Metabolic-Endocrine

12

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Learning Objectives

- To understand the normal imaging appearance of bone marrow and bone marrow manifestations of metabolic and endocrine disorders.
- To understand the limitations of medical imaging in detecting metabolic bone disorders.
- To understand the imaging findings observed in symptomatic complications associated with metabolic bone disorders.

12.1 Introduction

All components of the musculoskeletal system can be involved with metabolic disorders (Table 12.1) as a result of endocrine diseases, genetic alterations, and environmental or nutritional aspects, with important worldwide variations in prevalence and severity. Early detection of these disorders is crucial because of the efficacy of preventive measures and availability of treatments. The current chapter will focuson the imaging appearance of metabolic disorders of bone marrow and of the mineralized skeleton. Marrow and bone disorders in athletes, the elderly, and individuals with eating disorders will be reviewed.

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Table 12.1 Metabolic disorders of the musculoskeletal system

Muscles	Sarcopenia, drug-induced myopathies (statins), necrosis (diabetes mellitus), fat atrophy (steroids)
Tendons	Chondrocalcinosis, hypercholesterolemia, hyperuricemia, hypervitaminosis A, drug-induced tendinopathy (quinolones)
Cartilage	Chondrocalcinosis, hyperuricemia, recurrent hemorrhage, hemochromatosis, ochronosis
Synovium	Hyperuricemia, amyloidosis
Bone	Osteopenia, osteoporosis, rickets, osteomalacia, renal osteodystrophy, hyperparathyroidism, thyroid acropachy, acromegaly, drug-induced disorders
Bone marrow	Hemosiderosis, marrow aplasia, anemias, serous atrophy, fatty atrophy, drug-induced disorders

12.2 Metabolic Marrow Disorders

12.2.1 Structure, Function, and Development of Bone Marrow

The marrow cavity is divided into compartments by bony trabeculae. There are two types of marrow, red or hematopoietic marrow and yellow or fatty marrow.

Red (hematopoietic) marrow is hematopoietically active and is composed of various hematopoietic stem cells, lymphocytes, and lymphoid nodules, supported by reticulum cells and fat cells. Red marrow contains approximately 40% water, 40% fat, and 20% protein [1]. Blood supply is through centrally located nutrient arteries. Bone marrow lacks lymphatic channels [1].

Yellow (fatty) marrow is composed predominantly offat and contains approximately 15% water, 80% fat, and5% protein. Marrow adipose tissue has been increasingly recognized as an active and dynamic endocrine organ that responds to changes in nutrition and environmental milieu and can function as a biomarker for skeletal integrity and bone strength [2].

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12.2.1.1 Normal Development of Bone Marrow

The normal distribution and MR appearance of bone marrow changes with age. It is important to be familiar with the appearance of bone marrow during aging to be able to determine whether processes represent a disease or normal variations of marrow. Bone marrow and related disorders are best assessed using MRI [3].

12.2.1.2 Red to Yellow Marrow Conversion

At birth, hematopoietic marrow is present throughout the entire skeleton including the epiphyses. Normal physiologic conversion of red to yellow marrow occurs in a predictable fashion. It begins in the hands and feet progressing to the peripheral (appendicular) and then the central (axial) skeleton. In the peripheral skeleton, this process begins in the epiphyses, followed by the diaphyses, the distal metaphyses, and then the proximal metaphyses. Marrow conversion is typically complete by 25 years of age. However, residual red marrow can be present in the metaphyses of long bones in the adult, and in some cases subchondral red marrow can be seen in the proximal epiphyses of the humerus and femur. Although distribution of red marrow varies from person to person, it is usually symmetric in the same person [3](Fig. 12.1).

12.2.1.3 Reconversion of Yellow to Red Marrow

Reconversion from yellow to red marrow is triggered by demand for increased blood cell production and can be seen in anemia, smoking, obesity, athletes, high altitude, or with certain treatments. It occurs in the mirror-opposite sequence of red to yellow marrow conversion. In the axial skeleton, the proximal metaphyses convert first, followed by the distal metaphyses, and the diaphyses, while the epiphyses usually only convert to red marrow in severe cases. The extent of reconversion depends on the duration and severity of the initiating cause [3–5] (Fig. 12.1).

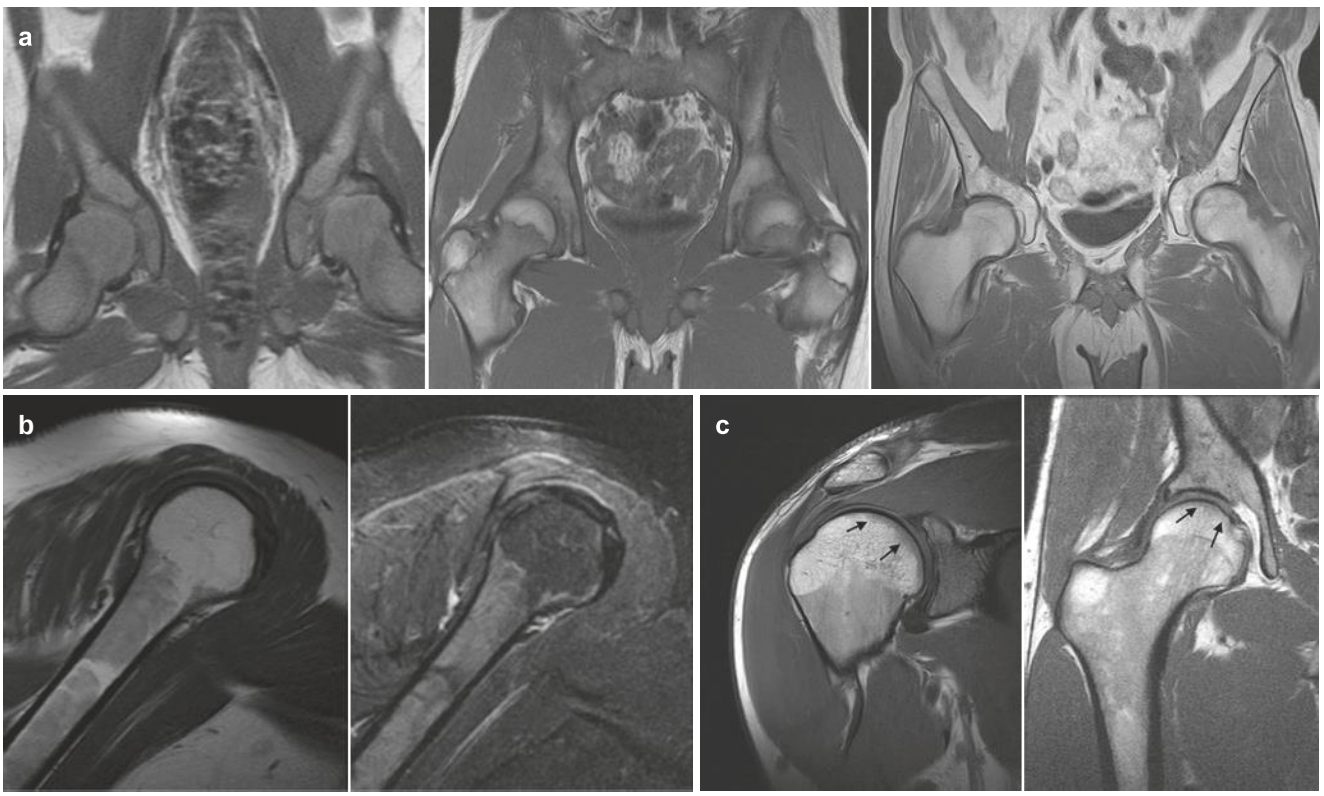


Fig. 12.1 Normal appearance of bone marrow. (**a**) T1-weighted MR images of the pelvis showing normal physiologic conversion of red (hematopoietic) to yellow (fatty) marrow. At birth, marrow is red and diffusely hypointense. During adolescence, physiologic conversion from red to yellow marrow occurs, involving first the epiphyses and epiphysis equivalents, such as the greater trochanters, and diaphyses, while the metaphyses still contain red marrow. In adulthood, there is nearly complete conversion to fatty marrow. (**b**) Marrow reconversion in an athlete. Sagittal T1-weighted and sagittal fat-suppressed T2-weighted MR images show red marrow in the proximal humeral metaphysis and the diaphysis. Red marrow is of intermediate signal and higher in signal than muscle on the T1-weighted image. (**c**) Normal appearance of residual red marrow in the humeral and femoral heads. The curvilinear subchondral distribution of red marrow involving the humeral and femoral heads (arrows) is a normal finding. (**d**) Chemical shift MR imaging of normal marrow in a woman with obesity showing red marrow on the in-phase image and normal signal drop on the out-of-phase image

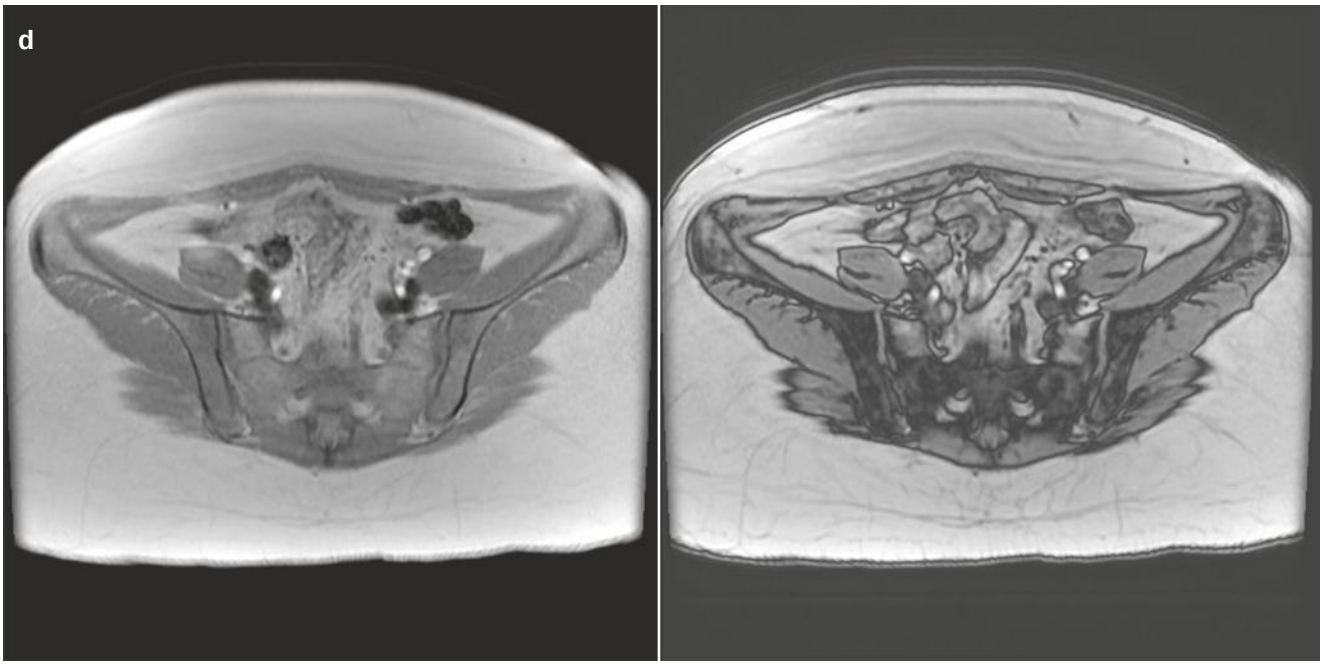


Fig. 12.1 (continued)

Key Points

- Normal physiologic conversion of red to yellow marrow occurs in a predictable fashion starting in the epiphyses, followed by the diaphysis, and the metaphyses.
- Residual red marrow can be found in the metaphyses and subchondral location of the epiphyses of the humeral head and femoral head of adults.
- Marrow reconversion from yellow to red marrow occurs in the reverse order as does conversion from red to yellow marrow.
- Although distribution of red marrow varies from person to person, it is usually symmetric in the same person.

12.2.2 Magnetic Resonance Imaging of Bone Marrow

12.2.2.1 Anatomic Imaging

The major determinants of the MRI appearance of bone marrow are its fat and water content. T1-weighted spin- echo or fast spin-echo/proton density-weighted sequences are most suitable to evaluate bone marrow. Bone marrow is typically hyperintense on T1-weighted images and follows the signal intensity of subcutaneous fat. On fat-suppressed or fluid-sensitive sequences (fat-suppressed T2-/proton density-weighted or short tau inversion recovery (STIR) sequences) bone marrow signal is hypointense. Red marrow demonstrates low signal intensity on T1-weighted images, reflecting its increased water content, and intermediate signal intensity with progressive T2 weighting. On T1-weighted images, normal red marrow is equal to or higher in signal intensity than adjacent muscle or intervertebral disks [1, 3, 4] (Fig. 12.1).

12.2.2.2 Chemical Shift Imaging

Chemical shift imaging also known as water-fat imaging, or Dixon method, represents a MRI-based method that generates water and fat images. Separation of water and fat signal is based on the chemical shift difference between water and fat resonance frequencies and can provide a quantitativemeasurement of the signal fraction of both water and fat [6].This property is used to develop water and fat images by emphasizing in-phase or out-of-phase tissue properties, thus suppressing fat or water signal. Chemical shift imaging can be used as a problem solver to distinguish between benign and malignant etiologies of focal bone marrow abnormalities if conventional anatomic MR images are equivalent. Malignant processes are associated with the replacement of normal bone marrow resulting in a lack of signal intensity loss in the opposed-phase images compared to the in-phase images. Previous studies have calculated a cutoff of 0.75–0.8for the ratio of opposed-phase signal to the in-phase signal in order to differentiate between malignant and benign processes [6, 7] (Fig. 12.1).

12.2.2.3 Proton MR Spectroscopy

Single-voxel proton magnetic resonance spectroscopy (^{1}H-MRS) can provide both quantitative and qualitative information of bone marrow and is typically used in research investigations. Point-resolved spectroscopy (PRESS) and stimulated echo acquisition mode (STEAM) single-voxel ^{1}H-MRS pulse sequences can be employed to obtain fat spectra at

various skeletal sites. The signal within a voxel is divided into two major peaks, a lipid and a water peak which can be determined to assess the lipid-to-water ratio or fat fraction of bonemarrow. The amount of marrow adipose tissue by ^{1}H-MRS in combination with bone mineral density by dual-energy X-ray absorptiometry has been found to be a biomarker for bone health and bone strength, being more valuable than either parameter alone in evaluating skeletal integrity [8, 9].

Key Point

- Normal red marrow demonstrates T1 signal that is equal to or higher than adjacent muscle or intervertebral disks.

12.2.3 Bone Marrow Changes in Athletes

The hematologic responses to physical training are multifactorial. Endurance athletes often have expanded plasma volume and red blood cell mass. Moreover, many endurance athletes have elevated erythropoietin levels, probably due to hypoxia during exercise. Erythropoietin causes increased erythropoiesis and elevated red cell mass. Depleted iron reserves or increased hematopoiesis with elevated erythropoietin levels and reticulocyte count can contribute to the development of hematopoietic hyperplasia in this population. Hematopoietic bone marrow hyperplasia of the spine and extremities has been identified on MRI in endurance athletes [4, 5] (Fig. 12.1).

12.2.3.1 Female Athlete Triad

The female athlete triad refers to a constellation of three clinical entities that are often observed in physically active girls and women. The female athlete triad involves anyone of the three components: (1) low energy availability with or without disordered eating, (2) menstrual dysfunction, and (3) low bone mineral density. Energy availability directly affects menstrual status, and in turn, energy availability and menstrual status directly influence bone health [10, 11]. It is important to identify adolescents who exhibit subclinical abnormalities of the female athlete triad andthus allow for early intervention to prevent fractures later in life. Adolescence is a crucial time of maximal bone accrual toward attainment of peak bone mass, which is a key determinant of future fracture risk. Processes that affect bone health in adolescence are likely to be permanent, leading to increased fracture risk later in life [10].

Imaging findings of the female athlete triad include paucity of body fat and high marrow adipose tissue, low bone mineral density, and impaired bone microarchitecture [12]. Women with the female athlete triad are at high risk for stress fractures and often have higher MRI grades of stress injuries compared with eumenorrheic athletes and injuries being associated with a prolonged time to return to sport [13](Fig. 12.2).

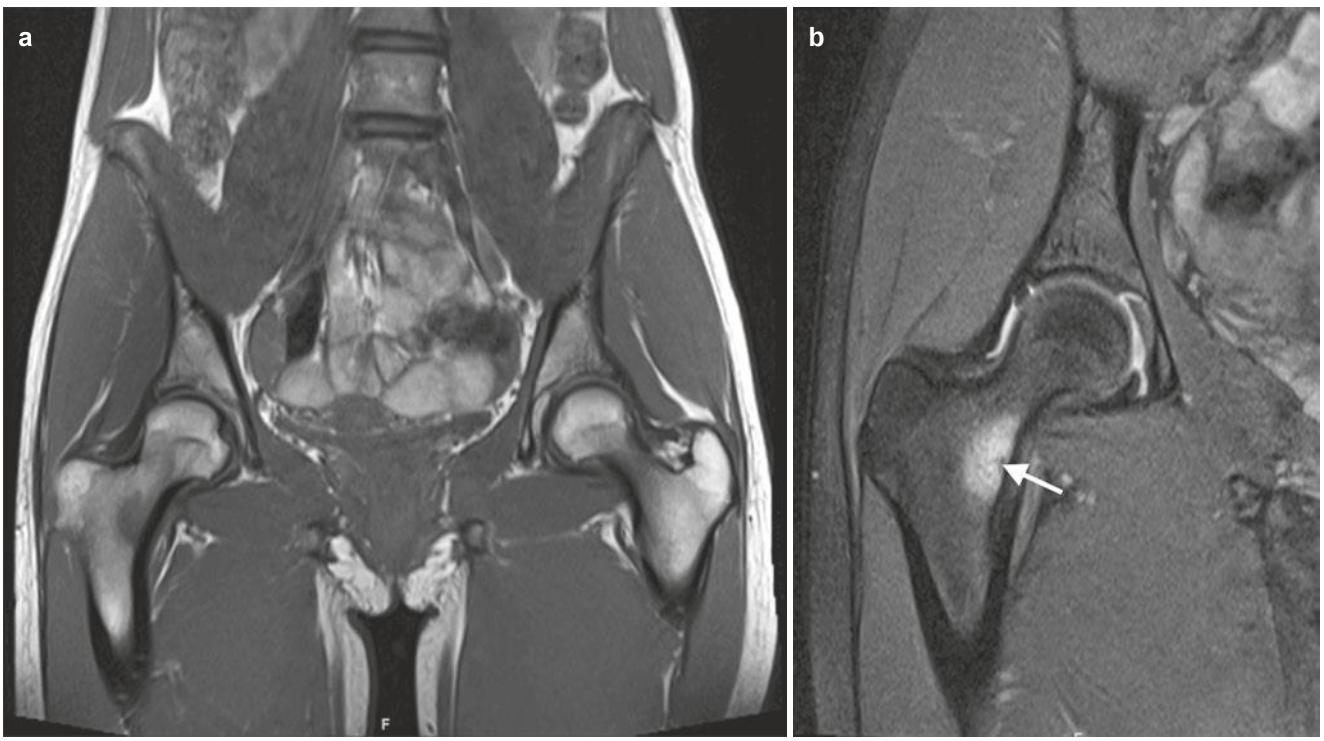
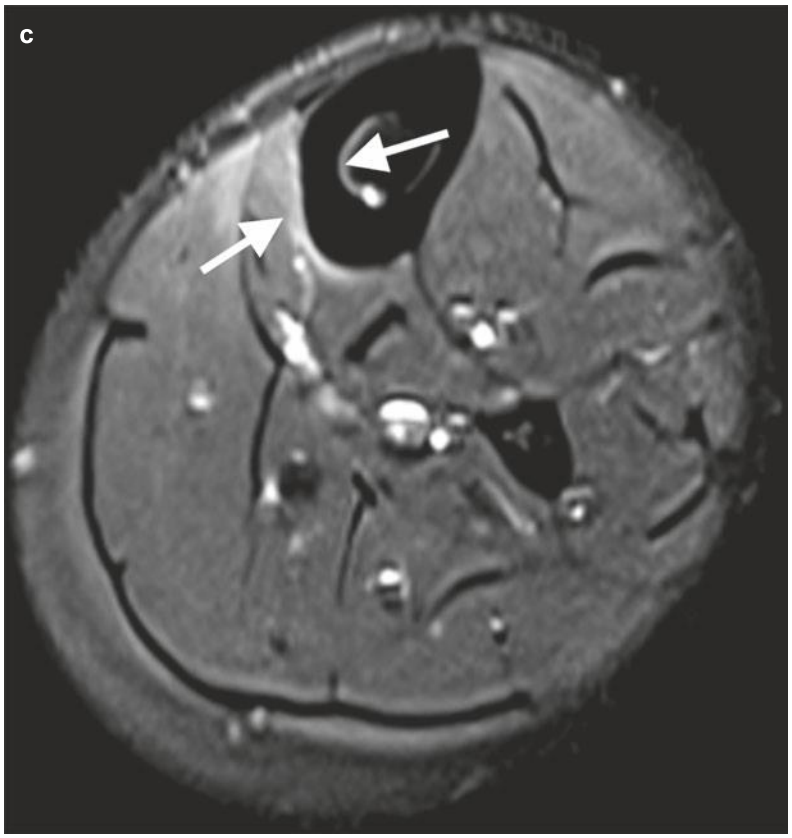


Fig. 12.2 MRI of a 19-year-old runner with the female athlete triad and right-sided hip pain. (**a**) Coronal T1-weighted MR image of the pelvis demonstrates paucity of body fat and signal abnormality of the right femoral neck. (**b**) Coronal fat-suppressed T2-weighted MR image

shows a stress fracture of the right femoral neck (arrow). (**c**) MRI of the lower leg 6 months after the pelvic MRI demonstrates tibial stress reac- tion with periosteal and endosteal edema (arrows)

Fig. 12.2 (continued)



Key Point

- Young female athletes with paucity of body fat and stress fracture on MRI should be evaluated for the female athlete triad.

12.2.4 Bone Marrow Changes in Eating Disorders

Bone marrow can respond to nutritional challenges andmight serve as a biomarker for bone quality. Patients with anorexia nervosa often develop marrow changes that can include decreased marrow cellularity with an increase in fatcontent or a reduction in both fat and hematopoietic cells with an increase in extracellular material, rich in hyaluronic acid, referred to as gelatinous transformation of bone marrow or serous atrophy. In cases of undernutrition (phase II of starvation), body fat can be metabolized with a paradoxical increase in fatty marrow. Bone marrow is relatively resistant to lipolysis and metabolism until other available fat depots have been utilized. During the late phase of starvation (stage III), marrow fat can be metabolized and replaced by hyaluronic acid-rich mucopolysaccharides, leading to gelatinoustransformation or serous atrophy of bone marrow in which there is a significant decrease in the number and size of adipocytes ("atrophy") and hematopoietic cells [14].

On MRI, early fat conversion produces increased signal intensity on T1-weighted images, while late-stage serous atrophy is characterized by water-like signal intensity (i.e., decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images) [14, 15]. Other etiologies of serous atrophy of bone marrow include cachexia, rapid weight loss following bariatric surgery, AIDS, endocrine disorders, and scurvy. Due to bone weakening, insufficiency fractures are common but are often obscured on MRI by the signal abnormalities associated with serous atrophy, often requiring radiographs of CT to detect the fracture [14] (Fig. 12.3). Ofnote, in a multicenter study examining the MRI appearance of serous atrophy, almost one-fourth of cases were misinterpreted as technical error requiring unnecessary repeat imaging [14].

Key Points

- Serous atrophy of bone marrow is often associated with fractures which can be obscured by abnormal MRI signal, requiring radiographs or CT to identify fractures.
- The abnormal MRI signal of serous atrophy of bone marrow is often misinterpreted as technical error requiring unnecessary repeat imaging.

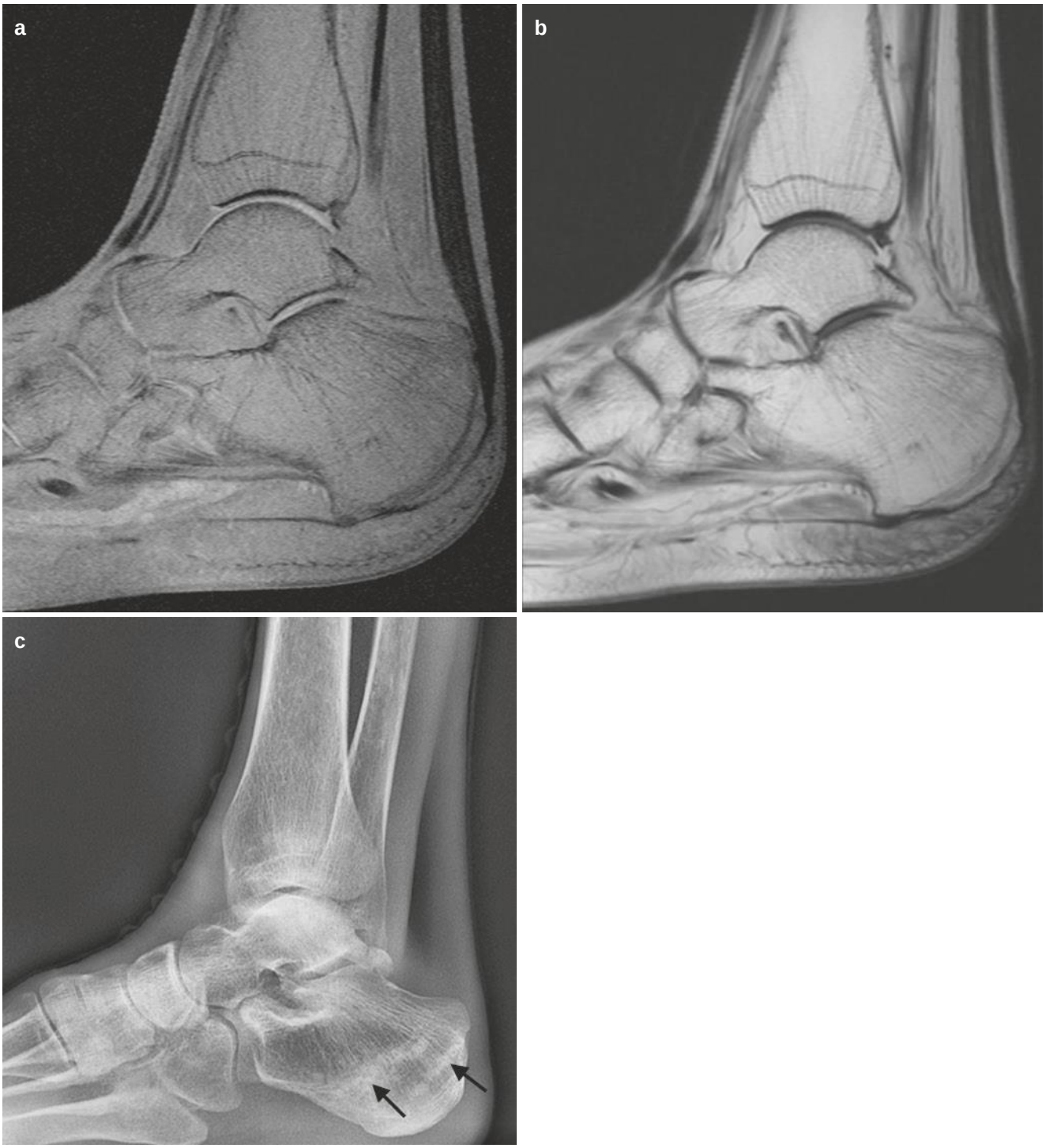


Fig. 12.3 Serous atrophy of bone marrow in a woman with anorexia nervosa referred for MRI of the ankle to rule out stress fracture. (**a**) Sagittal T1-weighted MR image shows diffusely hypointense marrow signal and paucity of subcutaneous fat with similar abnormal hypointense signal. No signal abnormality to suggest fracture. (**b**) Sagittal short tau inversion recovery (STIR) MR image shows diffuse abnormal hyperintense signal of bone marrow and subcutaneous fat. No findings to suggest insufficiency fracture. (**c**) Radiograph of the ankle demonstrates calcaneal stress fractures (arrows), which were obscured on the MRI by the abnormal marrow signal from serous atrophy

12.3 Metabolic Bone Disorders

12.3.1 Structure, Function, and Development of Bone

Bone is a specialized connective tissue made up of a matrix of collagen fibers, mucopolysaccharides, and inorganic crystalline mineral matrix (calcium hydroxyapatite) that are distributed along the length of the collagen fibers.

Bone remains metabolically active throughout life (bone remodeling) at a variable degree (bone turnover) with bone being constantly resorbed by osteoclasts (osteoclastic activity) and accreted by osteoblasts (osteoblastic activity) [16]. Since bone turnover mainly takes place on bone surface, trabecular bone, which has a greater surface-to-volume ratio than compact bone, is consequently some eight times more metabolically active than cortical bone.

The amount of bone in the skeleton at any moment is entirely dependent on the peak bone mass attained during the third decade of life and on the balance between bone resorption and formation. Bone turnover is under the influenceof general factors including age and hormones but is also locally modified by many factors such as physical forces.

Key Points

- Bone remains metabolically active throughout life (bone remodeling) at a variable degree (bone turnover). Trabecular bone is more metabolically active than cortical bone.
- In adults, the amount of bone depends on the peak bone mass attained after adolescence and on bone turnover that is under the influence of systemic and local factors.

12.3.2 Osteoporosis

Osteoporosis, by far the most common metabolic bone disease in Western countries, is a systemic skeletal disease characterized by reduction in bone mass (amount of mineralizedbone per volume unit) and by altered trabecular structure dueto a loss of trabeculae interconnectivity with a consequent increase in bone fragility and susceptibility to fracture [17] (Fig. 12.4).

Fracture risk increases with advancing age and progressive loss of bone mass. The incidence of hip fracture has doubled over the past three decades and is predicted to continue to grow beyond what one would have predicted from increased longevity. At 1 year after hip fracture, 40% of patients are unable to walk independently, 60% have difficulty with one essential activity of life, 80% are restricted inother leaving activity, and 27% will be admitted to a nursinghome for the first time [17]. Questionnaires are available to evaluate fracture risk (FRAX).

Generalized osteoporosis is associated with many diseases and can be either primary or secondary [17]. Presence of a vertebral fracture is a powerful predictor of future fracture, and the accurate identification and clear reporting of vertebral fracture by radiologists have a crucial role to play in the diagnosis and appropriate management of the patient [17].

Several quantitative techniques including dual-energy X-ray absorptiometry (DXA), quantitative computed tomography (QCT), and quantitative ultrasonography (QUS) enable accurate and precise assessment of mineral bone density [2]. Artificial intelligence is likely to play a role in the detection of osteoporosis and assessment of fracture risk by using radiographs and CT examinations [18].

Radiography is relatively insensitive in detecting early bone loss, and 30–40% loss of bone tissue usually remains occult on radiographs. Radiographic bone density is also affected by patient characteristics and technical parameters used to obtain the radiographs.

The main radiographic features of generalized osteoporosis include disappearance of the trabecular network and cortical thinning. Resorption and thinning of trabeculae initiallyaffect secondary trabeculae that are parallel to the biomechanical stresses, and the primary trabeculae that are perpendicular to the biomechanical stresses may appear more prominent because they are affected at a later stage. Corticalthinning occurs as a result of endosteal, periosteal, or intra- cortical (cortical tunnelling) bone resorption. Intracortical tunnelling generally occurs in disorders with rapid boneturnover such as diffuse osteoporosis and reflex sympathetic osteodystrophy. Subperiosteal bone resorption on radiographs is specific for hyperparathyroidism.

Osteoporosis remains occult on MR images although an inverse relationship between trabecular bone density and marrow fat has been reported in some forms of osteoporosis. In the absence of trauma, the presence of multiple vertebral fractures including acute (with bone marrow edema-like changes) and healed (with increased marrow fat) fractures suggests increased bone fragility and, hence, osteoporosis.

Key Points

- Osteoporosis is characterized by a reduction in bone mass and by altered bone architecture with a consequent increase in bone fragility and susceptibility to fracture.
- Radiographs are not sensitive for the detection of osteoporosis, but disappearance of the trabecular network and cortical thinning can be seen in severe osteoporosis.
- Osteoporosis remains occult at MRI.
- Accurate identification and clear reporting of vertebral fractures by radiologists have a crucial role to play in the diagnosis and appropriate management of patient with undiagnosed osteoporosis.

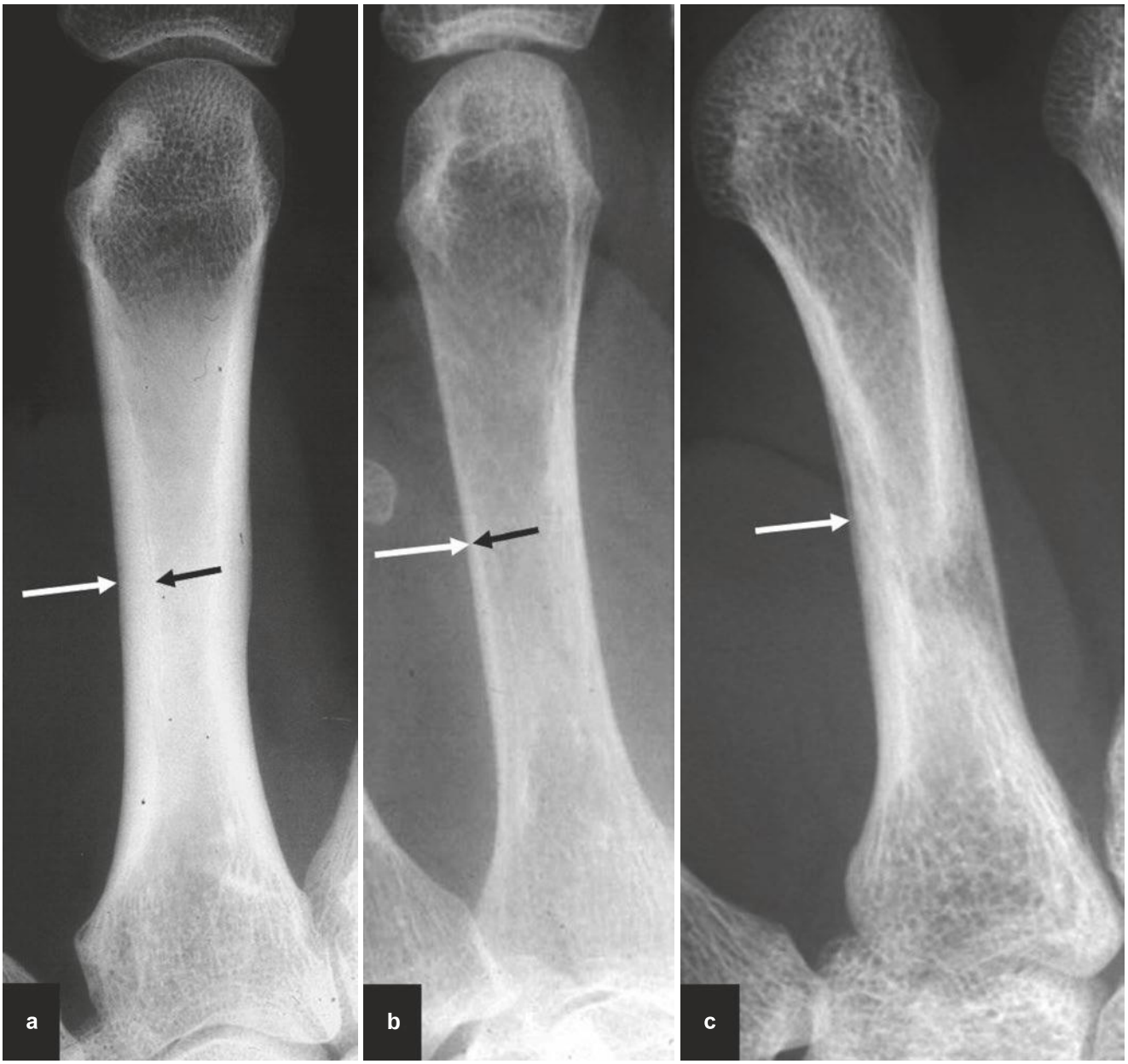


Fig. 12.4 Second metacarpal bones from three patients with (**a**) normal bone, (**b**) osteoporosis, and (**c**) osteomalacia. Osteopenia is characterized by quantitative bone abnormality with decreased bone density and cortical thinning (arrows in **b**). Osteomalacia is characterized by qualitative bone abnormality with intracortical lucencies (arrows in **c**)

12.3.3 Rickets and Osteomalacia

Rickets (in children) and osteomalacia (in adults) are characterized by inadequate or delayed mineralization of oste- oid substance in cortical and trabecular bone (Fig. 12.4) [19].

Looser's zone, the radiological hallmark of osteomalacia, corresponds to a relatively large and ill-delimited lin- ear cortical lucency (the fracture). It typically involves the ribs, the superior and inferior pubic rami, and the inner margins of the proximal femora or lateral margin of the scapula.

Widened physeal growth plate and metaphyseal cupping and fraying are the radiological signs of rickets that are best seen at rapidly growing ends of bone such as distal femur and radius or anterior ends of ribs.

Additional radiological findings of rickets/osteomalacia include bone deformities, osteopenia, or a coarsened pattern of the cancellous bone [19].

At MRI, no specific findings for osteomalacia can be found. Presence of multiple trabecular bone fractures with variable appearance on fluid-sensitive sequences could suggest osteomalacia but is also observed in patients under steroid therapy (Fig. 12.5). Looser's zones are difficult to detect on MR images.

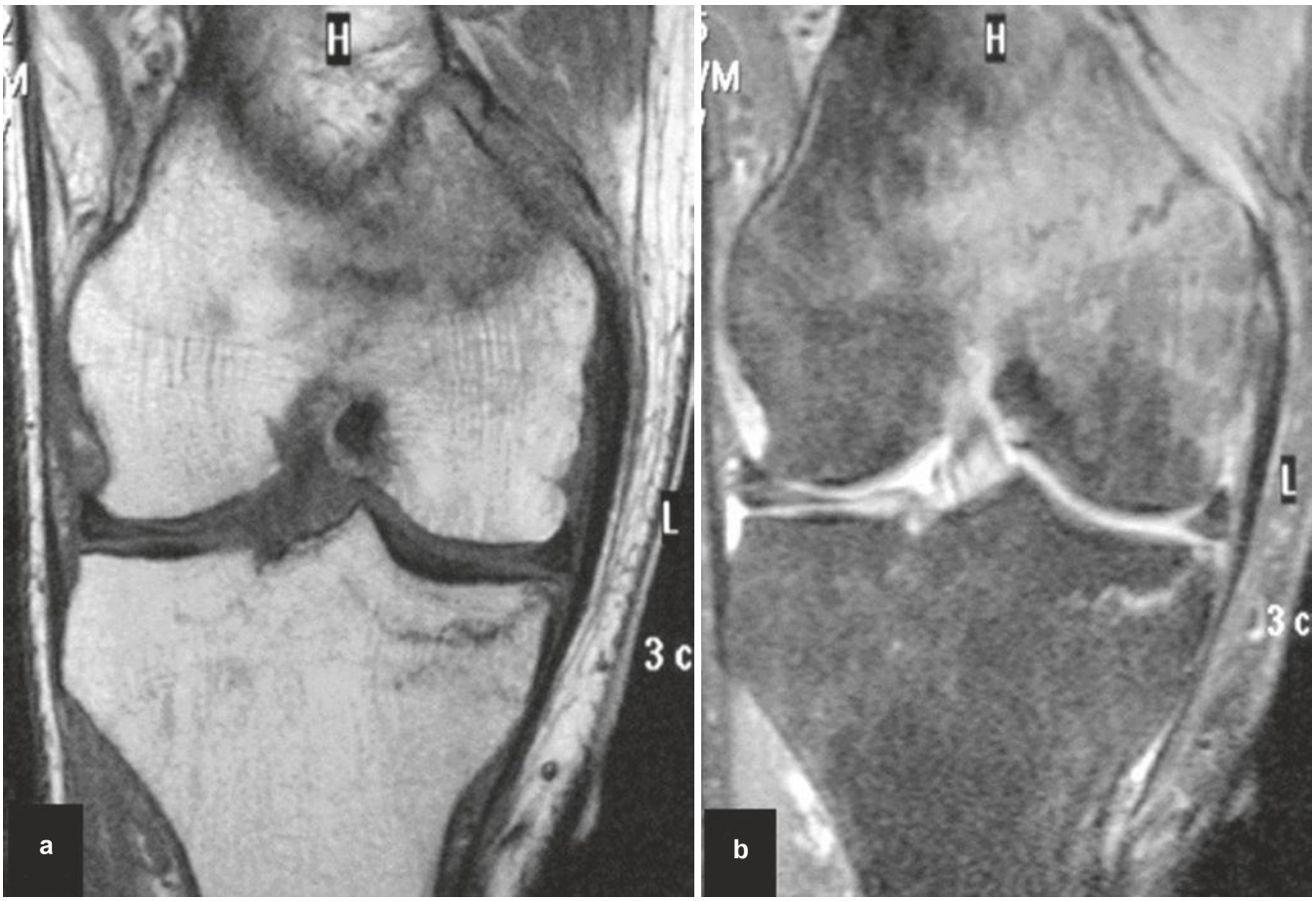


Fig. 12.5 Osteomalacia. Coronal (**a**) T1-weighted and (**b**) fat-suppressed proton density-weighted MR images of the left knee of a 70-year-old man with drug-induced osteomalacia. Multiple trabecular insufficiency fractures (low signal intensity bands) with variable amount of bone marrow edema-like changes are well depicted

Key Point

- Rickets (in children) and osteomalacia (in adults) are characterized by inadequate or delayed mineralization of osteoid substance.

12.3.4 Renal Osteodystrophy and Hyperparathyroidism

Renal osteodystrophy refers to all musculoskeletal manifestations associated with chronic renal failure including osteoporosis, osteomalacia, secondary hyperparathyroidism, and soft tissue calcifications.

The radiographic sign specific for hyperparathyroidism is subperiosteal bone resorption that occurs everywhere but is best visible on the radial side of the second finger phalanges and/or phalangeal tufts [19].

Marginal erosions mimicking synovial disorders can also develop in small joints, most likely due to traction at capsular insertions. They are usually discrete and radiological joint space is preserved. Osteoclastoma or brown tumors are lytic bone lesions associated with chronic hyperparathyroidism. Osteosclerosis may also be encountered in renal osteodystrophy. It is commonly appreciated in the vertebrae, pelvis, ribs, and metaphyses of long tubular bones. In vertebrae, sclerosisis frequently confined to the end plates, producing a characteristic appearance of alternating bands of different density (the "rugger jersey" spine).

Soft tissue calcifications may develop anywhere, in the vessel walls but also in the muscles and tendons. Massive amorphous calcification can develop in the soft tissue around articulations and probably reflect poorly controlled renal osteodystrophy.

Key Points

- Renal osteodystrophy refers to all musculoskeletal manifestations associated with chronic renal failure.
- Subperiosteal bone resorption bets appreciated on the radial aspect of the index phalanges is specific for hyperparathyroidism.

Presence of chondrocalcinosis (knees and wrists) in patients younger than 50 years of age should raise the possibility of hypercalcemia, but hemochromatosis may also present with osteoporosis.

12.3.5 Insufficiency Fractures

Insufficiency fractures result from a normal or slightly increased stress on diffusely weakened bones (Table 12.2). Medical imaging plays a crucial role in the diagnosis of insufficiency and pathological fractures because of the lack of a suggestive clinical history. The distinction between these two conditions can be challenging or even impossible as in patients with multiple myeloma.

Imaging findings of insufficiency fractures depend on the predominantly involved bone (trabecular or cortical bone) and on the time delay between fracture and imaging (acute, subacute, chronic) [20, 21].

Insufficiency fractures generally result from normal compressive forces associated with weight-bearing and are therefore perpendicular to the weight-bearing trabeculae. Due to the absence of trauma, bone deformity and soft tissue swell-ing can be limited.

Early radiographic diagnosis of insufficiency fractures is difficult and periosteal, and cortical callus formation accounts for their delayed detection on radiographs(Table 12.3). Trabecular callus is typically linear and perpendicular to the dominant trabecular network as a result of compressive forces.

Bone marrow edema (BME)-like change is the predominant MR feature of trabecular insufficiency fractures with unclear distinction between stress-induced and fracture-associated changes (Table 12.4).

Table 12.2 Classification of fractures

	Bone strength	Applied forces	Clinical clues
Traumatic fracture	Normal	Increased, acute	History
Fatigue fracture	Normal	Increased, chronic	History
Insufficiency fracture	Diffuse decrease	Normal	None
Pathological fracture	Focal decrease	Normal	None

Table 12.3 Radiologic appearance of trabecular and cortical insufficiency fractures

Radiographs/CT	Acute	Chronic	Healed
Trabecular #	Normal	Mild sclerosis	Normal
Cortical #	Cortical interruption	Periosteal reaction	Cortical thickening

Table 12.4 MR appearance of trabecular and cortical insufficiency fractures

MR imaging	Acute	Chronic	Healed
Trabecular #	Extensive edema	Band, edema	Normal
Cortical #	Subtle marrow and soft tissue edema	Subtle marrow and soft tissue edema Periosteal callus	Normal

In trabecular insufficiency fractures, low signal intensity bands within BME-like changes are important to detect for diagnostic accuracy. Their conspicuity depends on imaging plane and sequences.

In cortical insufficiency fractures, cortical interruption is barely visible on routine MR images. Edema-like changes on the inner and outer sides of the cortex may suggest cortical fractures but tumors or infection should also be considered. Ultra-short TE MR images or CT may contribute to a specific diagnosis by depicting cortical interruption.

12.3.5.1 Vertebral Insufficiency Fractures

Spontaneous vertebral insufficiency fractures predominantly involve the thoracolumbar spine and never occur above T3 level. The anterior and central midportion of the vertebral body withstands compression forces less well than theposterior and outer ring element of the vertebrae, resultingin wedge or end plate fractures or, less commonly, crushfractures.

Vertebral deformity is the radiographic hallmark of vertebral body fracture on radiographs. BME-like changes adjacent to the vertebral end plate and sparing the posterior elements can be seen on MR images of acute fracture.

The low signal bands located a few millimeters underneath the vertebral end plate are important to detect as their presence adds specificity and contributes to narrow the large differential diagnosis associated with vertebral body BME (Fig. 12.6).

12.3.5.2 Pelvic Bone Insufficiency Fractures

Pelvic bone insufficiency fractures predominate in the pubic and ischiatic rami and sacral wings [22]. There is an increased prevalence of these lesions in patients with previous radiation therapy (importance of differential diagnosis with metastases).

MR imaging is the best imaging modality for sacral and supra-acetabular fractures. CT may contribute to the diagnosis by demonstrating cortical interruption or callus formationin trabecular fractures (sacrum).

12.3.5.3 Femoral and Tibial Insufficiency Fractures

Early recognition of femoral insufficiency fractures is extremely important because of possible progression to frac-

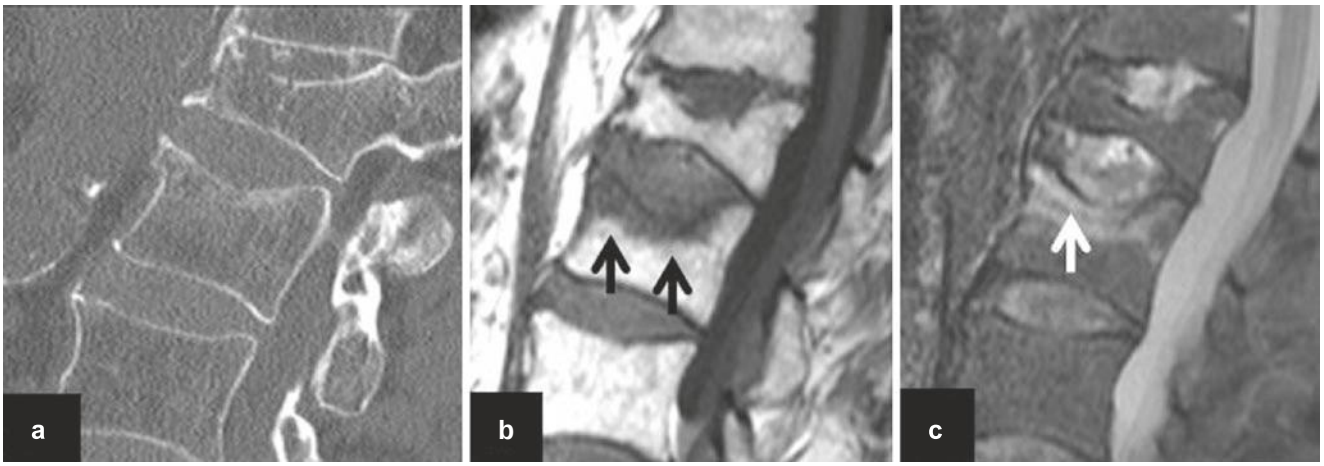


Fig. 12.6 Osteoporosis and vertebral fracture in an 82-year-old woman. (**a**) Sagittal reformatted CT image of the upper lumbar spine shows L1 and L2 vertebral fractures. (**b**) Corresponding T1-weighted MR image shows decreased signal intensity of L2 (recent fracture) (arrows in **b**) and normal signal intensity of L1 (healed fracture). (**c**) On the corresponding sagittal short tau inversion recovery (STIR) image, a low signal intensity band (arrow in **c**) indicates the fracture plane within the bone marrow edema-like signal intensity changes

ture displacement with subsequent increased morbidity. Any region of the femur and of the tibia can be involved.

Atypical insufficiency fracture of the femoral shaft has gained particular attention as they occur in patients with long-standing bisphosphonate therapy for osteoporosis(Fig. 12.7) [23]. These chronic uncomplete cortical fracturescan be multiple and bilateral; they involve preferentiallythe lateral femoral cortex. Transverse cortical lucencies with periosteal beaking can be seen on radiographs or CT reformations. Lesions are barely visible at MRI, because of the limited amount of edema due to lesions chronicity and the limited conspicuity of cortical changes at MRI [24, 25]. Similar fractures occur in the lateral or anterior aspect of the diaphysis in femurs with Paget's disease and in the medial aspect of femurs in patients with osteomalacia.

Longitudinal insufficiency fractures are associated with extensive medullary and soft tissue edema [26]. Cortical interruption, a key diagnostic finding, is visible on short-axis MR or better CT images (with optimal bone settings). The lesion may be confused with infection or tumor at MR imaging because of the longitudinal extent of marrow infiltration.

12.3.5.4 Subchondral Insufficiency Fractures

Subchondral insufficiency fractures generally involve the convex epiphyses of the weight-bearing bones (femoral head and condyles, talar dome, and metatarsal heads) [22, 27–30] (Fig. 12.8).

Radiographs are generally normal but may show subtle subchondral sclerosis. MR imaging reveals BME-like changes that predominate near the subchondral cortical bone of the involved epiphysis. Subtle deformity of the bone contours and low signal intensity bands located at a few millimeters from the subchondral bone plate are generally present.

The differential diagnosis of subchondral BME-like changes is large and includes bone and articular disorders.

12.3.6 Complications of Insufficiency Fractures

12.3.6.1 Displacement of Insufficiency Fractures

Insufficiency fractures usually heal normally with appropriate conservative therapy.

Partial cortical insufficiency fractures of the long bones may progress to overt displaced fractures if not recognized in part because the causative stresses (torsion, compression, traction) favor displacement.

Delayed secondary displacement of trabecular fractures may occur due to persistent compression stresses. Sequelae due to bone deformity can be important, mainly in the spine.

12.3.6.2 Delayed Union and Nonunion

Delayed union and subsequent nonunion of trabecular insufficiency fractures may occur if immobilization is difficult toachieve, if vasculature is sparse, or if the bone metabolism isaltered (radiation therapy, steroids).

Progressive collapse of the vertebral body or the epiphysis may occur in association with nonunion. In the past, this condition was known as "spontaneous osteonecrosis" because of the analogy with systemic osteonecrosis (associated with steroid, alcohol, and others) as both conditions may display intraosseous cleft with or without vacuum phenomenon and do not heal. The so-called spontaneous osteonecrosis of the knee (SONK) (Fig. 12.9) is the best-known of these conditions, but some authors consider that this term is a misnomerand should not be used [27, 28].

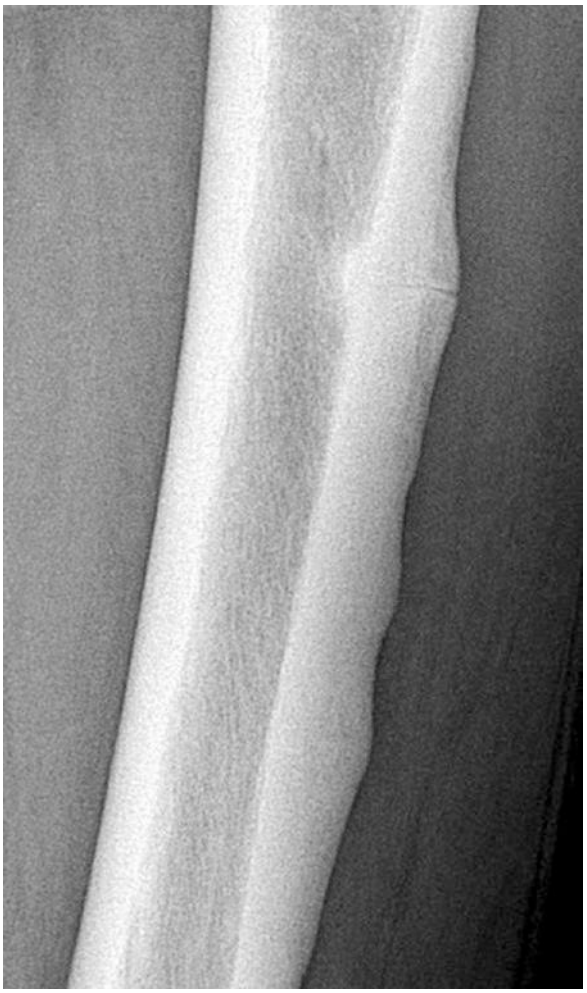


Fig. 12.7 Atypical femoral shaft fractures in a 78-year-old woman with history of long-standing bisphosphonate therapy for osteoporosis. Magnified radiograph of the femoral shaft shows a transverse linear cortical lucency with chronic periosteal reaction (white arrows) on the lateral cortex of the subtrochanteric femur indicative of a partial and chronic fracture of the lateral cortex. Other similar lesions are also visible inferiorly

Vertebral osteonecrosis may lead to spinal cord compression, a very uncommon feature in vertebral insufficiency fractures.

Distinction between "spontaneous osteonecrosis" and "subchondral insufficiency fracture" remains important for therapeutic purpose. Spontaneous osteonecrosis will not heal, whereas subchondral insufficiency fractures may heal. Several MR features may contribute to the recognition of subchondral insufficiency fractures that are more likely to progress to osteonecrosis [29, 30].

Key Points

- Medical imaging plays a crucial role in the diagnosis of insufficiency fractures because of the lack of a suggestive clinical history.
- Imaging findings of insufficiency fractures depend on the involved bone (trabecular or cortical bone) and on their age (acute, subacute, chronic).
- Bone marrow edema-like changes are a predominant but nonspecific finding in recent trabecular insufficiency fractures. Additional low signal intensity bands are important for a specific diagnosis but may be absent.
- Diagnosis of cortical insufficiency fractures is challenging at MRI because of inherent limitations in detecting cortical interruption that can be seen at CT.

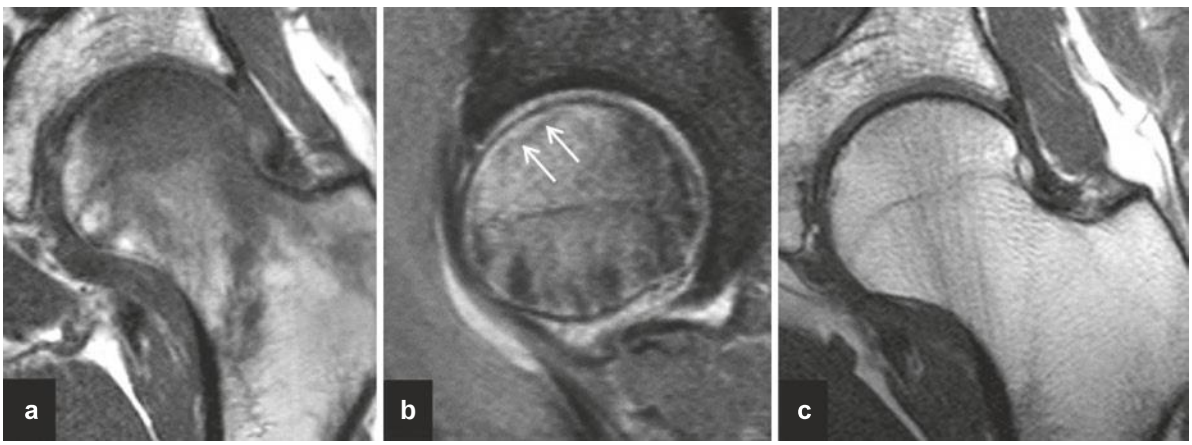


Fig. 12.8 Subchondral insufficiency fracture of the femoral head in a 57-year-old man. (**a**) Coronal T1-weighted MR image of the left hip demonstrates extensive ill-delimited bone marrow edema-like signal changes in the proximal femur that predominate in the subchondral area. (**b**) Sagittal fat-suppressed proton density-weighted image demonstrates a subchondral low signal intensity band (arrows) in the femoral head suggestive of an acute trabecular fracture. (**c**) A follow-up MR image demonstrates complete healing of the lesion

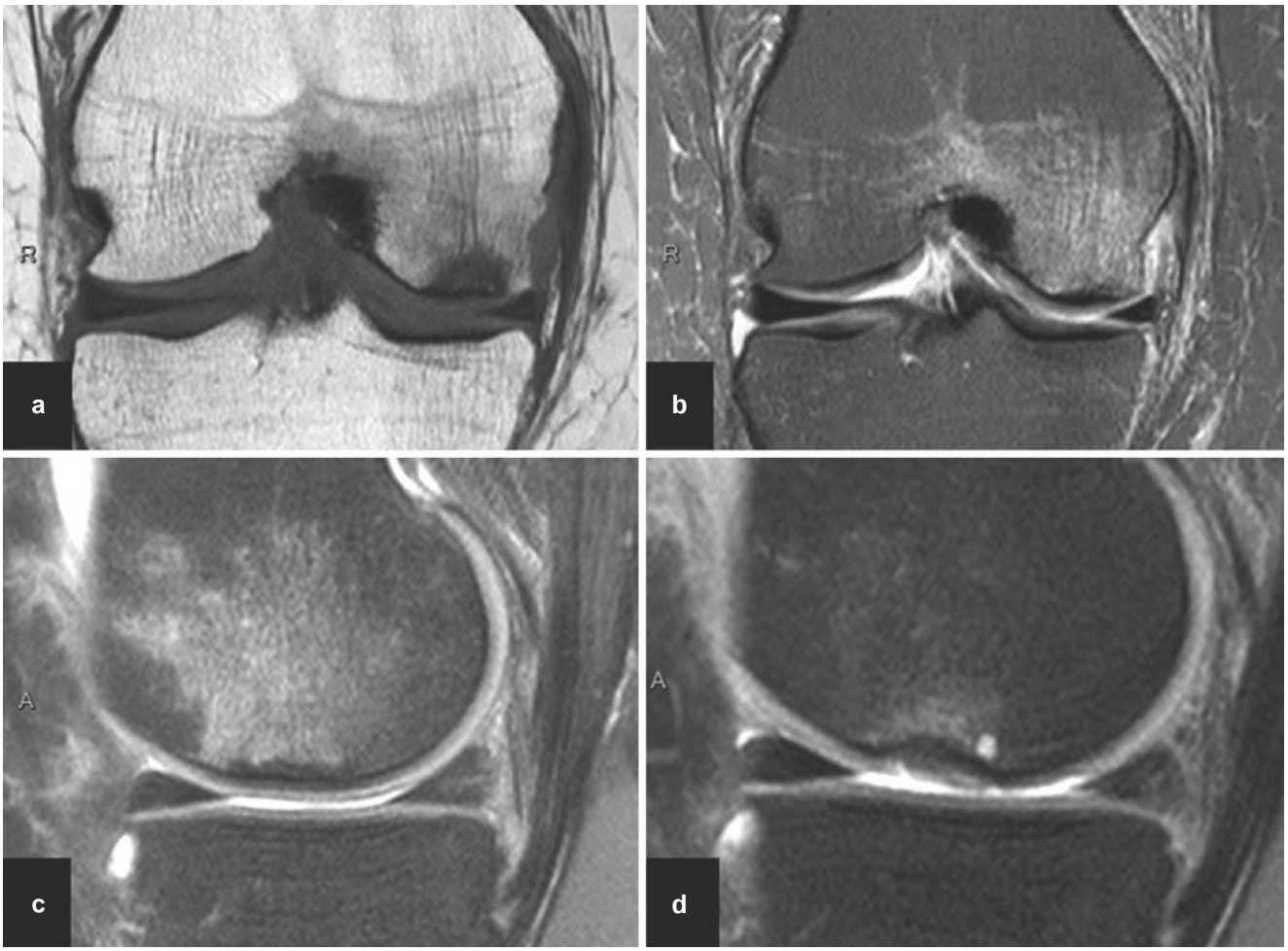


Fig. 12.9 Subchondral insufficiency fracture of the medial femoral condyle in a 65-year-old woman. (**a**) Coronal T1-weighted (**a**) and fat-suppressed proton density-weighted (**b**) MR images demonstrate bone marrow edema-like changes of the medial femoral condyle. On the sagittal fat-suppressed proton density-weighted MR image (**c**), a crescent-shaped low signal intensity line is depicted in the subchondral area with normal bone contour. (**d**) Fat-suppressed proton density-weighted MR image obtained 6 months later demonstrates focal collapse of the condyle and cyst-like bone changes which required subsequent unicompartmental joint replacement

12.4 Concluding Remarks

All components of the musculoskeletal system can be involvedby metabolic disorders as a result of endocrine diseases, genetic alterations, and environmental or nutritional aspectswith important worldwide variations in prevalence and severity. Early detection of these disorders is crucial because of the availability of efficient therapies and preventive measures. Metabolic disorders are usually clinically silent until complications do occur including mechanical failure (bone fracture, tendon tear), necrosis, and inflammation (arthritis). Imaging plays an important role in detecting complications of metabolic disorders but has limited value in their quantification. MR imaging is the method of choice in assessing bone marrow, with T1-weighted imaging being most helpful.

Take Home Messages

- Metabolic disorders can involve all components of the musculoskeletal system.
- Early detection of metabolic disorders is crucial to guide therapy.
- Osteoporosis, the most common metabolic bone disorder, is characterized by a reduction in bone mass and by altered bone architecture with increased fracture risk.
- Musculoskeletal complications of metabolic disorders include mechanical failure (bone fracture, tendon tear), necrosis and inflammation (arthritis).
- MR imaging is the method of choice in assessing bone marrow and insufficiency fractures.