Radiology of Systemic Disorders Affecting the Foot and Ankle

Here's a review of the findings seen on foot and ankle x-rays.

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Goals and Objectives

After completing this CME, the reader should be able to:

- **Discuss and recognize the radiographic features of osteopenia.**
- **Distinguish between the radiographic findings associated with chronic and regional osteoporosis.**
- **Describe and identify the radiographic features of metabolic, endocrine, and nutritional disorders that affect the lower extremity.**
- **Describe and recognize the characteristic radiographic appearance of skeletal dysplasias seen in the lower extremity.**
- **List disorders that are associated with metastatic calcification, calcinosis, and dystrophic calcification.**

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*Following this article, an answer sheet and full set of instructions are provided (pg. 138).—**Editor***

Many systemic disorders demonstrate associated radiographic findings in the lower extremity. Although these conditions typically are not clinically diagnosed by their foot or ankle manifestations, the practitioner should be familiar with these radiologic presentations so they are not misdiagnosed as other conditions. The metabolic diseases, in particular osteoporosis, include several endocrine and nutritional disorders (hyperparathyroidism, acromegaly, scurvy, and rickets, for example). Paget's disease may be localized in the lower extremity. Skeletal dysplasias present characteristic presentations that may be recognized as incidental findings; examples include osteopetrosis, osteopoikilosis, osteochondromatosis, and osteogenesis imperfecta. Finally, soft tissue manifestations of systemic disorders are frequently encountered in the foot and leg.

Decreased bone density, or increased radiolucency of bone,[1,2] is a prominent radiographic feature of numerous metabolic disorders. Misconceptions abound regarding the use of terms relating to this finding;[3] therefore, the following definitions are strictly adhered to in this article. The term osteopenia is used to refer

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to the non-specific radiographic finding of decreased bone density. The terms decalcification, under-mineralization, and demineralization are not used, because they refer more specifically to the underlying physiology and pathology of bone.[4] Moreover, the term osteoporosis is reserved for the clinical entity.

Generalized, diffuse osteopenia is a highly subjective radiographic finding; one must be careful with this "finding" because radiographic technique and/or processing can profoundly influence the visual appearance (that is, the darkness or lucency) of bones. For example, a dark film can result from high-kVp technique, high-mAs technique, increased time in developer, or raised developer temperature. Instead, observe specific, more objective features involving the cortices and/or cancellous bone of the second, third, and fourth metatarsals in the dorsoplantar (DP) foot view. Patterns associated with cortical bone thinning in generalized (chronic, long-standing) osteopenia include endosteal resorption, intracortical tunneling, and/or subperiosteal resorption1 (Figure 1).

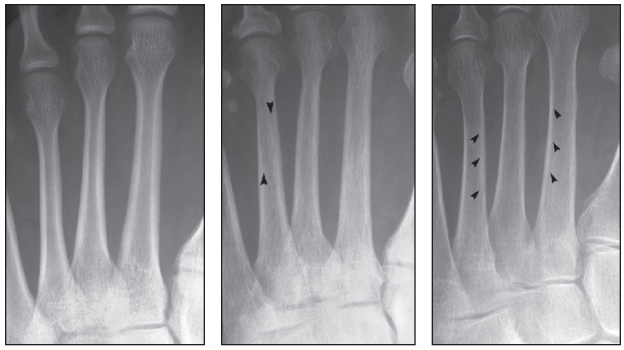

Figure 1: Chronic osteopenia, cortical bone. A, Normally, the endosteal and subperiosteal surfaces of the cortex are well defined and continuous and the remainder of the cortex is radiopaque and homogeneous in density. B, In chronic cortical osteopenia, the endosteal surfaces are ill defined, and lucent striations (intracortical tunneling) may be seen running through the cortex, parallel to the shaft. C, Endosteal resorption, resulting in cortical thinning.

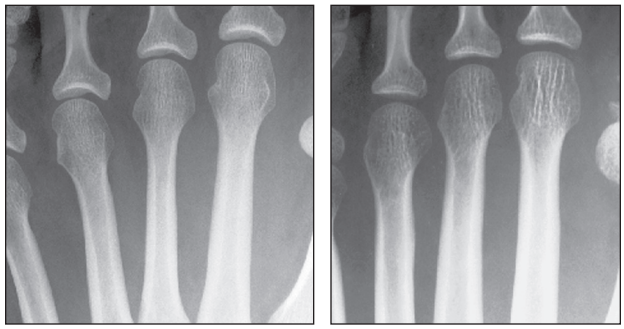

Figure 2: Chronic osteopenia, cancellous bone. A, Cancellous bone is made of primary and secondary trabeculae. The primary trabeculae—also known as stress trabeculae—are found along lines of stress. Secondary trabeculae are found perpendicular or oblique to the primary or stress trabeculae. Secondary trabeculae give spongiosa a fine, homogeneous appearance. B, With chronic osteopenia, secondary trabeculae are resorbed, leaving the primary trabeculae to stand out in relief. As bone is laid down on the remaining primary trabeculae, they become coarse in appearance.

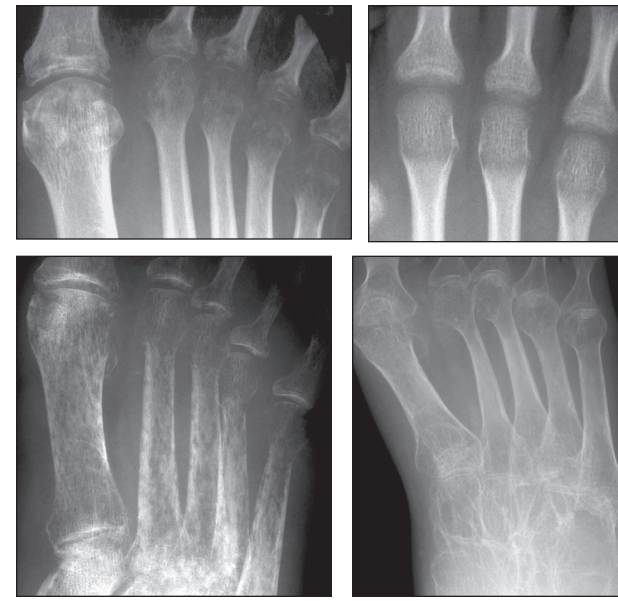

Figure 4: Regional osteoporosis. A, Periarticular, spotty (acute) osteopenia of cancellous bone at metatarsophalangeal joints. B, Metaphyseal ill-defined transverse bands of decreased density, all metatarsals (acute osteopenia). C, Severe acute osteopenia, with spotty, permeative decreased density of cancellous and cortical bone, and subperiosteal resorption. D, Severe osteopenia secondary to polio. Findings parallel those seen in chronic osteopenia (generalized osteoporosis).

A cancellous bone pattern associated with generalized osteopenia is prominent primary trabeculations, which may stand out in relief because secondary trabeculae are resorbed and subsequent bone is laid down on the remaining primary trabeculae (Figure 2). Cortical and cancellous bone resorption is best identified using the DP foot view and concentrating on the second, third, and fourth metatarsals. The medial oblique view should not be used; striations that mimic intracortical tunneling normally are seen at the diametaphyseal region. Caution should be exercised so that these findings are not used to diagnose osteoporosis or other metabolic disease. Chronic osteopenia is commonly associated with systemic disorders, which are primarily diagnosed by other, more characteristic, radiologic features.

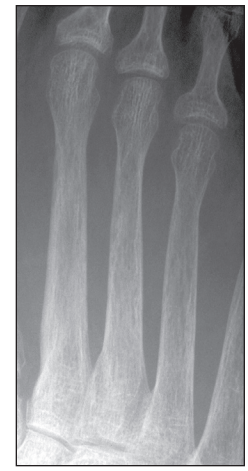

Figure 3: Generalized osteoporosis. This elderly, postmenopausal patient demonstrates the characteristic features of chronic osteopenia, including endosteal resorption, intracortical tunneling, and prominent, coarsened primary trabeculae.

In contrast, the primary radiographic feature of acute osteopenia is spotty (mottled, moth-eaten) loss of bone density, particularly in periarticular regions. Acute osteopenia is associated with causes of regional osteoporosis (discussed next), such as immobilization or disuse.

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Osteoporosis

Osteoporosis is a metabolic disease that accompanies many other disease processes, including most of the endocrine and nutritional disorders discussed later in this article. It is characterized by progressive loss of bone mass and may result in pathologic fracture. Osteoporosis is not easily diagnosed with plain films, because 30% to 50% of bone calcium must be lost before osteopenia is visually apparent. Quantitative methods are necessary to detect early osteoporosis; studies include single-energy x-ray absorptiometry (wrist and heel), radiographic absorptiometry (hand), single-photon absorptiometry (wrist), quantitative computed tomography (spine), and dual-photon absorptiometry (spine, hip, body). It has been suggested that the appearance of calcaneal trabecular patterns provide an index for assessing osteoporosis;[5] however, controversy exists as to whether or not these trabecular patterns correlate with actual bone density.[6] Osteoporosis has been classified according to its etiology: generalized, regional, and localized.[1]

Normal radiographic anatomy may be misinterpreted as osteopenia.

Generalized osteoporosis affects the entire skeleton and is associated with aging (senile osteoporosis), post-menopause, medications (steroids), endocrine states (hyperparathyroidism, diabetes mellitus), deficiency states (scurvy, malnutrition), anemia, and alcoholism, to name a few. The radiographic features are more commonly visualized in the axial skeleton. Typical radiographic findings in the lower extremity (Figure 3) include one or any combination of the following:

Prominent primary trabeculations
Thinning of the cortices
Intracortical striations (tunneling)

Normal radiographic anatomy may be misinterpreted as osteopenia. For example, the trabeculations in the first-metatarsal head and neck are normally prominent and appear coarsened.

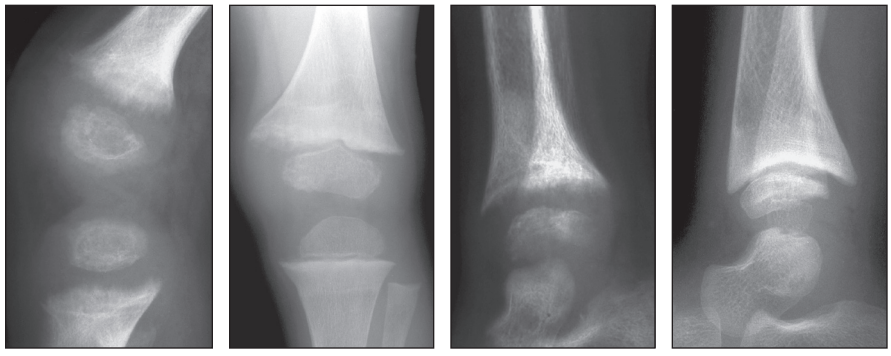

Figure 5: Rickets. A, Knee, lateral view. Characteristic features include an ill-defined, lucent and frayed zone of provisional calcification, with widening of the physis. B, Knee, anteroposterior (AP) view. Following treatment, the zone of provisional calcification has become more defined, although still irregular and ill-defined medially. Rickets. C, Ankle, lateral view. Same date as A; similar findings demonstrated, with additional metaphyseal cupping. D, Ankle, lateral view. Metaphyseal cupping very prominent following treatment; also note defect along surface of talar dome.

Regional osteoporosis affects one extremity. It is associated with disuse (immobilization) and reflex sympathetic dystrophy syndrome (RSD). Typical radiographic findings (Figure 4) include one or any combination of the following:

Spotty (moth-eaten, mottled, patchy) osteopenia in cancellous bone, especially periarticular regions

Ill-defined transverse bands of decreased density at subchondral or metaphyseal locations

Subperiosteal bone resorption (in severe cases, especially RSD)

Long-standing disuse (paralysis is often associated with coarse, prominent primary trabeculations and cortical thinning)

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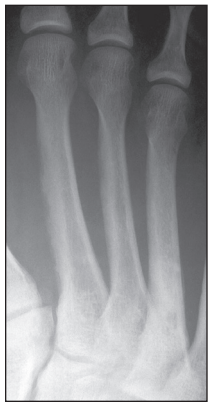

Figure 6: Hyperparathyroidism. Subperiosteal bone resorption along the medial aspects of the metatarsal shafts.

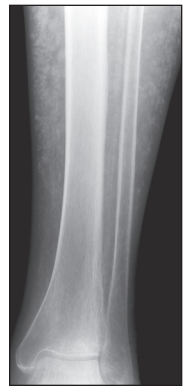

Figure 7: Renal osteodystrophy. Soft tissue calcification throughout the leg.

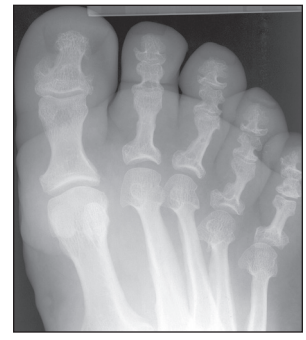

Figure 8: Acromegaly. Characteristic features include enlargement of bones (especially metatarsal heads and shafts and phalangeal bases) and soft tissue thickening (increased soft tissue density and volume).

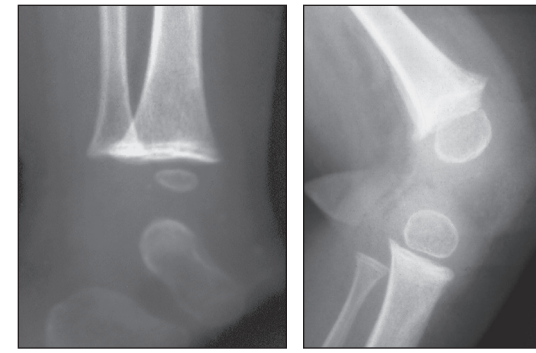

Figure 9: Scurvy. A, Ankle. Note the transverse line of increased density along the metaphysis. B, Knee (different patient). In addition to the sclerotic line across the metaphysis, beaks are visible at the metaphyseal margins, and Wimberger's sign is evident.

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Localized osteoporosis affects one bone. Examples of pathology demonstrating localized osteoporosis include osteomyelitis, arthritis, and neoplasm. The medial aspect of the fifth-metatarsal head is normally radiolucent and should not be mistaken as pathology.

Osteomalacia and Rickets

Osteomalacia and rickets are characterized histologically as disorders with excessive amounts of uncalcified osteoid.[7] Etiologies include vitamin D deficiency and hypophosphatemia. When present in the adult skeleton, osteomalacia is the name given to this condition; in the growing skeleton of infancy and childhood, it is called rickets.

The radiographic features of osteomalacia are non-specific, consisting primarily of osteopenia. Bowing deformity of long tubular bones may occur. Transversely oriented, incomplete radiolucencies, known as pseudofractures or Looser's zones, may be seen in tubular bones.[1]

The radiographic features of rickets consist of both non-specific and characteristic findings (Figure 5). Non-specific findings include general retardation of body growth, osteopenia, and bowing deformity of long tubular bones. Characteristic changes occur at the growth plate region of tubular bones and include widening of the physis, decreased density at the zone of provisional calcification, irregularity of the physeal margin of the zone of provisional calcification (which has been described as "fraying" and a "paint-brush appearance"), and widening and cupping of the metaphysis.[1]

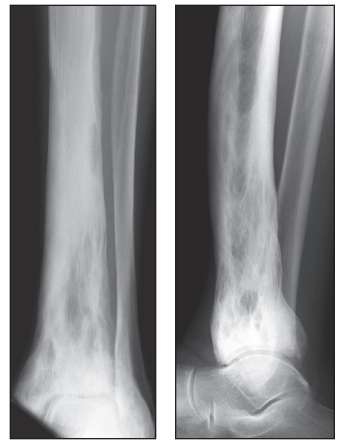

Figure 10: Paget's disease. A, Mortise view; B, lateral view. The bone is predominantly sclerotic in the diaphysis and metaphysis, with mixed well-defined lucent areas intermixed. Probably the third stage. (Accompanying text on following page.)

The characteristic feature of hyperparathyroidism is subperiosteal bone resorption.

Hyperparathyroidism

The general term hyperparathyroidism refers to increased levels of parathyroid hormone.[1] The characteristic feature of hyperparathyroidism is subperiosteal bone resorption (Figure 6). Other sites of bone resorption include periarticular, intracortical, endosteal, subchondral, and at entheses.

Soft tissue calcification and geographic, lucent lesions known as Brown tumors may also be seen.

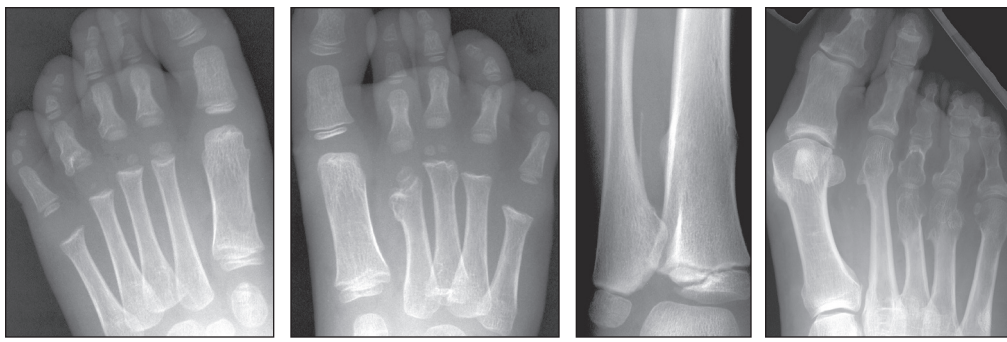

Figure 11: Hereditary multiple exostoses. A, DP view, left foot. Small exostoses are becoming visible along the medial and lateral aspects of the first-metatarsal distal metadiaphysis and along the lateral aspects of the third- and fourth-toe proximal phalangeal bases. B, Same patient, DP view, right foot. Significant involvement of the second and third metatarsals, with shortening of the second. Hereditary multiple exostoses. C, Same patient, AP view, ankle. Exostoses are seen along the medial aspect of the distal fibular diaphysis and the medial and lateral aspects of the tibial diaphysis. D, Different patient, adult. Involvement of the second through fourth metatarsals and third-toe proximal phalanx.

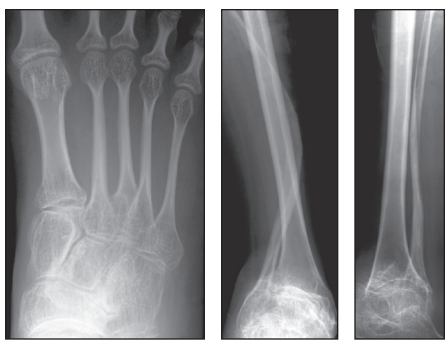

Figure 12: Osteogenesis imperfecta. A, DP view, foot. Characteristic features include narrowed girth of tubular bones and cancellous osteopenia. B-D, Different patient demonstrating severe osteoporosis and severe deformity of the fibula. Osteogenesis imperfecta. A, DP view, foot. Characteristic features include narrowed girth of tubular bones and cancellous osteopenia. B-D, Different patient demonstrating severe osteoporosis and severe deformity of the fibula.

Renal Osteodystrophy

Patients with chronic renal failure demonstrate bony abnormalities known as renal osteodystrophy.[1] The radiographic findings manifested may parallel those of hyperparathyroidism, osteoporosis, and osteomalacia (or rickets if in the child). Calcification of soft tissue and vessels is a frequent finding (Figure 7).

Acromegaly

Excess pituitary somatotrophic growth hormone results in abnormal growth of bone, cartilage, and fibrous tis-

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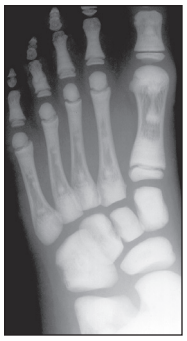
sue.[7] In the adult, this is known as acromegaly; gigantism results in the child with open growth plates. Radiographically (Figure 8), the patient with acromegaly has an increase in skin volume; the heel pad thickness is greater than 25 mm in the male and 23 mm in the female (when local causes are excluded). The joint spaces are widened by cartilage thickening. Bones become prominent; the metatarsal heads and distal phalanx ungual tuberosities are enlarged, metatarsal shafts are thickened, and spurs are found at entheses. Interestingly, however, the proximal phalangeal shafts appear narrow in girth.



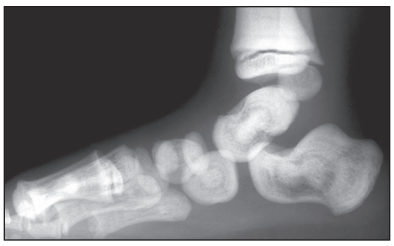


Figure 13: Osteopetrosis. A, DP view, 7-year-old; B, Lateral view, same patient at 3 years of age. Classic bone-within-bone appearance.

Hypovitaminosis C (Scurvy)

Scurvy is caused by insufficient dietary intake of vitamin C.[8] The characteristic radiographic features (Figure 9) occur in the developing child. In the metaphyseal region they include a transverse line of increased density, a transverse line of decreased density adjacent to this line of increased density (the scurvy line), and a small, beaklike outgrowth of bone along the margins of the metaphysis. Extensive periostitis may be seen along the entire length of the bone. The epiphysis appears as an outer shell of increased density surrounding a central lucency (Wimberger's sign).[1]

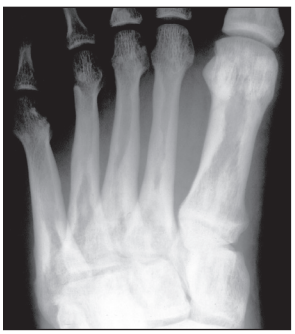


Figure 14: Melorheostosis. The wavy, thickened endosteal cortical surface simulates wax flowing down a candle. This patient also has osteopoikilosis, as demonstrated by the circular increased densities in periarticular regions.

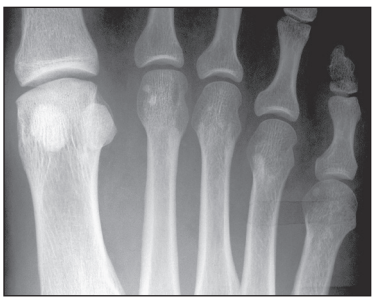


Figure 15: Osteopoikilosis. Multiple islands of cortical bone density are identified in the lesser-metatarsal heads.

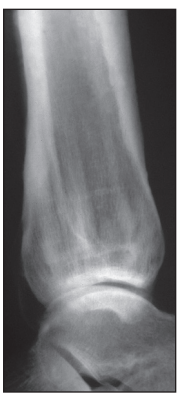


Figure 16: Osteopathia striata. Lateral view, ankle. Mixed linear bands of increased and decreased density run through the metadiaphyseal region of the distal tibia, parallel to the bone's long axis.

Paget's Disease (Osteitis Deformans)

Paget's disease (Figure 10) is characterized by excessive and abnormal remodeling of bone.[1] Its etiology is unknown, and occurs more frequently in people who are 40 years and older. Paget's disease is typically asymptomatic in the foot, which may explain the infrequent reports at this location in the literature.[9] Four radiographic stages have been recognized. The first stage is described as osteolytic. It starts in the subchondral area, spreading to the metaphysis and eventually the diaphysis resulting in what has been described as a "blade of grass" appearance. The second stage demonstrates both osteolysis and osteosclerosis. The diaphysis appears lucent, whereas the epiphyseal and metaphyseal regions are sclerotic. Bone in the third stage is predominantly sclerotic. Findings include cortical thickening, bone enlargement, and coarsened trabeculae. The fourth phase is malignant degeneration, reported in 1% to 10% of patients.[8] Other complications associated with Paget's disease are fracture, osteomyelitis, and joint disease.[1]

The radiographic finding described as a "blade of grass" appearance is associated with Paget's disease.

Hereditary Multiple Exostoses (Osteochondromatosis)

Hereditary multiple exostoses is a skeletal dysplasia resulting from a disturbance of chondroid production resulting in heterotopic proliferation of epiphyseal chondroblasts.[10] The individual lesions appear identical to the bone tumor osteochondroma, that is, multiple cartilage-capped exostoses adjacent to the diaphyseal side of the physis (Figure 11). This condition is autosomal dominant and appears in the first two decades of life as painless bumps near the ends of long bones. Its distribution is frequently symmetric, and it can cause bone deformity and shortening. Approximately 5 percent of these lesions transform into chondrosarcoma.[7]

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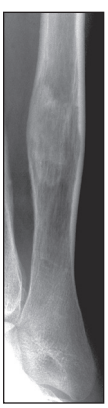

Figure 17: Fibrous dysplasia. The geographic lesion in the distal fibular diaphysis demonstrates a hazy, ground glass appearance centrally. Notice its intramedullary location.

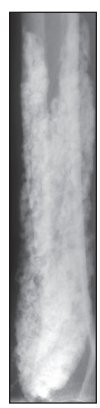

Figure 18: Metastatic calcification: secondary hyperparathyroidism. The spectrum of radiographic abnormality may be seen in these patients. This example demonstrates significant soft tissue calcification in the distal leg.

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Osteogenesis Imperfecta

An inherited skeletal dysplasia that is the result of abnormal metaphyseal and periosteal ossification caused by deficient osteoid production is osteogenesis imperfecta.[10] Abnormal maturation of collagen occurs in both mineralized and non-mineralized tissues. Radiographic features include diffuse osteopenia (occasionally coarse trabeculae and a honeycomb appearance), diminished bone girth, and flared metaphyses (Figure 12). An obvious complication is fracture.

The Sclerosing Dysplasias

Four skeletal dysplasias are the result of abnormal metaphyseal and periosteal ossification caused by either excessive osteoid production or deficient osteolysis. The primary radiographic feature is increased bone density. They include osteopetrosis, melorheostosis, osteopoikilosis, and osteopathia striata.

Osteopetrosis (Albers-Schönberg disease) is an inherited, autosomal dominant (delayed type) condition presenting radiographically with diffuse bone sclerosis (Figure 13). Its characteristic feature has been described as a "bone within a bone" appearance.[11] These patients are relatively aymptomatic.[1]

Melorheostosis is a rare dysplasia that is not believed to be hereditary.[1] the patient may or may not have associated symptomatology.[9] The radiographic presentation is typically limited to a single limb and consists of hyperostosis along the bone's periphery, extending along its entire length in many cases (Figure 14). This picture simulates wax flowing down the side of a candle and has a wavy, sclerotic bony contour usually along the bone's endosteal surface.

Figure 21: Vascular calcification: phleboliths. A, Phleboliths may be solitary or multiple, as demonstrated in this example of the distal leg. B, Numerous phleboliths are seen throughout the calf in this patient with chronic venous insufficiency.

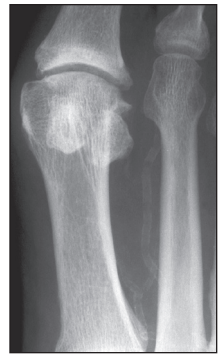

Figure 22: Vascular calcification: Moenckeberg's sclerosis. The classic presentation is shown in this diabetic patient (DP view). Tubular, serpiginous calcifications are easily identified in the first intermetatarsal space following the course of arterial vessels.

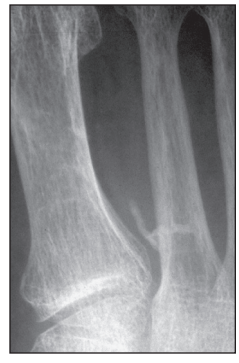

Figure 23: Vascular calcification: atherosclerosis. In contrast to Moenckeberg's sclerosis, the calcifications that follow the course of arterial vessels are patchy and solid.

Osteopoikilosis is an inherited skeletal dysplasia that is not associated with symptomatology. Numerous small, well-defined and homogeneous circular foci of increased density are seen radiographically, mimicking multiple bone islands (Figure 15). The distribution of these lesions is periarticular and symmetric, found at the ends of long bones.[1] They may also be found in tarsal bones.

Osteopathia striata is most likely inherited and is typically asymptomatic.[9] Radiographically, linear, regular bands of increased density extend from the metaphysis to the diaphysis, running parallel to the shaft (Figure 16).

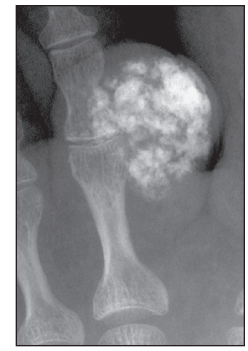

Figure 19: Generalized calcinosis: tumoral calcinosis. A large periarticular mass demonstrates calcification adjacent to the proximal interphalangeal joint of a toe.

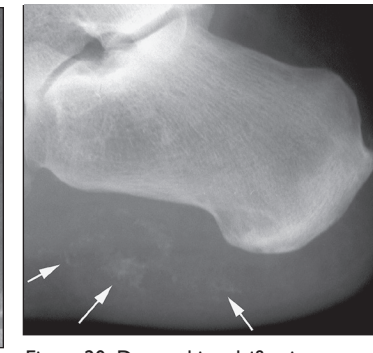

Figure 20: Dystrophic calcification: trauma. This patient related a recent injury to the heel. Multiple areas of ill-defined soft tissue calcification are identified (arrows).

Osteopoikilosis is characterized by numerous small, well-defined and homogeneous circular foci of increased density.

Fibrous Dysplasia

Pathologically, normal bone undergoing physiologic resorption is replaced with fibrous tissue.[12] It may occur in one (monostotic) or many (polyostotic) bones. In tubular bones its location is intramedullary and diaphyseal, with only occasional epiphyseal involvement.[1] Radiographically the lesion is somewhat radiolucent with a hazy, ground glass (homogeneous) quality, and may appear expansile with endosteal scalloping (Figure 17).

Soft Tissue Manifestations of Systemic isorders

The primary radiographic soft tissue findings are calcification and ossification. Calcification appears as irregular punctate, circular, linear, or plaque-like radiodense areas with no cortical or trabecular structure.[1] Ossification, in contrast, demonstrates a trabecular pattern and a thin, cortex-like periphery. However, if the lesion is small, the distinction may be impossible to determine visually.

Calcification

Conditions that lead to soft tissue calcification have been categorized as metastatic calcification, generalized

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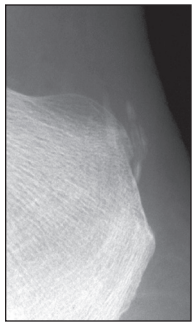
Radiology *(from page 134)*

calcinosis, and dystrophic calcification.[1] Metastatic calcification (Figure 18) results from a disturbance in calcium or phosphorus metabolism. Examples include hyperparathyroidism, hypoparathyroidism, renal osteodystrophy, hypervitaminosis D, and sarcoidosis. Generalized calcinosis (Figure 19) presents as calcium deposition in the skin and subcutaneous tissue in the presence of normal calcium metabolism. Examples include collagen vascular disorders (scleroderma, dermatomyositis), idiopathic tumoral calcinosis, and idiopathic calcinosis universalis.

Tumoral calcinosis is rare in the foot but has been reported adjacent to a sesamoid[13] and a first-metatarsal-head medial eminence,[14] at the tip of digits,[15] and posterior to the ankle joint. With dystrophic calcification (Figure 20), calcium is deposited in damaged or devitalized tissue in the absence of a generalized metabolic derangement. Examples include neoplasm, inflammation, and trauma. Soft tissue calcification has been associated with local corticosteroid injection of the heel[16] and intra-articular corticosteroid injection of the small joints of the hand.[17] Extensive involvement of the foot suggests sarcoma.[18]



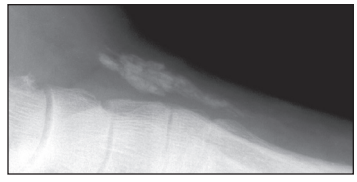
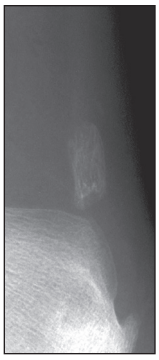


Figure 24: Calcific tendonitis. A, Achilles tendon calcification near its enthesis. B, Calcification of extensor tendons along the dorsum of the midfoot.

Vascular calcifications are frequently encountered in the foot and leg. Phleboliths, found in veins, are circular or elliptical in shape (Figure 21); they possess a thin calcific outline with a relatively lucent center. Phleboliths may be a solitary finding or multiple. Phleboliths have also been described associated with hemangioma of the foot.[19] Two types of arterial calcification have been described: Moenckeberg's sclerosis and atherosclerosis. Moenckeberg's sclerosis (Mönckeberg's arteriosclerosis, medial calcific sclerosis) is calcification of the vessel's tunica media layer. These lesions generally do not obstruct blood flow through the vessel. They are commonly seen in patients with diabetes.



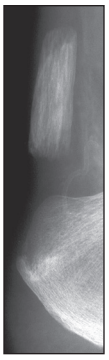


Figure 25: Soft tissue ossification: Achilles tendon. A, Notice the trabeculations in the lesion, which distinguish it from calcification. B, A much larger ossification; this image also demonstrates Moenckeberg's sclerosis.

Radiographically, Moenckeberg's sclerosis has a characteristic appearance consisting of dual, curvilinear tubular calcifications running parallel to one another (Figure 22). They may be serpiginous and follow the anatomic course of the vessel. Not uncommonly the path of calcification is discontinuous. The term atherosclerosis refers to plaque formation along the tunica intima layer of the vessel. Progressive calcification at any particular site can lead to obstruction of the vessel. Radiographically, one notes patchy calcifications in the soft tissues that follow the path of vessels (Figure 23).

The site of soft tissue calcification may provide a clue to the underlying systemic disorder.[1] Periarticular calcification is associated with hyperparathyroidism, hypervitaminosis D, and collagen vascular disease. Calcifications of tendons and bursae are associated with calcium pyrophosphate dihydrate (CPPD) and calcium hydroxyapatite crystal deposition disease. Arterial calcification is frequently seen with diabetes mellitus, hypervitaminosis D, and renal osteodystrophy. Calcification of cartilage (chondrocalcinosis) suggests CPPD deposition and hemochromatosis. Sheet-like collections in the lower leg are associated with tissue injury and compartment syndrome.

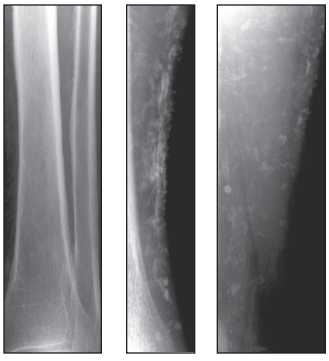
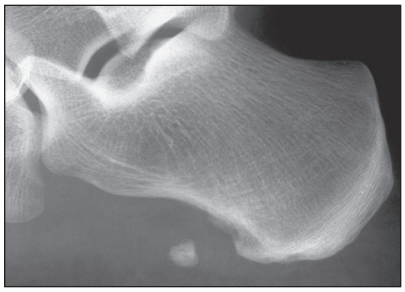


Figure 26: Generalized periostitis: venous stasis. A, Irregular, wavy periostitis is identified along the lateral and medial margins of the distal tibia and fibular diaphysis, respectively. B, This patient, with chronic venous insufficiency (AP view), demonstrates solid periostitis along the distal tibial diaphysis with diffuse soft tissue calcification (phleboliths).

The site of soft tissue calcification may provide a clue to the underlying systemic disorder.[1]



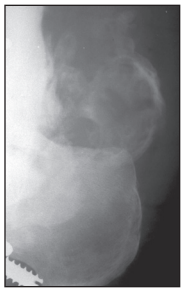


Figure 27: Heterotopic soft tissue ossification. A, Myositis ossificans circumscripta. A lesion identified in the plantar fascia musculature. B, Myositis ossificans associated with neuropathic osteoarthropathy, posterior ankle.

The term calcific tendinitis (Figure 24) has been used to describe tendon calcification resulting from the deposition of calcium hydroxyapatite crystals.[20] Its etiology is closely associated with trauma, but inju-

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Radiology (from page 135)

ry is not prerequisite. Pain is the most common associated symptom. Holt and Keats illustrate examples involving the Achilles, peroneus, and forefoot flexor tendons.[20] Similar reports include examples involving the peroneal tendon,[21,22] although they are not identified specifically as calcific tendinitis. Radiographic evidence of adjacent bone erosion, mimicking malignancy, has been reported.[23]

The term myositis ossificans is used to describe non-neoplastic heterotopic soft tissue calcification.

Ossification

Causes of soft tissue ossification include neoplasm (sarcoma), trauma (myositis ossificans, ossifying hematoma), and venous insufficiency.

Achilles tendon ossification (Figure 25) is frequently associated with pain.[24-26] Interestingly, MRI analysis of several cases failed to demonstrate inflammatory changes, even with acute symptoms.[27] Morris and associates have presented a classification of Achilles tendon lesion based on anatomic location, including calcification and ossification.[28]

Generalized periostitis is associated with venous stasis (Figure 26), hypertrophic osteoarthropathy (primary and secondary), thyroid acropachy, and hypervitaminosis A.[1,11] Radiographically, varying degrees of periosteal new bone formation is possible, but typically it is thick and undulating (irregular).

The term myositis ossificans is used to describe non-neoplastic heterotopic soft tissue calcification. Four categories are described in the literature, relating to underlying etiology (or lack of):

1) Myositis ossificans progressiva is a rare genetic dysplasia with associated congenital osseous abnormalities.[7]

2) Myositis ossificans circumscripta, caused by localized trauma, is the most common form (Figure 27A). Several cases in the foot have been described.[29-32]

3) Myositis ossificans associated with neurologic disease (Figure 27B) may be due to a variety of neurologic causes.[7]

4) Pseudomalignant myositis ossificans is similar to the circumscripta form but without a history of antecedent trauma.[33]

Yochum and Rowe[34] warn that the early stages of myositis ossificans circumscripta may mimic sarcoma. It initially presents as a soft tissue swelling that proceeds to an ill-defined calcific density followed by ossification. A radiolucent zone may be seen between the lesion and adjacent bone, which helps to distinguish it from sarcomatous soft tissue extension. **PM**

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Continued on page 137

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CME EXAMINATION

SEE ANSWER SHEET ON PAGE 139.

1) Osseous resorption in cortical bone may appear as:

A) band-like/transverse radiolucent areas in the metaphysis.
B) subchondral spotty radiolucent areas.
C) intracortical tunneling.
D) diffuse, homogeneous "coarsening" of trabeculae.

2) A major cause of diffuse osteopenia is:

A) osteoporosis.
B) osteoarthritis.
C) osteosarcoma.
D) osteopoikilosis.

3) Generalized subperiosteal bone resorption is characteristic of:

A) pseudohypoparathyroidism.
B) hyperparathyroidism.
C) fluorosis.
D) hypervitaminosis A.

4) Generalized soft tissue thickening, increased girth of metatarsals, decreased girth of proximal phalanges, and joint space widening are best associated with:

A) hyperparathyroidism.
B) pseudohypoparathyroidism.
C) acromegaly.
D) hypothyroidism.

5) Short metatarsals or phalanges are best associated with:

A) hypothyroidism.
B) pseudohypoparathyroidism.
C) hyperparathyroidism.
D) hypervitaminosis A.

Continued on page 137

6) The following is NOT one of the three classes of osteoporosis:
- A) generalized.
- B) regional.
- C) localized.
- D) field.

7) A radiographic finding seen with reflex sympathetic dystrophy is:
- A) spotty loss of bone density.
- B) intracortical tunneling.
- C) coarse trabeculations.
- D) cortical thinning.

8) A major cause of regional osteoporosis in the lower extremity is:
- A) swimming.
- B) running.
- C) walking.
- D) immobilization.

9) A radiographic feature of rickets is:
- A) straight, narrow tubular bone.
- B) sclerotic epiphysis.
- C) narrowing of the metaphysis.
- D) irregular outline or zone of provisional calcification.

10) Hypovitaminosis C is also known as:
- A) rickets.
- B) scurvy.
- C) lead poisoning.
- D) hypervitaminosis D.

SEE ANSWER SHEET ON PAGE 139.

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Radiology of Systemic Disorders Affecting the Foot and Ankle (Christman)

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2.	A	B	C	D	7.	A	B	C	D
3.	A	B	C	D	8.	A	B	C	D
4.	A	B	C	D	9.	A	B	C	D
5.	A	B	C	D	10.	A	B	C	D

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