

Imaging in the Assessment and Management of Overuse Injuries in the Foot and Ankle

James Teh, B.Sc., M.R.C.P., F.R.C.R.,¹
Ravi Suppiah, B.H.B., M.B.Ch.B., P.G. Dip.S.M.,²
Robert Sharp, M.A., F.R.C.S., F.R.C.S.(Trauma & Orth),³
and Julia Newton, M.R.C.P., D.Phil., Dip.S.E.M.²

ABSTRACT

Overuse injuries of the ankle and foot are common in the general and athletic populations. The wide spectrum of overuse injuries includes ligamentous injuries, soft tissue and osseous impingement, osteochondral lesions, tendon injuries, and stress fractures. Some conditions such as impingement syndromes and stress fractures may be missed on initial physical examination, and patients with such injuries often present to a sports or orthopedic clinic with persistent symptoms. With the increasing participation in sports, health-care professionals involved in the care of athletes at all levels must have a thorough understanding of overuse conditions of the foot and ankle, and the use of imaging in the management of these conditions. This article covers the clinical presentation, pertinent anatomy, imaging features, and management of overuse injuries of the foot and ankle.

KEYWORDS: Overuse, stress fracture, impingement, tendinopathy, foot, ankle

An overuse injury develops as a consequence of unaccustomed or excessive, repetitive activity. Overuse injuries of the foot and ankle may result in impingement syndromes, tendinopathy, ligament injury, and stress fractures. They are a common cause of pain and disability in the general and athletic population. Many of the conditions are difficult to diagnose clinically, and imaging therefore has a key role in the assessment and management of these patients. Diagnostic pathways tend to vary widely between institutions. In general, plain radiographs should be the initial investigation. For cases of suspected soft tissue injury either magnetic resonance imaging (MRI) or ultrasound may be used according to local expertise. For bone lesions, MRI is generally the investigation of choice, but there are times when computed

tomography (CT) should be performed, for example in the context of acute trauma and in the follow-up of stress fractures. This article discusses the clinical presentation, pertinent anatomy, imaging features, and management of overuse injuries of the foot and ankle. Achilles disorders are covered in a separate article.

IMPINGEMENT SYNDROMES

Impingement syndromes around the ankle may result in a combination of bone, soft tissue, and nerve impingement. They represent an important cause of chronic pain and debility in the athletic population. The diagnosis is usually clinically based, and radiological evaluation is not always necessary.¹

¹Department of Radiology, ²Department of Sports and Exercise Medicine, ³Department of Foot and Ankle Surgery, Nuffield Orthopaedic Centre NHS Trust, Headington, Oxford, United Kingdom.

Address for correspondence and reprint requests: James Teh, M.R.C.P., F.R.C.R., Department of Radiology, Nuffield Orthopaedic Centre NHS Trust, Windmill Road, Headington, Oxford OX3 7LD, UK (e-mail: jamesteh1@googlemail.com).

Sports Injury of the Lower Extremity: Role of Imaging in Diagnosis

and Management; Guest Editors, Jeremiah C. Healy, F.R.C.P., F.R.C.R., F.F.S.E.M. and Justin C. Lee, M.R.C.S., F.R.C.R.

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Posterior Impingement Syndrome

The typical presentation is of posterior ankle pain of insidious onset in subjects such as ballerinas who regularly perform forced plantar flexion activities.² Radiographs may demonstrate an os trigonum or Stieda process. Ultrasound or MRI may reveal synovial thickening at the posterior aspect of the ankle with hypoechoic nodular thickening and flexor hallucis longus (FHL) tenosynovitis^{3,4} (Fig. 1).

Posteromedial Impingement Syndrome

Posteromedial ankle impingement syndrome usually follows severe inversion injury in which the deltoid ligament is crushed between the medial malleolus and talus.⁵ On ultrasound and MRI there is loss of striation of the posterior tibiotalar ligament as well as abnormal signal abutting and encasing the medial tendons⁶ (Fig. 2).

Anterior Impingement Syndrome

Anterior impingement is a common problem in football players. Typically repeated ankle dorsiflexion activity results in chondral damage, trabecular microfractures, or periosteal hemorrhage, with subsequent anterior spur formation.⁷ Plain radiographs demonstrate anterior spurs and may be the only imaging required. On MRI bone edema, synovitis, and articular cartilage loss may be seen (Fig. 3).

Anterolateral Impingement

Ankle inversion injury may result in lateral ligament instability, which causes recurrent synovitis and

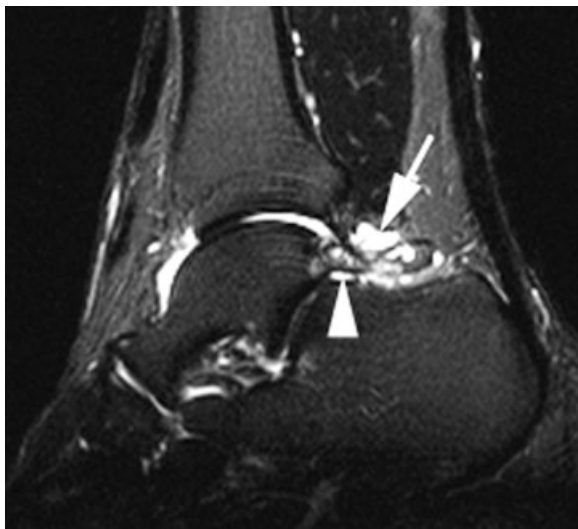


Figure 1 Posterior impingement syndrome. Sagittal short tau inversion recovery image demonstrates bone edema within an os trigonum (arrowhead) with associated synovitis (arrow).

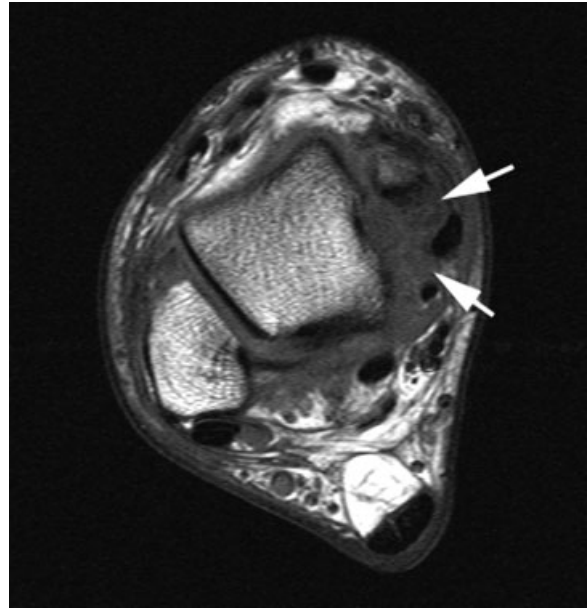


Figure 2 Posteromedial impingement syndrome in a 25-year-old soccer player. Axial T1-weighted image shows posteromedial synovial thickening (arrows) with loss of striation of the tibiotalar ligament.

hemorrhage followed by scarring and synovitis. On MRI there is distension of the anterolateral gutter with capsular irregularity. In advanced cases there may be molding of synovial tissue to form a hyalinized *meniscoid lesion*,⁸ which may also be seen on ultrasound⁹ (Fig. 4).

Anteromedial Impingement

Anteromedial impingement is poorly understood but is increasingly recognized. Patients typically give a history

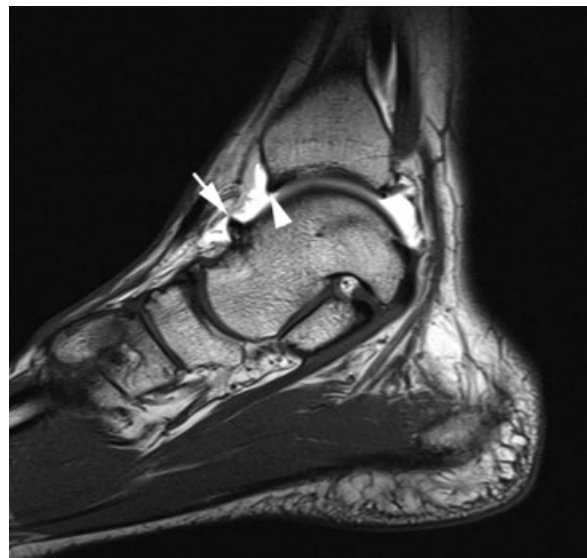


Figure 3 Anterior impingement syndrome. Sagittal T1-weighted magnetic resonance arthrogram showing anterior osteophytes (arrow and arrowhead).



Figure 4 Anterolateral impingement syndrome. Axial T2-weighted image demonstrating distension of the anterolateral gutter with synovial stranding (arrow) and laxity of the anterior talofibular ligament.

of supination injury followed by anteromedial pain. Repetitive microtrauma leads to synovitis with anteromedial spur formation.¹⁰ The imaging findings have not been described on conventional MRI or ultrasound. On MR arthrography, joint distension allows appreciation of irregular anteromedial synovial thickening (Fig. 5).

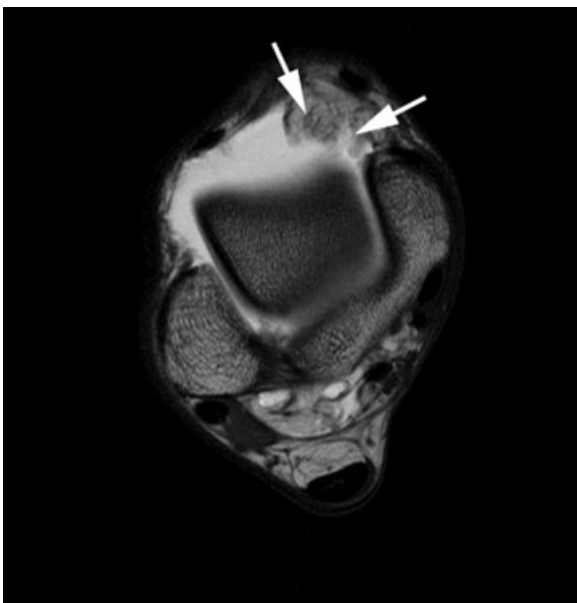


Figure 5 Anteromedial impingement syndrome. Axial T1-weighted magnetic resonance arthrogram showing distension of the anterior joint space with anteromedial synovitis (arrows).

Management of Impingement Syndromes

Relative rest from the culprit activity and physiotherapy should be integral to the management. Image-guided injection may give lasting relief. In resistant cases, especially when imaging studies demonstrate bony structures contributing to impingement, surgery may be required.

SINUS TARSI SYNDROME

Presentation

Sinus tarsi syndrome is characterized by focal pain and tenderness in the sinus tarsi that is responsive to injection of local anesthetic. The symptoms are often associated with a feeling of instability and aggravation by weightbearing activity. Most cases arise following trauma, but the syndrome can be associated with inflammatory arthritis or foot deformities such as pes planus.¹¹

Anatomy and Imaging

The sinus tarsi is a cone-shaped cavity located between the neck of the talus and the anterosuperior surface of the calcaneus. It contains fat, vessels, nerves, and ligaments. With sinus tarsi syndrome there is replacement of normal fat signal intensity in the sinus tarsi with fluid or inflammatory tissue, with resultant low T1 signal intensity and high T2 signal intensity¹¹ (Fig. 6). Associated features include lateral ligament tears, tibialis posterior tendon tears, and ganglia arising from the posterior subtalar joint.¹²



Figure 6 Sinus tarsi syndrome. Sagittal T1-weighted image demonstrating complete replacement of the fat signal within the sinus tarsi (arrow).

Management

Conservative treatment consists of corticosteroid injections, along with physiotherapy and correction of foot biomechanics. Sinus tarsectomy or subtalar joint synovectomy is reserved for persistent symptoms.¹³

TARSAL TUNNEL SYNDROME

Clinical Presentation

Tarsal tunnel syndrome refers to an entrapment neuropathy of the posterior tibial nerve or its branches as they transverse the tarsal tunnel.¹⁴ Trauma, tarsal coalition, or any space-occupying lesion may cause the syndrome. Repetitive overpronation that can occur on the inside foot of a track runner is also recognized as a predisposing factor. Patients complain of paraesthesia or a burning sensation over the heel, plantar surface of the foot, and first three toes. A positive Tinel's test may be present. Nerve conduction testing can be useful because the symptoms are often nonspecific.

Anatomy and Imaging

The tarsal tunnel is a continuation of the deep posterior compartment of the lower leg into the medial aspect of the foot.¹⁴ The medial ankle tendons and neurovascular bundle pass through the tunnel. The tibial nerve divides into terminal branches of the medial calcaneal nerve and the medial and lateral plantar nerve. Imaging is used to exclude space-occupying lesions such as ganglia, nerve sheath tumors, tendinopathy, accessory muscles, or varicosities (Fig. 7).

Management

Rest, orthotics and steroid injections into the tarsal tunnel may be beneficial. Even in the absence of a mass lesion, surgical decompression may be curative.

TENDON PATHOLOGY

Peroneal Tendons

CLINICAL PRESENTATION

Peroneal tendon injuries should be considered in all patients who present with chronic lateral ankle pain. The main conditions affecting the peroneal tendons are tenosynovitis, tendinosis, tendon tears, and tendon subluxation.

PERONEAL TENDINOPATHY

Peroneal tendinopathy is closely linked to lateral ligamentous injury. Anatomical variations such as a shallow retromalleolar groove, a low-lying muscle belly, an

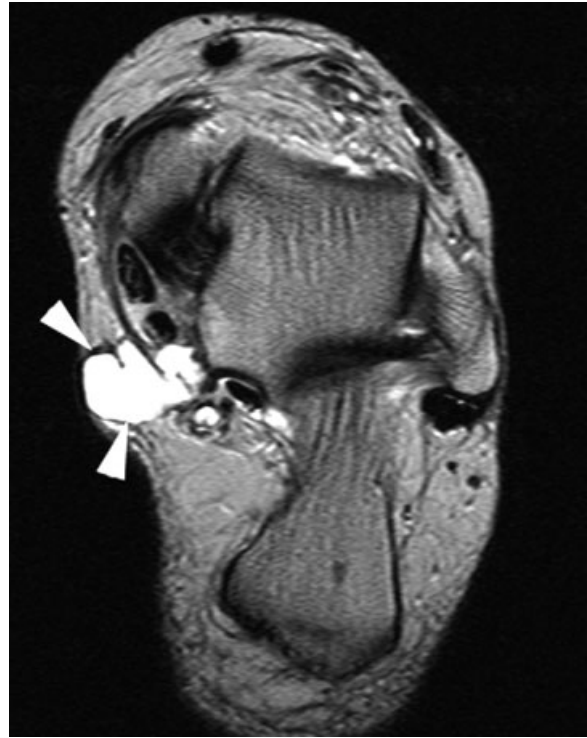


Figure 7 Tarsal tunnel syndrome. Axial T2-weighted image demonstrating a ganglion within the tarsal tunnel (arrowheads).

accessory peroneus quartus, or enlarged peroneal tubercle are associated with peroneal tendinopathy.¹⁵ On MRI, thickening or altered signal in the tendon indicates tendinosis. On ultrasound, there may be loss of the normal fibrillar structure of the tendon with areas of altered echogenicity and increased vascularity on Doppler (Fig. 8). If the cross-sectional area of tendon sheath fluid is greater than that of the tendon or if complex fluid is present, tenosynovitis can be diagnosed. Impingement

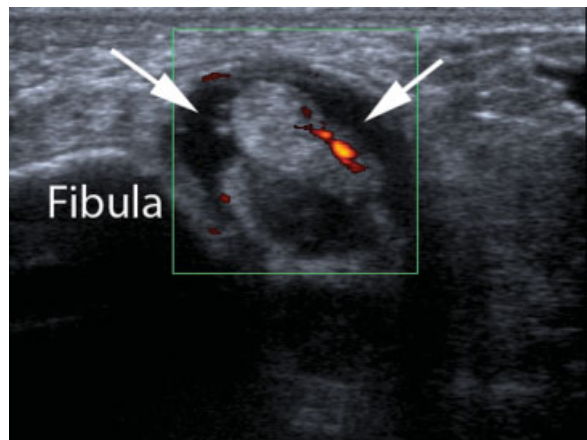


Figure 8 Peroneal tenosynovitis. Transverse ultrasound at the level of the lateral malleolus indicating peroneal tenosynovitis (arrows) with minor increased vascularity on Doppler.

of the tendon may occur at the level of the cuboid as the peroneus longus courses through the cuboid tunnel¹⁶ (Fig. 9). If there is fluid signal within the substance of the tendon, loss of continuity of fibers or nonvisualization of the tendon, a tear should be considered.

PERONEAL TENDON SPLITS

Chronic impingement of the peroneus brevis between the fibula and peroneus longus may result in a peroneus brevis split. In the early stages the peroneus brevis becomes boomerang shaped, with the peroneus longus lying in the concavity. Eventually the peroneus brevis may be seen as two separate structures on either side of the peroneus longus on axial images¹⁷ (Fig. 10). Peroneus longus tears can occur with peroneus brevis tears at the level of the lateral malleolus or be isolated at the level of the midfoot.

Painful os peroneum syndrome (POPS) describes a spectrum of disorders caused by chronic repetitive injury that occurs in patients who have an os peroneum. The syndrome may be associated with a fracture of the os peroneum, with tenosynovitis or tendinosis.¹⁸ A peroneus longus tear may occur near the os peroneum with retraction of the os, which normally is situated at the level of the calcaneocuboid joint.¹⁹ Os peroneum fragment separation of ≥ 6 mm suggests an os peroneum fracture with a peroneus longus tendon tear.¹⁹

PERONEAL SUBLUXATION

Peroneal tendon subluxation usually occurs as a result of peroneal retinaculum injury. A hypoplastic peroneal fibular groove may also play a role. Subluxation may

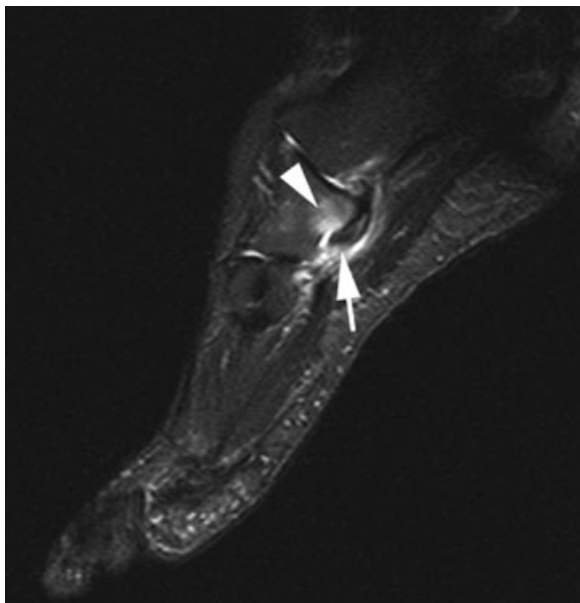


Figure 9 Cuboid tunnel syndrome. Sagittal short tau inversion recovery image demonstrating peroneus longus tenosynovitis at the level of the cuboid (arrow), with bone edema in the cuboid (arrowhead).

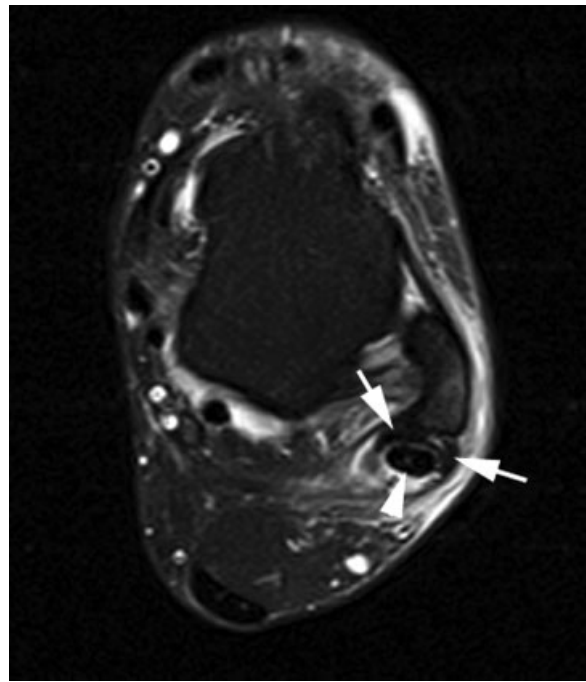


Figure 10 Peroneus brevis split. Axial T2 fat-saturated image demonstrating a longitudinal split of peroneus brevis (arrows), with the split tendon lying on either side of the peroneus longus tendon (arrowhead).

not be seen on static MRI. Therefore, if peroneal subluxation is suspected, dynamic ultrasound is the preferred imaging modality.²⁰ Either or both peroneal tendons may sublux. The tendons may remain in the retromalleolar groove but can reverse their positions, a condition described as *retromalleolar intrasheath dislocation* (Figs. 11 and 12).

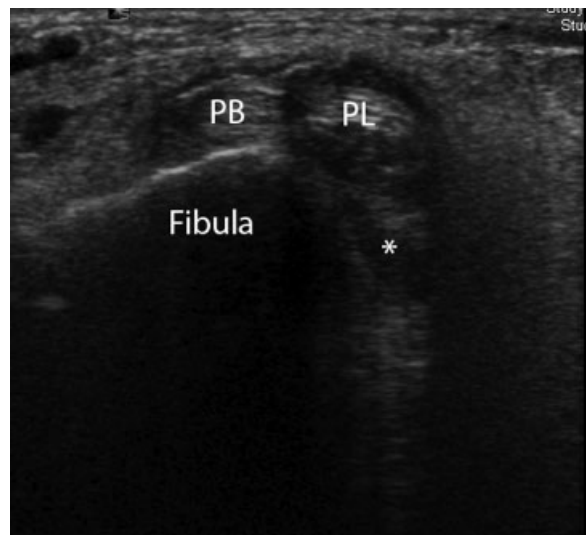


Figure 11 Peroneal subluxation. Transverse ultrasound scan at the level of the lateral malleolus demonstrating anterior subluxation of the peroneus brevis (PB) and peroneus longus (PL) tendons. The asterisk shows the normal position of the tendons.

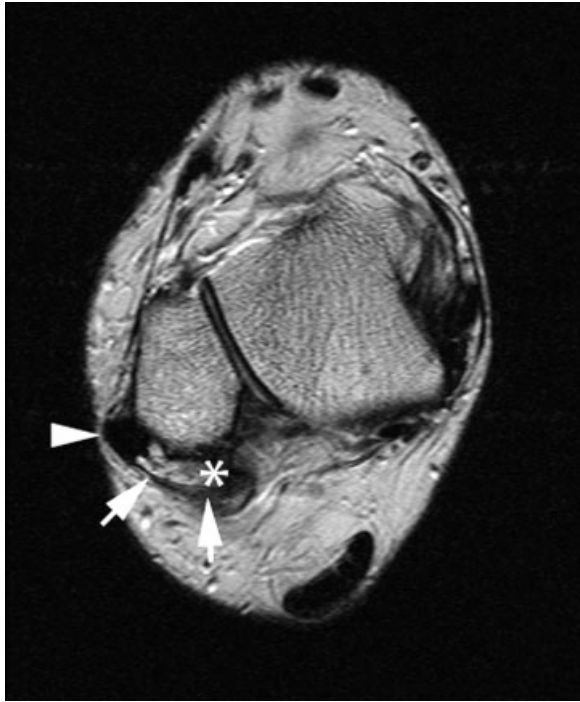


Figure 12 Peroneal subluxation. Axial T2-weighted image demonstrating a lax peroneal retinaculum (short arrows) and laterally subluxed peroneal tendons (arrowhead). The asterisk shows the normal position of the tendons.

MANAGEMENT

Peroneal tendinopathy is usually treated conservatively with rest, orthotics, and resistance exercises. Injections may result in tendon rupture and are therefore not recommended. Conservative management may be attempted in acute dislocations, although operative management is preferred in athletes. Recurrent dislocations should be managed surgically.

Tibialis Posterior Tendon and Spring Ligament

CLINICAL PRESENTATION

Tibialis posterior dysfunction is an overuse condition that is more common in middle-aged women, the obese, and people with diabetes.²¹ Patients usually present with medial pain and swelling with gradual loss of the plantar arch, resulting in pes planus.

TIBIALIS POSTERIOR TENDINOPATHY

The tibialis posterior acts as an important stabilizer of the arch and is the main everter of the foot. Tendinosis may be diagnosed if the tendon is thickened or if there is altered texture. Distinction between severe tendinosis and partial tears may be difficult due to morphological overlap between the two conditions. The presence of intrasubstance longitudinal splits indicates a type 1 tear. The normal tibialis posterior tendon is around twice the

size of the adjacent flexor digitorum longus tendon. A tibialis posterior tendon that is the same size or smaller than the adjacent flexor digitorum tendon indicates chronic attrition and partial tearing (type 2 tear). With complete tears, a gap and tendon retraction may be evident (type 3 tear). Tibialis posterior tendinopathy is commonly associated with injury to the calcaneonavicular portion of the spring ligament and sinus tarsi²² (Fig. 13). On MRI the spring ligament is best seen on axial images and should appear as a low signal structure on all sequences, extending from the sustentaculum to the navicular, lying deep to the tibialis posterior tendon.²³ On ultrasound there is a striated appearance with no flow on Doppler.²⁴ Insufficiency of the spring ligament is usually manifest by thickening and laxity, with blurring of the margins.

MANAGEMENT

Nonoperative treatment of tibialis posterior tendinopathy includes orthotics and physiotherapy. Injections are not advised due to the increased risk of tendon rupture. Operative treatment of early-stage disease includes tenosynovectomy, with debridement or repair of tears.²¹ With more advanced disease, tendon transfer, usually with the flexor digitorum longus, is performed in conjunction with a calcaneal osteotomy. The most severe cases may require hindfoot fusion.

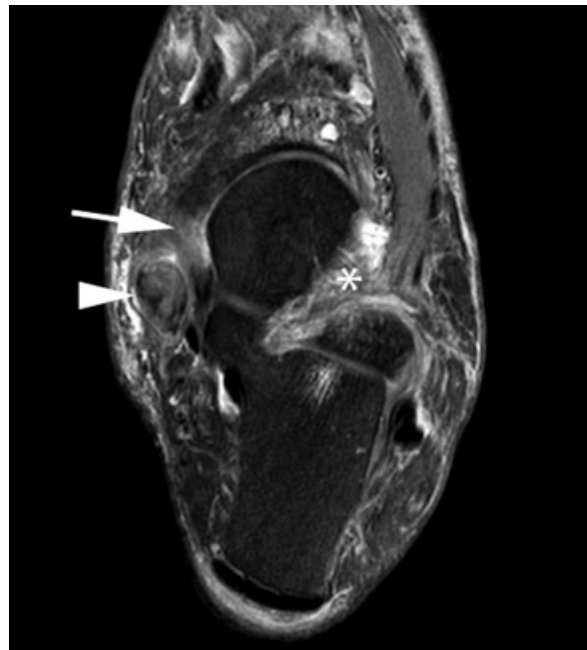


Figure 13 Tibialis posterior tendinopathy with spring ligament insufficiency and sinus tarsi syndrome. Axial oblique T2 fat-saturated image showing a markedly swollen, partially torn tibialis posterior tendon with altered signal (arrowhead), a lax spring ligament with increased signal (arrow), and edema in the sinus tarsi (asterisk).

Painful Accessory Navicular

CLINICAL PRESENTATION

The accessory navicular is one of the commonest accessory ossicles of the foot. Although usually asymptomatic, there may be pain, swelling, and localized tenderness.

ANATOMY AND IMAGING

Three types of accessory navicular bone are described:²⁵ Type I is a sesamoid bone within the posterior tibial tendon, anatomically separate from the navicular. Type II is an accessory ossification center lying medial to the navicular. It is bridged by a fibrocartilaginous synchondrosis to the navicular bone and serves as the point of attachment for the tibialis posterior tendon. Type III is a fused or partly fused accessory navicular, forming a cornuate navicular. Type II is the most commonly symptomatic variant, presenting with localized medial foot pain and tenderness with associated soft tissue inflammation. Radiographs may be all that is required to confirm the clinical suspicion. Ultrasound allows evaluation of the tibialis posterior tendon insertion and comparison with the contralateral side but is unable to determine the presence of bony stress change. Bone scintigraphy has a high sensitivity but lacks specificity. MRI is the examination of choice for demonstrating both bone changes and tibialis posterior tendinosis²⁶ (Fig. 14).

MANAGEMENT

If conservative management is not successful, the accessory ossicle can be fused or removed.



Figure 14 Symptomatic type 2 accessory navicular. Sagittal short tau inversion recovery image demonstrating a type 2 accessory navicular (arrowhead) with minor bone edema at the synchondrosis. The tibialis posterior tendon (arrow) appears normal.

Extensor Tendons

Closed injuries of the extensor tendons are uncommon. The anterior tibial tendon can be injured by friction against the inferior extensor retinaculum, talonavicular osteophytes, or bony excrescences, leading to tenosynovitis or rupture. Downhill runners, football players, and skiers who are subject to forced plantar flexion may be susceptible. The typical presentation for anterior tibial tendon rupture is localized pain with a foot drop. Characteristic MRI findings include tendon thickening (> 5 mm) and diffuse or signal abnormalities of the tendon.²⁷ Ultrasound also allows excellent depiction of tibialis anterior tendinopathy and is useful for marking the extent of tendon retraction before surgery in cases of rupture. The stump may retract to the level of the ankle joint, with fluid or scar tissue filling the gap.

PLANTAR FASCIA LESIONS

Plantar Fasciitis

CLINICAL PRESENTATION

Plantar fasciitis is the most common cause of heel pain and tenderness. Runners and obese patients are frequently affected, as are patients with seronegative arthropathy. Discomfort is usually worst during the first few steps in the morning and improves with movement initially but then deteriorates with continued activity.

ANATOMY AND IMAGING

The plantar fascia is the most important structure for supporting the longitudinal arch.²⁸ Repetitive activity results in microtears and traction at the enthesis, which heals by fibroblastic proliferation, resulting in thickening. The central and medial bundles are most commonly involved. Plantar calcaneal spurs are often seen, but the finding is present in up to 25% of the asymptomatic population. On sagittal MRI the plantar fascia is visualized as a well-defined 2- to 4-mm-thick low signal band on all sequences. The main feature of plantar fasciitis on MRI is fusiform swelling at the calcaneal insertion.²⁹ Focal bone edema may be present. The associated inflammatory reaction can cause entrapment of the lateral plantar nerve (Baxter's neuropathy), which manifests as denervation of the abductor digiti minimi muscle. On ultrasound the plantar fascia has an echogenic fibrillar structure, and it is best assessed in the longitudinal plane.²⁸ With plantar fasciitis there is fusiform thickening, with focal or generalized loss of reflectivity. Enthesal new bone formation or spur formation may be seen (Fig. 15).

MANAGEMENT

Conservative therapy includes rest, physiotherapy, and massage. Orthotics should be prescribed to provide local

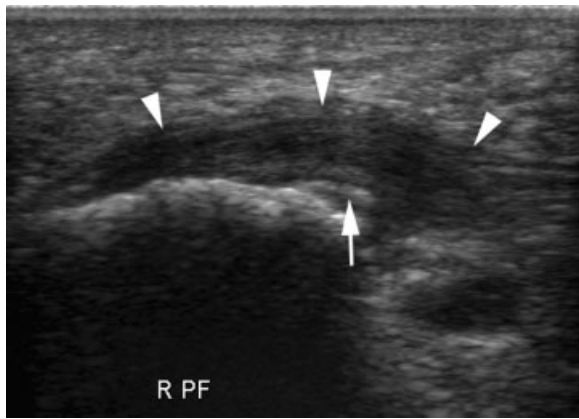


Figure 15 Plantar fasciitis. Longitudinal ultrasound scan demonstrating a swollen plantar fascia insertion (arrowheads) with enthesopathic change (arrow).

comfort and correct gait disturbances. Corticosteroid injections and dry needling can improve symptoms, but meta-analysis shows no good evidence for sustained improvement.³⁰ Injections are associated with a risk of fascial rupture and fat pad atrophy. Extracorporeal shock wave therapy using a variety of techniques has been shown to give modest benefit. Plantar fascia release is the last resort in treatment.

Plantar Fascia Rupture

CLINICAL PRESENTATION

Plantar fascia rupture may occur in the context of overuse in patients with underlying plantar fasciitis or as an acute injury in running or jumping athletes. There is a strong association with previous steroid injections. Patients usually experience a pop with a tearing pain, followed by swelling.

IMAGING

On MRI there is interruption of the low signal plantar fascia with high signal intensity on a short tau inversion recovery (STIR) or T2 fat-saturated sequence.²⁸ Most tears occur close to the calcaneal insertion. In the acute phase there is usually surrounding soft tissue high signal indicating edema and hemorrhage. On ultrasound there is loss of continuity of the plantar fascia with loss of the normal fibrillar structure, with hypoechoic tissue around the injury indicating edema and hemorrhage.

MANAGEMENT

The treatment protocol usually comprises 2 to 3 weeks of nonweightbearing in a removable boot followed by protected weightbearing for 1 to 3 weeks. This may be combined with ultrasound therapy, ice, and deep massage.

Table 1 Classification of Osteochondral Lesions

| | |
|---------|--|
| Stage 1 | Subchondral bone edema with intact overlying cartilage |
| Stage 2 | Partially detached stable osteochondral fragment |
| Stage 3 | Completely detached but in situ fragment |
| Stage 4 | Displaced osteochondral fragment |

Adapted from Berndt and Harty.³¹

Osteochondral Injury

CLINICAL PRESENTATION

Osteochondral lesions of the talar dome are referred to as osteochondritis dissecans, osteochondral defects, and osteochondral fractures. They are usually due to impaction following twisting injury of the ankle.³¹ Some lesions are related to repetitive stress, abnormal stress due to instability, or microemboli resulting in infarcts. They present with ankle pain, swelling, and dysfunction. Early recognition is important because premature osteoarthritis may occur.

IMAGING

MRI is useful for detecting the presence, location, and size of the lesion, as well as assessing the integrity of the overlying cartilage, the degree of attachment, and the location and viability of fragments. Treatment is based on the degree of detachment of the osteochondral fragment and therefore requires precise classification. Osteochondral lesions have been classified into four stages (Berndt and Harty classification) based on radiographic and surgical findings³¹ (Table 1). Using arthroscopy as the gold standard, Mintz et al described disease-negative (grades 0 and 1) and disease-positive (grades 2 to 5) status, and found that MRI had a sensitivity of 95% and a specificity of 100% (Table 2). Medial talar dome lesions tend to occur following inversion injury with impaction of the tibia against the posteromedial dome, giving a cup-shaped lesion (Fig. 16). Lateral lesions follow inversion injury with the foot in dorsiflexion giving thinner, more wafer-like lesions, which tend to be more symptomatic and unstable than medial lesions (Fig. 17). A high T2 signal intensity interface between the normal bone and the fragment suggests instability, whereas low signal suggests healing.³² Focal defects of the overlying cartilage and cyst like lesions at the inter-

Table 2 Grading of Osteochondral Lesions

| | |
|---|--|
| 0 | Normal cartilage |
| 1 | Abnormal signal but intact cartilage |
| 2 | Fibrillation or fissures not extending to bone |
| 3 | Flap present or bone exposed |
| 4 | Loose undisplaced fragment |
| 5 | Displaced fragment |

Adapted from Mintz et al.⁴⁶



Figure 16 Medial talar dome osteochondral lesion with in situ fragment. Coronal proton-density fat-saturated sequence showing a cup-shaped lesion with a low signal in situ fragment (arrowhead).

face between the fragment and the bone are also features of instability. If the fragment is of low signal intensity on all sequences, osteonecrosis is likely, whereas if normal marrow signal is seen on a T1-weighted image, it



Figure 17 Lateral talar dome lesion. Coronal proton-density fat-saturated sequence showing a wafer-like osteochondral lesion (arrowhead) with a low signal subchondral fracture line and early cyst formation.

suggests the presence of viable bone marrow.³³ Administering intravenous gadolinium can further assess viability. If the fragment enhances, it indicates vascularized viable tissue. CT may allow accurate delineation of the fragment and assess the degree of detachment (Fig. 18). The accuracy of grading of osteochondral lesions is improved by arthrography. If contrast is interposed between the fragment and the donor site, it indicates instability.

MANAGEMENT

The treatment aims are to promote revascularization and healing and to prevent detachment of the fragment. Nonoperative management is recommended when the cartilage is intact and the lesion is considered stable. Operative treatment is advocated for unstable lesions, particularly when there is articular incongruity or osteonecrosis of the fragment.³⁴ Arthroscopic microdrilling and open procedures, such as osteochondral autograft transfer system and autologous chondrocyte implantation, may be employed.

Stress Fractures

CLINICAL PRESENTATION

Stress fractures may be categorized into fatigue and insufficiency fractures. Fatigue fractures occur due to continuous repetitive strain in normal healthy bone. They usually occur in athletes who have rapidly increased their training or in subjects such as military recruits who undertake unaccustomed exercise. Insufficiency fractures

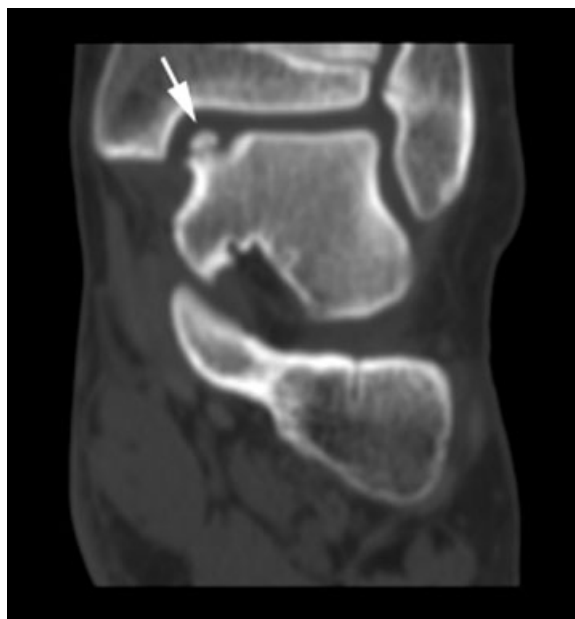


Figure 18 Medial talar dome lesion. Computed tomography with coronal reformat showing a well-defined in situ fragment (arrow).

occur due to normal activity in bone that is mineral deficient. They usually occur in elderly osteoporotic women but can occur in younger patients with underlying hormonal, nutritional, or metabolic disorders. The presentation is typically of localized pain on impact, which settles with rest.

PATHOGENESIS AND IMAGING

A stress reaction occurs as the first stage in the development of a stress fracture. This manifests as edema and hyperemia. As the injury progresses, an irregular, often incomplete break may occur. Usually the fracture runs perpendicular to the cortex. Shortly after the fracture occurs, periosteal callus is laid down, followed by cortical thickening and irregularity. Eventually there is bony remodeling.

Plain Radiographs

The first-line investigation for a suspected stress fracture should be the plain radiograph because they are useful if positive and help rule out other conditions. However, the sensitivity of radiographs for detecting early stress fractures is relatively poor, ranging from 15 to 56%.^{35,36} Therefore, if the clinical features are suggestive but initial radiographs are negative, the index of suspicion should remain high and further imaging should be performed. Follow-up radiographs usually show callus formation and are therefore useful for assessing the evolution stress fractures. Early stress injury in cortical bone results in poor definition of the cortex and faint intracortical lucent striations. This is followed by periosteal reaction and endosteal thickening as buttressing occurs. Eventually a radiolucent cortical fracture line becomes visible.

Magnetic Resonance Imaging

MRI is superior to other modalities for the evaluation of stress fractures with its excellent contrast resolution, multiplanar capability, and lack of ionizing radiation. MRI has been shown to be more sensitive than CT and scintigraphy (88% compared with 42% and 74%, respectively) for detection of tibial stress fractures.³⁷ Routine examination should include T1-weighted and edema-sensitive sequences in at least two orthogonal planes. Intravenous gadolinium is not usually required but may improve sensitivity. Initially there is bone, periosteal, and soft tissue edema that is only demonstrated on edema-sensitive sequences. A stress reaction appears as poorly defined abnormal signal intensity within the bone marrow similar to that of a bone contusion with intermediate/low intensity on T1-weighted images and high signal on STIR.³⁸ Eventually a fracture line appears as a band of linear decreased signal intensity extending perpendicular to the cortex into the medullary canal.

Surrounding bone and soft tissue edema is usually present.

Bone Scintigraphy

Before the advent of MRI, bone scintigraphy using technetium-99m analogs was the gold standard for diagnosing early stress fractures. Scintigraphy demonstrates increased bone uptake at sites of increased osteoblastic activity before changes on plain radiographs and CT,³⁵ becoming positive within 72 hours of injury, with a sensitivity approaching 100%. However, tumors, infection, and bone infarction can cause false positives, so correlation with other imaging modalities is essential. Scintigraphy is of particular value when MRI cannot be performed.

Computed Tomography

CT can identify subtle cortical abnormalities associated with stress fractures such as periostitis, osteopenia, cortical lucency, and bony bridging. CT has an important role in the evaluation and healing of stress fractures of the foot, particularly those involving the navicular³⁹ (Fig. 19). CT can also characterize lesions such as osteoid osteomas by visualization of the nidus.³⁵

Ultrasound

Ultrasound can detect cortical breaks and periosteal reactions but is not routinely used for the primary diagnosis of stress fractures. The periosteal reaction associated with metatarsal stress fractures can be demonstrated before changes on radiographs. On Doppler, increased vascularity may be present around the fracture site (Fig. 20).

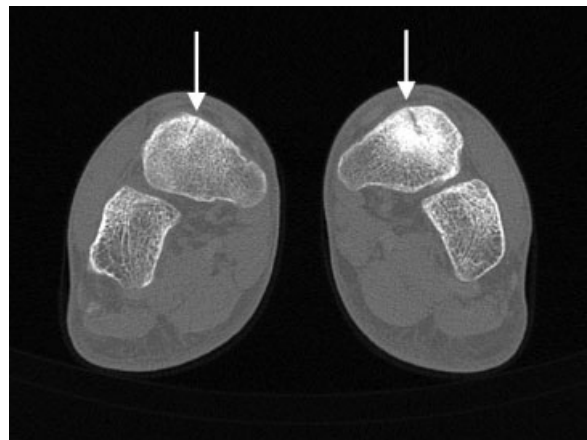


Figure 19 Bilateral navicular stress fractures. Coronal computed tomography scan showing bilateral fracture lines (arrows) in the sagittal plane with surrounding sclerosis.

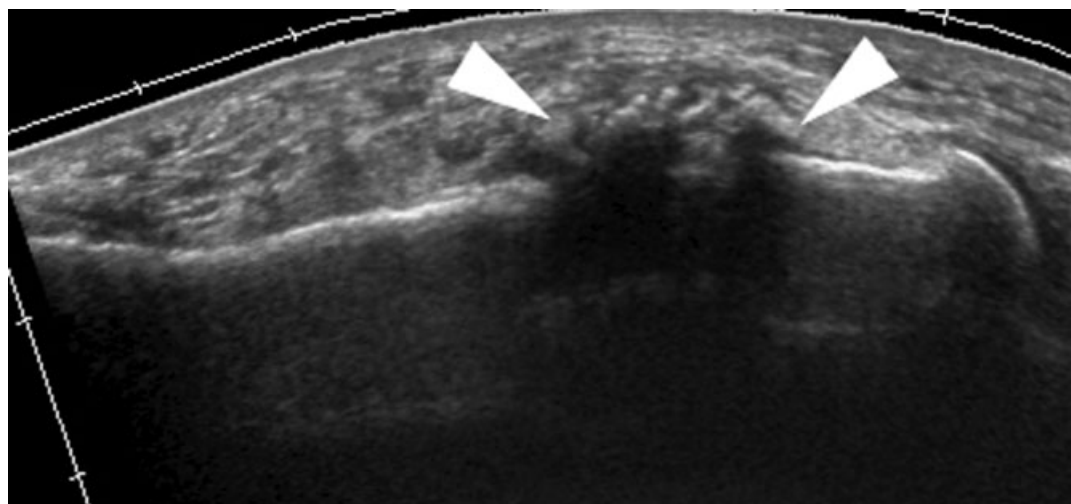


Figure 20 Metatarsal stress fracture. Longitudinal extended field-of-view ultrasound image showing periosteal reaction (arrowheads) at the fracture site.

STRESS FRACTURES AFFECTING THE FOOT

Metatarsal stress fractures, or *march fractures*, usually affect the shaft of the second and third rays. Stress fractures localized at the base of the second or third metatarsals are classically seen in ballet dancers. Stress fractures of the fifth metatarsal shaft usually occur in the proximal third.

Calcaneal stress fractures may be missed on up to 85% of cases on radiographs.⁴⁰ When visible they typically appear as a thin band of sclerosis that parallels the posterior cortical margin running perpendicular to the trabeculae. Most fractures occur in the posterior part of the calcaneus (Fig. 21). Anterior process

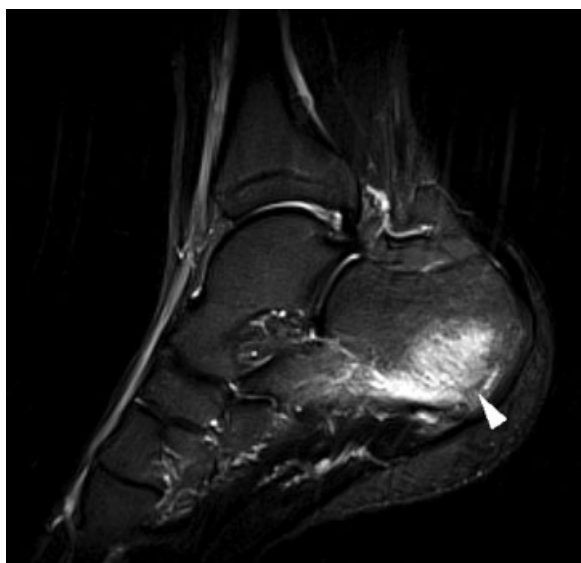


Figure 21 Calcaneal stress fracture. Sagittal short tau inversion recovery sequence showing bone edema with a subtle low signal fracture line parallel to the posterior cortex (arrowhead).

fractures are associated with nonosseous calcaneonavicular coalition and are rare, comprising ~1% of calcaneal fractures.

Navicular stress fractures tend to occur in sprinting and jumping athletes. Patients complain of insidious onset of pain or a cramping sensation brought on by activity. In the early stages patients may be able to continue running if the forefoot is not involved in foot strike. Navicular stress fractures are often difficult to diagnose on radiographs and can result in significant disability if the diagnosis is delayed.⁴¹ MRI is therefore recommended if there is a clinical suspicion. CT also has an important role in delineating and following up on these fractures.⁴² The fracture may be partial or complete and usually involves the relatively avascular central or lateral portion of the navicular in the sagittal plane, which is the site of maximum shear stress. The fracture may have a Y-shaped configuration. Cuneiform and cuboid stress fractures are uncommon but may be associated with plantar fascia injury, changes in gait, and large body habitus.

MANAGEMENT

Simple stress fractures of the foot and ankle settle with rest and protected weightbearing in an air-cast boot, followed by a graded return to training. The approximate time to full training is 4 to 8 weeks from initiation of treatment. Certain fractures such as those to the base of the second or fifth metatarsal and the navicular may require internal fixation.

METATARSALGIA

Pain within or around the metatarsophalangeal (MTP) joints (metatarsalgia) is a common problem with a wide differential diagnosis including trauma, Freiberg's infraction, tendinopathy, and Morton's neuroma.

Freiberg's Disease

Freiberg's disease is a condition characterized by collapse of the second or third metatarsal head, thought to be related to repetitive trauma and avascular necrosis. It occurs more commonly in females and typically affects adolescents. On radiographs there is collapse and irregularity of the metatarsal head. On MRI a subchondral fracture line may be seen in the metatarsal head, in association with synovitis at the metatarsophalangeal joint. If an early diagnosis is made, then offloading the metatarsal head may be sufficient. If synovitis is a major feature, steroid injection may give pain relief. Surgery should be reserved for recalcitrant cases.

Morton's Neuroma

A Morton's neuroma is not a true neuroma but rather perineural fibrosis due to entrapment of the interdigital nerve at the level of the metatarsal heads. It most commonly occurs in the third, followed by the second interspace. Women are affected more than men. The typical presentation is of pain, with burning and tingling sensations. On lateral compression a click may be felt (Mulder's click).

PATHOLOGY AND IMAGING

On MRI, Morton's neuromas are best demonstrated on coronal T1-weighted images through the level of the metatarsal heads, appearing isointense to slightly hyperintense relative to muscle. On T2-weighted and STIR images Morton's neuromas tend to be hypointense, resulting in poor lesion conspicuity. If there is associated intermetatarsal bursitis, fluid signal is seen. Ultrasound is an excellent modality for assessing Morton's neuromas because the imaging findings can be correlated with symptoms. In the sagittal plane the lesion appears as a low signal rounded hypoechoic nodule that may be partly compressible.⁴³

MANAGEMENT

Some clinicians consider imaging unnecessary for diagnosis, but imaging can exclude other causes of metatarsalgia and can be used to guide therapeutic injection. Footwear modification and targeted cortisone injections are the main nonoperative measures. If symptoms persist, excision can be performed.

Plantar Plate Injury

Plantar plate insufficiency may result from wearing high-heeled shoes, hypermobility, hyperextension injury, and overuse injury. *Turf toe* refers to an acute injury resulting from a severe hyperextension injury associated with playing sports on a hard surface such as artificial turf, with rupture of the plantar plate close to the distal insertion. The first or second metatarsophalangeal joints are usually affected. Following the injury, running and

push-off is compromised. Long-term sequelae include hallux rigidus and hallux valgus.

ANATOMY AND IMAGING

The plantar plate is a fibrocartilaginous structure attaching the metatarsal neck to the base of the proximal phalanx, blending with the sesamoids and flexor hallucis brevis tendon to resist hyperextension and provide structural support.⁴⁴ On ultrasound the plantar plate appears as a low echogenicity band with a grainy homogeneous texture. On MRI, the normal plantar plate is a smooth, curvilinear, low signal structure abutting the plantar aspect of the metatarsal head. Tears manifest as discontinuity of the plate with an area of increased T2 signal intensity. There may be associated bone edema, synovitis, flexor tendon sheath synovitis, and hyperextension of the proximal phalanx (Fig. 22).

MANAGEMENT

Most metatarsophalangeal joint injuries can be managed nonsurgically with insoles and strapping. More severe injuries may require long-term immobilization in a boot or cast.

Sesamoiditis

PRESENTATION

Mechanical overuse, stress fractures, osteochondritis, chondromalacia, and avascular necrosis of the hallucial

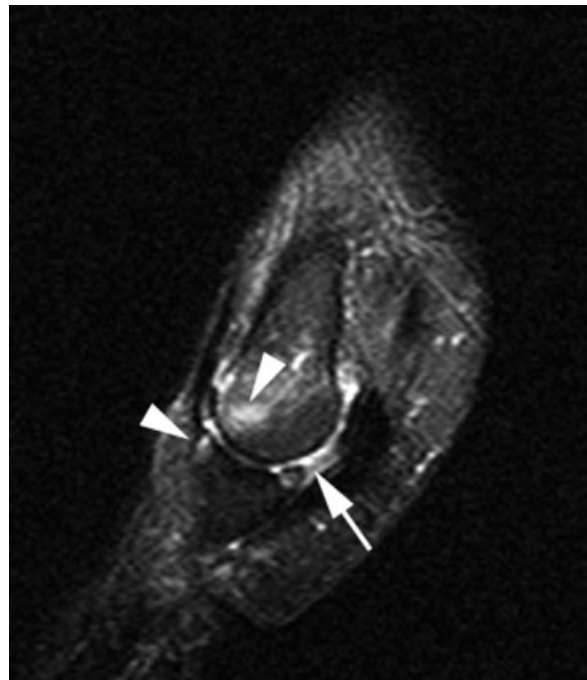


Figure 22 Turf toe. Sagittal short tau inversion recovery sequence showing a tear of the plantar plate (arrow), with bone edema on both sides of the metatarsophalangeal (arrowheads) joint.

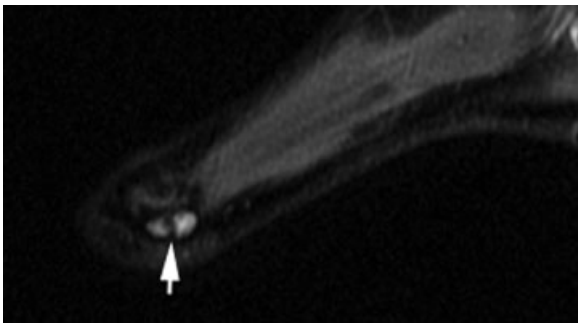


Figure 23 Sesamoiditis. Sagittal short tau inversion recovery sequence demonstrating a medial sesamoid fracture with associated bone edema (arrow). Notice slight malalignment of the fracture.

sesamoid bones belong to the same pathological spectrum, covered by the term *sesamoiditis*. The onset may follow repeated forefoot loading or acute trauma. Symptoms are more common in the medial sesamoid and in bipartite sesamoids, which occur in up to 33% of the population.⁴⁴ On examination the pain is reproduced on weightbearing tiptoe activities, the FHL tendon is often tight, and movement of the first MTP is painful and restricted.

ANATOMY AND IMAGING

The hallux sesamoids are embedded within the medial and lateral slips of the flexor hallucis brevis tendon. The sesamoids absorb most of the weightbearing force of the medial forefoot. On radiographs the involved sesamoid may be sclerotic and fragmented, but there may be no obvious abnormality. Bipartite sesamoids can be difficult to differentiate from sesamoid fractures on all modalities. Fractures typically have sharp, uncorticated margins (Fig. 23). On MRI, painful sesamoids typically demonstrate bone edema on fluid-sensitive sequences. Osteonecrosis of the hallux sesamoids may occur secondary to fracture, dislocation, infection, or repetitive microtrauma. On MRI there is low signal change on all sequences.⁴⁵

MANAGEMENT

The mainstay of management is to offload the sesamoid and to use orthotics. Ultrasound-guided steroid injection may help. Surgery should be reserved for recalcitrant cases because it may affect the biomechanics of the forefoot and can predispose to hallux valgus. Excision of both sesamoids invariably results in claw toe deformity and should be avoided.

CONCLUSION

Overuse injuries of the foot and ankle often cause pain and debility. Imaging plays a key role in the assessment, understanding, and management of these conditions. A good knowledge of the spectrum of overuse injuries is

essential to ensure appropriate investigation and subsequent early treatment.

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