 29(3):353-356
- Roberts CS, Beck DJ Jr, Heinsen J, Seligson D (2002) Review article: diagnostic ultrasonography: applications in orthopaedic surgery. Clin Orthop (401):248-264