Osteonecrosis in the Foot

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Abstract

Osteonecrosis, also referred to as avascular necrosis, refers to the death of cells within bone caused by a lack of circulation. It has been documented in bones throughout the body. In the foot, osteonecrosis is most commonly seen in the talus, the first and second metatarsals, and the navicular. Although uncommon, osteonecrosis has been documented in almost every bone of the foot and therefore should be considered in the differential diagnosis when evaluating both adult and pediatric foot pain. Osteonecrosis is associated with many foot problems, including fractures of the talar neck and navicular as well as Kohler's disease and Freiberg's disease. Orthopaedists who manage foot disorders will at some point likely be faced with the challenges associated with patients with osteonecrosis of the foot. Because this disease can masquerade as many other pathologies, physicians should be aware of the etiology, presentation, and treatment options for osteonecrosis in the foot.

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Osteonecrosis, also called avascular necrosis, refers to cellular death within bone caused by a lack of circulation. This circulatory compromise may arise from a mechanical disruption of the vessels or from occlusion of either arterial inflow or venous outflow. Potential sources of occlusion include thrombosis, embolism, circulating fat, and abnormally shaped cells (eg, sickle cells).[1] Corticosteroid use, alcohol intake, and Gaucher's disease produce osteonecrosis by increasing intramedullary pressure, thereby decreasing perfusion.[2-6] Trauma remains the most common mechanical cause of osteonecrosis throughout the body, particularly in the foot.[7]

Following circulatory compromise, few histologic changes are seen within the first week. In the second week, the death of hematopoietic cells, capillary endothelial cells, and lipocytes can be confirmed microscopically. The lipocytes release lysosomes that acidify the tissue, the osteocytes begin to shrink, and the water content in the bone increases. These changes represent the first abnormalities detectable on magnetic resonance imaging (MRI). Regardless of the cause of osteonecrosis, the final radiographic finding remains the same: a resultant relative increase in the radiodensity of the bone. This change is caused by bony collapse, saponification of fat, creeping substitution, and a relative density difference between the avascular and surrounding vascularized bone. Without the ability to repair itself, such dysvascular bone eventually collapses, appearing fragmented and sclerotic. This process increases with additive microtrauma.[1]

The radiographic modalities used to diagnose osteonecrosis continue to advance. Conventional radiographs are useful for diagnosis only

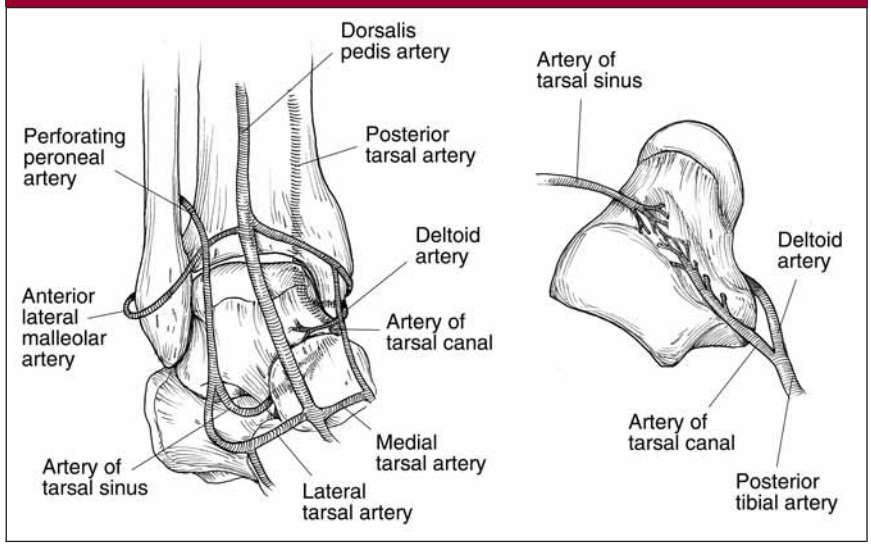
after the development of sclerosis, articular collapse, or a crescent sign. The relative usefulness of bone scintigraphy is dependent on the stage of osteonecrosis. During the acute ischemic phase, the entire affected area may demonstrate high uptake from increased osteoblastic activity. In later stages, however, a demarcated rim of increased activity surrounding the hypoperfused necrotic core will become apparent. Single-photon emission computed tomography (SPECT) can aid in maximizing resolution. Current MRI techniques are sensitive to changes in bone marrow composition. Diffuse marrow edema is seen in early osteonecrosis and produces a low signal intensity on T1-weighted images and a high signal intensity on T2-weighted images. In advanced stages of the disease, both T1- and T2-weighted images demonstrate low signal intensity.[7] Although positron emission tomography scanning has been used to determine blood flow to the femoral head with suspected osteonecrosis, its use for detecting this disease is still not well-defined.[8]

Although a relatively uncommon diagnosis, osteonecrosis has been identified in almost every bone in the foot and has been extensively studied in the talus, the first and second metatarsals, and the navicular.[7] Because this disease can masquerade as many other pathologies, it is mandatory that the orthopaedist understand the etiology and management of osteonecrosis. Appropriate recognition and treatment can affect outcome; thus, osteonecrosis must be considered when evaluating foot pain in any population.

Osteonecrosis in the Hindfoot: The Talus

The talus is commonly affected by osteonecrosis. Although atraumatic causes have been estimated to account for up to 25% of talar osteonecrosis, the etiology usually can be traced back to an injury.[9]

Figure 1



Extraosseous vascular supply to the talus. (Reproduced from Fortin P, Balaszy J: Talus fractures: Evaluation and treatment. *J Am Acad Orthop Surg* 2001;9:114-127.)

Vascular Supply

The talus has no tendinous attachments or muscular origins; thus, it relies on direct vasculature for its blood supply. Furthermore, its lack of vascular redundancy (more than half of this bone is covered with articular cartilage) makes each vascular contribution vitally important.

All three primary arteries of the foot support the extraosseous supply[10-12] (Figure 1). The posterior tibial artery is the principal blood supply for the talar body via the artery of the tarsal canal and the deltoid artery. The artery of the tarsal canal passes between the sheaths of the flexor digitorum longus and the flexor hallucis longus to enter the tarsal canal and supply most of the talar body. The deltoid artery arises near the origin of the artery of the tarsal canal and enters the talar body medially.

The artery of the tarsal sinus is formed from branches of the dorsalis pedis and perforating peroneal arteries and merges with the artery of the tarsal canal. Together they feed most of the talar neck and head.[11]

Two smaller contributors are the peroneal artery, which supplies the posterior process via direct branches, and the superomedial branches of the dorsalis pedis, which supply the talar head.

Incidence and Etiology

Osteonecrosis of the talus commonly follows talar neck fractures, which typically are the result of forced dorsiflexion caused by high-energy trauma.[10] Hawkins[13] developed a commonly used classification system that stratifies the future risk of osteonecrosis based on fracture displacement and joint congruency. Type I talar neck fractures are nondisplaced and have an approximately 10% chance of developing osteonecrosis. Type II fractures are displaced and have an associated disruption of the subtalar articulation, carrying an approximate 40% risk of osteonecrosis. Type III fractures are also displaced, with incongruity of both the ankle and subtalar joints. Type III fractures have an approximate 90% incidence of osteonecrosis.

Canale and Kelly[14] further refined the classification, adding a fourth type, which signifies extrusion of

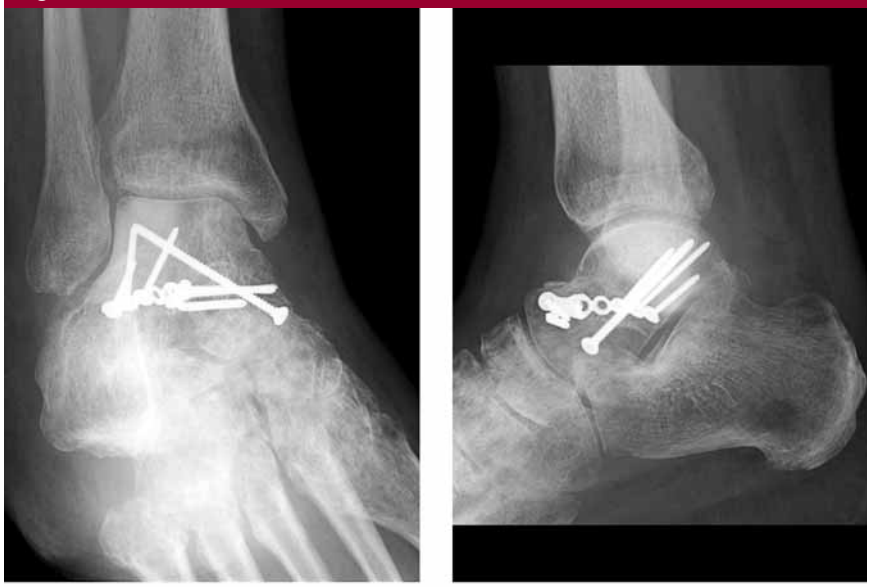
Table 1

Ficat and Arlet Radiographic Appearance of Osteonecrosis of the Talus

Stage	Radiographic Appearance
I	Normal
II	Cystic and sclerotic lesions, normal contour of the talus
III	Crescent sign, subchondral collapse
IV	Narrowing of the joint space, secondary changes in the tibia

Reproduced with permission from Delanois RE, Mont MA, Yoon TR, Mizell M, Hungerford DS: Atraumatic osteonecrosis of the talus. *J Bone Joint Surg Am* 1998;80:529-536.

Figure 2



A **B**

Anteroposterior **(A)** and lateral **(B)** radiographs of a displaced talar neck fracture treated with surgical fixation. These radiographs, taken 12 weeks after injury, demonstrate bony sclerosis of the lateral aspect of the talar body, which is indicative of osteonecrosis. (Courtesy of C.W. DiGiovanni, MD, Providence, RI.)

the talar body and subluxation of the talonavicular joint. Such injury virtually guarantees osteonecrosis. Because the blood supply to the talus has a significant retrograde component from head to body, most patients with talar neck fracture exhibit osteonecrosis in the talar body.

The risk of osteonecrosis increases as fractures of the talar body become more displaced and comminuted.[15] In general, talar body fractures do not differ significantly from talar neck fractures in terms of the development of osteonecrosis.[16] Talar head fractures signify high-energy talar injury. These fractures are associated with osteonecrosis as a result of concomitant fractures of the neck and body.

Less common atraumatic etiologies of talar osteonecrosis include alcoholism,[17] sickle cell disease,[18] corticosteroid use, dialysis,[19] hemophilia,[20] hyperuricemia,[21] and lymphoma.[22] Delanois et al[23] documented a series of cases of atraumatic osteonecrosis that primarily affected the posterolateral talar dome. Most of these patients, however, had a history of corticosteroid use.

Diagnosis and Treatment

The clinical presentation of talar osteonecrosis is primarily determined by the integrity of the articular surface. Before articular collapse, the patient may be asymptomatic. The pain and mechanical symptoms associated with increasing articular incongruity typically represent the primary complaints. Imaging should begin with plain radiography, which is notable for early findings of sclerosis, cystic changes, and advanced changes of collapse. An adaptation of the Ficat and Arlet classification characterizes the extent of talar disease[23] (Table 1). MRI further illustrates the extent of disease and can be used to further stage osteonecrosis of the talus.[24]

The traditional approach for managing displaced talus fracture has been timely, rigid anatomic reduction and internal fixation, with the intent of minimizing vascular insult, stiffness, posttraumatic arthritis, and osteonecrosis. The optimal timing of fracture management, however, remains poorly defined. Recent data suggest that treatment can be safely delayed in the absence of frank joint dislocation, open wounds, or neurovascular compromise.[25]

Most cases of posttraumatic osteonecrosis manifest within the first 10 months after injury, although the disease can take longer than 2 years to see on imaging studies.[25] Subchondral lucency (ie, Hawkins sign) seen at 6 weeks after injury is reliable evidence of revascularization.[14] In contradistinction, a sclerotic, radiodense talar body suggests osteonecrosis[23] (Figure 2; Table 1).

The treatment algorithm is dictated by clinical symptoms. Most protocols incorporate limited weight bearing and activity modification.[9] However, there is no consensus on

either the duration or degree of restricted weight bearing, or on the utility of bracing or immobilization, in minimizing the sequelae of osteonecrosis. Nonsurgical therapy often is sufficient to meet patient functional expectations. When nonsurgical management fails, however, the patient can be treated with a variety of surgical interventions.

Arthroscopy is useful in evaluating joint surfaces and removing loose bodies. More aggressive surgical intervention depends on the stage and extent of disease. Procedures range from open and arthroscopic methods of core decompression to ankle, subtalar, and pantalar arthrodesis with vascularized bone grafting.[26-28] Fusion must be approached cautiously in the patient with osteonecrosis because of the possibility of serious sequelae. Ankle arthroplasty, although controversial in the setting of osteonecrosis, may offer an alternative to fusion. Currently, however, there are no long-term results available for ankle arthroplasty in these often young patients. There are anecdotal reports of satisfactory functional results with salvage using a stainless steel talar body prosthesis.[29]

Delanois et al[23] presented their experience with 37 ankles with atraumatic talar osteonecrosis. Most ankles presented in Ficat stage II and were initially treated nonsurgically with an ankle-foot orthosis, nonsteroidal anti-inflammatory drugs, and partial weight bearing. Thirty-two of the ankles proceeded to core decompression, and three were treated with fusion. There was a correlation between disease severity at presentation with disease progression and the rate of ultimate surgical fusion.[23]

Prognosis

There are few outcome studies regarding traumatic talar osteonecrosis. In two small series, nonsurgical management yielded satisfactory results in 38% to 46% of patients.[30] Canale and Kelly[14] reported satisfactory results in all patients who were kept non–weight bearing for an average of 8 months. Interestingly, there was no clear correlation between the amount of talar dome involvement and symptom severity. Published results indicate that tibiotalar and tibiotalocalcaneal fusion can be reliably performed in the presence of osteonecrosis.[9,31]

Outcome data of atraumatic talar osteonecrosis is even more limited. In the series of Delanois et al,[23] 29 of 32 ankles treated with decompression had fair to excellent outcomes at a mean 7-year follow-up. Follow-up on the arthrodeses was limited, but promising. At mean 7 months postoperatively, all six ankles managed with arthrodesis had fused.

Osteonecrosis in the Midfoot: The Navicular

Navicular osteonecrosis can be either idiopathic or traumatic. Fractures of the navicular, which are often high-energy injuries, represent the most common etiology of osteonecrosis.[32] Kohler's disease and Müller-Weiss disease are manifestations of idiopathic osteonecrosis in the pediatric and adult populations, respectively.

Vascular Supply

The navicular vascular supply is precarious. The dorsalis pedis or one of its tributaries provides several perforating branches. The plantar surface is fed primarily through branches from the medial plantar artery. The intraosseous blood flow is centripetal and has a central watershed area, which places the navicular at significant risk of osteonecrosis in the presence of obstructed peripheral blood flow.[32]

Kohler's Disease

Kohler first described childhood navicular osteonecrosis at the turn of the 20th century; Williams and Cowell[33] subsequently described its natural history. Patients typically present between ages 2 and 9 years with the primary complaint of midfoot pain. Navicular sclerosis, fragmentation, and flattening are noted radiographically. However, these changes may represent a normal variant in asymptomatic patients; thus, the diagnosis of osteonecrosis must be corroborated by clinical suspicion and supportive radiographs.

Treatment and Prognosis

In the Williams and Cowell series,[33] all patients were asymptomatic and, with nonsurgical management, had a radiographically normal navicular at 9-year follow-up. Casting provided earlier resolution of symptoms. Weight-bearing status did not affect outcome. These results agree with those of the study by Ippolito et al,[34] which followed patients for more than 30 years.

Müller-Weiss Disease

Adult-onset navicular osteonecrosis, or Müller-Weiss disease (also known as Brailsford's disease[35]), was first described in the 1920s. The proposed etiology of this condition includes trauma as well as a delay in navicular ossification that results in an abnormal osseous product.[36] Pain that begins insidiously in the fifth decade is the most consistent clinical finding of Müller-Weiss disease. Heel varus is a hallmark of the disease that, when associated with pes planus, results in a paradoxical pes planovarus. Navicular sclerosis and fragmentation are noted radiographically. The talar head often points laterally, and changes in the orientation of the remainder of the forefoot bones are observed[36] (Figure 3; Table 2).

The anteroposterior view of the foot gives the clearest view of navicular pathology. In severe cases, the talus appears to contact the cuneiforms directly. These findings may mimic the appearance of tarsal coalition, Charcot neuropathy, or an accessory navicular. Surgical options include internal fixation of the na-

Figure 3

Graphic representation demonstrating the increasing grades of navicular deformity in Müller-Weiss disease. The lateral aspect of the navicular collapses, causing the talar head to move laterally and appear to contact the cuneiforms themselves. (Adapted with permission from Maceira E, Rochera R: Müller-Weiss disease: Clinical and biomechanical features. *Foot Ankle Clin* 2004;9:105-125.)

Table 2

Radiographic Appearance of Osteonecrosis of the Navicular[36]

Stage	Description
I	Normal radiographs Positive technetium Tc-99m scan, computed tomography, and magnetic resonance imaging
II	Subtalar varus
III	Navicular compression
IV	Hindfoot equinus
V	Complete extrusion of the navicular

vicular, triple arthrodesis, talonavicular arthrodesis, and talonavicular–cuneiform bone block arthrodesis. Outcomes with talonavicular–cuneiform bone block arthrodesis have been promising at limited follow-up.[37]

Traumatic Osteonecrosis of the Navicular

The increased incidence of navicular fractures may be the result of advances in automobile safety that protect occupants' vital structures but leave their feet unprotected.[38] Navicular injuries range in severity from small avulsions to highly comminuted fractures. Sangeorzan classified navicular body fractures into three main types.[32] Type 1 fractures divide the bone into an anterior and a posterior fragment. Type 2 fractures, the most common, propagate in a direction from dorsolateral to plantarmedial. Type 3 fractures produce comminution in the middle and lateral navicular, with disruption of the naviculocuneiform joint.

Type 1 and 3 fractures are most commonly associated with navicular necrosis, which presumably is caused either by soft-tissue stripping resulting from displacement or by surgical exposure. Anatomic reduction and fixation is recommended for displaced and comminuted fractures.[39] In the series of Sangeorzan et al,[32] 67% of the 21 patients with navicular fractures who were surgically treated had good results. Of the patients with a good result, osteonecrosis developed in six. Most of the patients with partial necrosis of the navicular still had good results. Anatomic fracture reduction and stabilization done while taking care to minimize iatrogenic vascular insult is the most effective means of preventing navicular osteonecrosis. Late treatment of symptomatic collapse to eliminate painful motion and maintain medial column length is done with either bone-block midfoot fusion or triple arthrodesis.

Common Locations for Osteonecrosis in the Forefoot

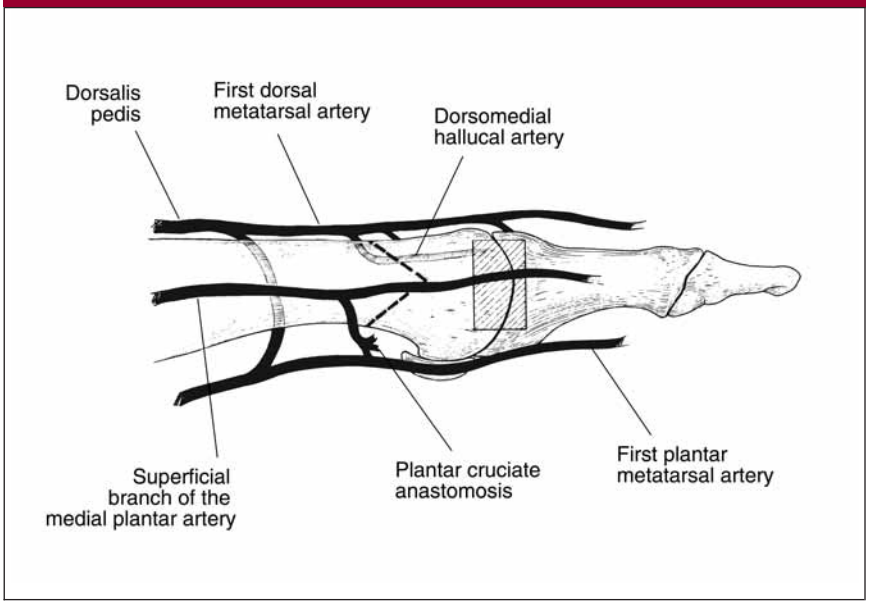
First Metatarsal

First metatarsal osteonecrosis, which primarily affects the head, occurs most commonly after hallux valgus correction. The etiology of this iatrogenic first metatarsal osteonecrosis is thought to be related to the amount of soft-tissue dissection and location of the osteotomy. Meanwhile, idiopathic osteonecrosis of the first metatarsal is rare.[40-42]

Vascular Supply

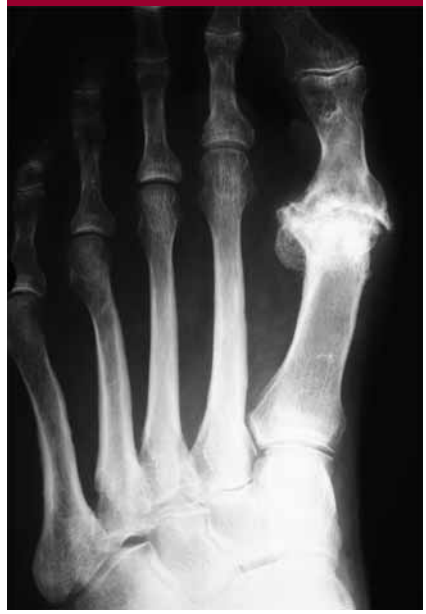
Injection studies have demonstrated that the first metatarsal head is enveloped by an extensive vascular network formed by branches of the dorsalis pedis and posterior tibial arteries.[43] The dorsalis pedis artery provides the first dorsal metatarsal artery and the first plantar metatarsal artery. The first dorsal metatarsal artery gives rise to two to three additional branches. The most superfi-

Figure 4



Lateral view of the vascular supply to the first metatarsal head with the associated Chevron osteotomy cut lines (dashed lines). The shaded area represents the area of the lateral release. (Adapted with permission from Easley ME, Kelly IP: Avascular necrosis of the hallux metatarsal head. *Foot Ankle Clin* 2000;5:591-608.)

Figure 5



Anteroposterior radiograph demonstrating cyst formation and collapse after osteotomy of the distal first metatarsal in a patient with osteonecrosis. (Courtesy of C.W. DiGiovanni, MD, Providence, RI.)

cial of these branches, the dorsomedial hallucal artery, provides a pericapsular network of perforators to the metatarsal head.

A deeper branch of the dorsal metatarsal artery courses plantarly and obliquely under the metatarsal shaft and joins with a branch from the posterior tibial artery. From this anastomosis, branches arise that feed the head. The nutrient artery enters the junction of the proximal and middle third of the bone. The posterior tibial artery contributes the superficial branch of the medial plantar artery that joins with the first plantar metatarsal artery. As shown in Figure 4, the dorsal metaphyseal vessels supply the dorsal two thirds of the head, while the plantar vessels supply the lower one third.[44] The capital arteries supply the medial and lateral fourths of the head.[45]

Incidence and Etiology

Because idiopathic osteonecrosis of the first metatarsal is rare, evidence is limited to case reports. Osteonecrosis following hallux valgus correction is more common, with most reports focusing on distal osteotomies as the cause of the osteonecrosis. The incidence of post-osteotomy osteonecrosis is reported to be as high as 20%.[46] Radiographic signs of osteonecrosis may not be predictive of clinical symptoms.[47]

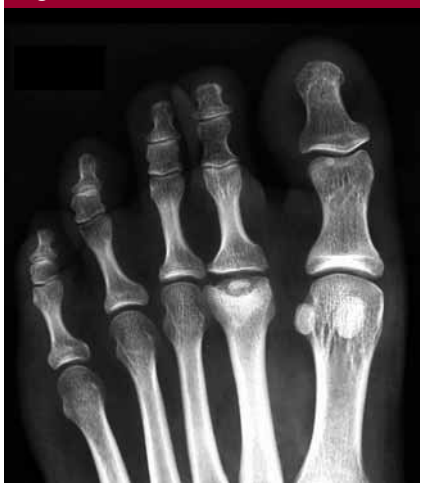
Debate regarding the causal relationship between distal soft-tissue release and osteonecrosis has centered on the vascular implications of this procedure. Retrospective reviews have not definitively linked osteonecrosis to osteotomy performed in conjunction with lateral release.[48,49] Cadaveric studies have demonstrated that perfusion to the metatarsal head is not adversely affected when the lateral release is performed in conjunction with a distal osteotomy. It is evident, however, that careless saw placement could sever the first dorsal metatarsal artery.[49]

Resch et al[50] prospectively followed 36 chevron osteotomies with technetium Tc-99m scintigraphy and reported that an initial filling defect was present in the 19 patients who had undergone concurrent lateral release. All final scans were normal, however. Although the debate over lateral release continues, it remains important to follow basic surgical concepts. Soft-tissue stripping should be performed only to the extent necessary, and power saw entry and exit should be well controlled.

Diagnosis

The diagnosis of first metatarsal osteonecrosis is based on radiographic evidence of bony sclerosis, fragmentation, and collapse (Figure 5). The finding could be incidental, either noted on a postoperative radiograph or identified during evaluation of a patient with medial forefoot pain. MRI allows earlier diagnosis, but it is generally not a cost-effective screening tool.

Figure 6



Anteroposterior radiograph of a patient with Smillie stage III Freiberg's disease. (Courtesy of K. Klaue, MD, Lugano, Switzerland.)

Treatment

Treatment of mildly symptomatic patients includes activity modification and full-length orthotic wear to reduce stress on the first metatarsal. Moderate symptoms warrant simple surgical intervention, such as synovectomy and débridement, or subchondral drilling.[51] Salvage procedures for severe symptoms include first metatarsophalangeal arthrodesis, performed in situ or via interpositional bone graft to preserve length.[52]

Prognosis

The early radiographic changes of postosteotomy osteonecrosis, including subchondral cysts, fragmentation, and sclerosis, may resolve with time. Typically, such patients progress well clinically and go on to radiographic union, even in the event of osteonecrosis. When first metatarsophalangeal arthrodesis is required for salvage, Myerson et al[52] reported healing within an average of 14 weeks.

Lesser Metatarsals

Osteonecrosis of the lesser metatarsals is another relatively common cause of forefoot pain. Attention has primarily focused on osteonecrosis of the metatarsal heads, especially in the second ray.

Vascular Supply

The second through fourth metatarsals have similar arterial supply patterns, originating from the dorsal and plantar metatarsal arteries. Branches pierce the diaphysis along the dorsal, medial, and lateral surfaces. The nutrient artery pierces the lateral base of these bones and bifurcates into both proximal and distal branches.[53] In contrast, the fifth metatarsal has a nutrient vessel that pierces the bone on its medial aspect. The proximal portion is supplied by metaphyseal branches. A watershed area is thereby formed between these two systems, which potentially could prevent fracture healing. There have been no case reports of osteonecrosis in this area.

Freiberg's Disease

Incidence and Etiology In 1914, Freiberg published a description of the disease that eventually bore his name.[54] Dubbed an "infraction" at that time, Freiberg suspected a traumatic etiology. In addition, he postulated that altered joint mechanics contributed to this condition. Freiberg's disease preferentially affects the second metatarsal, the longest metatarsal in the weight-bearing foot.

The mechanics behind resultant dorsal subchondral collapse of the metatarsal head have been evaluated in several studies. McMaster[55] originally described an osteochondral lesion produced by the proximal phalangeal base contacting the second metatarsal head with forced dorsiflexion. Gauthier and Elbaz[56] found that these lesions occur on the dorsal and anterior aspect of the metatarsal head. These authors postulated that collapse was caused by microfracture and necrosis of the subchondral bone. Helal and Gibb[57] linked this dorsal lesion with traumatic effusions.

Diagnosis Typically, Freiberg's disease presents in girls during the adolescent growth spurt. Classically, there is pain on weight bearing and tenderness over the affected metatarsal head. There may be fullness at the joint because of an effusion. Smillie[58] proposed a staging system for this condition, based on the radiographic appearance of the metatarsal head, which can demonstrate various stages of collapse: stage I, fracture of the epiphysis; stage II, subsidence of the central portion; stage III, central reabsorption; stage IV, loose body separation; and stage V, flattening, deformity, arthritis (Figure 6).

Treatment Nonsurgical management includes activity modification, limited weight bearing with crutches, orthosis use, and shoe modifications that off-load the affected metatarsal head and limit affected metatarsophalangeal joint motion. These modalities are effective in the early stages of the disease.

Several surgical options have been advocated. Simple débridement and loose body removal was first described by Freiberg. Other procedures have been used, including osteotomy, elevation of the depressed metatarsal head with bone grafting, core decompression, metatarsal head excision, metatarsal shortening, proximal phalanx hemiphalangectomy, and joint arthroplasty. There is little consensus as to which is the most appropriate or efficacious.[59,60]

Prognosis Many patients with Freiberg's disease respond to nonsurgical therapy and do not experience long-term sequelae. Sproul et al[60] examined patients in whom nonsurgical treatment failed. In most of these patients, partial resection of the dorsal metatarsal head with synovectomy provided long-term relief. Loosening of the dorsal metatarsal articular surface was a consistent surgical finding.

Sesamoids

Osteonecrosis of the sesamoids has been noted in a limited number of case reports.

Vascular Supply

Each sesamoid has both a proximal and a plantar major arterial perforator that arises from the first metatarsal artery. The proximal branch enters the sesamoid at the insertion of the flexor hallucis brevis; the plantar vessel penetrates the bone near its midline. These branches eventually meet in the center of each bone.[53]

Diagnosis

Sesamoid pathology comprises 12% of great toe complex injuries; osteonecrosis of these bones accounts for approximately 10% of this subset.[61] Several names have been given to this condition, including Trevor's disease, Schlatter's disease, and Ilfeld's disease. Pain is the most consistent presenting symptom of this extremely disabling condition. Plain radiographs aid in the diagnosis, but they may not be definitive because sesamoid fracture, congenital bipartism, and osteonecrosis may appear similar on such images. A bone scan may differentiate the high turnover state in fractures from the low turnover state in late-stage osteonecrosis. MRI also may aid in differentiating an acute fracture from osteonecrosis. On a computed tomography scan, the osteonecrotic sesamoid may appear as enlarged and deformed, with irregular densities and fragmentation. The radiographic appearance of the symptomatic foot should be compared with the contralateral side.[62] The speculated mechanism of sesamoid osteonecrosis is secondary to repetitive trauma, which may interrupt the vascular supply to the sesamoids, thus diminishing their ability to remodel.

Treatment

Nonsurgical management includes activity modification, modified weight bearing, and nonsteroidal anti-inflammatory drugs to control symptoms. When these measures fail, surgical removal of the affected bone is indicated.[62,63]

Rare Locations for Osteonecrosis in the Foot

Calcaneus

Unlike the talus, the calcaneus enjoys a rich vascular supply, which may explain the absence of posttraumatic calcaneal osteonecrosis. In the calcaneus, osteonecrosis has been reported in association with sickle cell disease and heart transplant. In addition, Sever's disease, once regarded as a form of osteonecrosis but now considered a childhood apophysitis, can lead to significant pain in the pediatric population.

Vascular Supply

Multiple arterial branches perforate the calcaneus. The artery of the tarsal sinus, with contributions from the lateral tarsal artery and the peroneal artery, supplies the anterior aspect of the superior surface. An anastomotic arcade between the anterior tibial and peroneal arteries feeds the posterior aspect of the calcaneus.

The lateral surface receives branches from the proximal lateral tarsal artery and the peroneal artery. The medial plantar artery and the medial calcaneal branches supply the medial surface.[53]

Etiology and Diagnosis

Rothschild et al[64] radiographically examined patients with sickle cell disease for bone abnormalities. Fourteen percent had erosive cortical changes in a "dot-dash" pattern between the insertion of the Achilles tendon and the subtalar articular surface,[64] with nearly half reporting hindfoot pain. All of the sickle cell patients were treated nonsurgically, and only one patient reported persistent pain at last follow-up.[64]

A second instance of calcaneal osteonecrosis was reported in a 38-year-old heart transplant patient who received two postoperative courses of corticosteroid.[65] The patient was successfully managed nonsurgically.[65]

Sever's Disease

Historically, Sever's disease was regarded as a form of calcaneal osteonecrosis. Today, however, it is considered to be a calcaneal apophysitis, and it represents one of the most common causes of foot pain in the immature athlete. Radiographically, the sclerotic apophysis seen in Sever's disease mimics the appearance of osteonecrosis in other bones. Although of unknown etiology, some have implicated an apophyseal traction injury from the Achilles tendon and the plantar fascia.[66] This mechanical disruption may impede the endochondral mechanism of microfracture healing, leading to a cycle of nonrepairing injury. Thus, although there may be an intrinsic dysvascularity as a result of these factors, current available evidence suggests that Sever's disease is not a form of osteonecrosis but rather is a self-limiting apophysitis devoid of long-term sequela.[66]

Cuneiforms

There are several case reports of cuneiform osteonecrosis.[67-69]

Diagnosis

Patients with cuneiform osteonecrosis present with insidious midfoot pain that is activity-related and occurs at night. The diagnosis is based on radiologic findings of cuneiform collapse and sclerosis in patients with clinical symptoms.

Treatment and Prognosis

In general, nonsurgical management consisting of modified activity and immobilization results in nearly universal symptom improvement or elimination. In one patient, medial cuneiform drilling resulted in symptom resolution.[69]

Cuboid

There is only one English-language case report of cuboid osteonecrosis, in a 63-year-old kidney recipient treated with prednisone.[70] Within 3 months, midfoot pain and swelling developed. Biopsy demonstrated necrosis from blood vessel infiltration with fungal elements. Definitive management consisted of cuboid excision.

Summary

Osteonecrosis must always be considered in the differential diagnosis of both the pediatric and adult patient with chronic foot pain. The disease most commonly affects the talus, navicular, second metatarsal, and first metatarsal. Its etiology is usually posttraumatic; however, systemic disease (eg, Gaucher's, sickle cell) also can produce osteonecrosis. Nonsurgical management remains the mainstay of initial treatment and can be effective in ameliorating symptoms. Surgery is recommended only when nonsurgical management is unsuccessful. Because of its varying incidence, presentation, and sequelae in these bones, there is currently no standard algorithm for managing osteonecrosis in the foot. Therapy is determined on an individual basis. Because of the disability that occa-sionally occurs in even optimally managed patients affected with this disease, more research is paramount to understanding the most effective means of treating and, more importantly, preventing osteonecrosis in the foot.

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Citation numbers printed in **bold type** indicate references published within the past 5 years.

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