An Overview of Hindfoot Pain and MRI Findings

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Heel pain is a common complaint among the general population and is present in 1 of 8 individuals, particularly those over age 50. Additionally, heel pain is one of the most prevalent complaints necessitating referral to a foot specialist. There are multiple etiologies for heel pain, which generally originates from 6 major anatomic structures: the plantar fascia, calcaneus, tarsal tunnel, tendons, bursae, and plantar fat pad.

This article will review the differential diagnosis for hindfoot pain as well as discuss pertinent MRI findings for each condition.

Plantar Fascia

The plantar fascia is a fibrous aponeurosis that arises along the medial calcaneal tuberosity. From the calcaneus, the plantar fascia divides into the medial, central, and lateral components (Figure 1).

The central band is the largest, adhering to the undersurface of the flexor digitorum brevis. At the midsole, the central band divides into 5 superficial and deep components extending toward the digits as the flexor tendons. This, along with the medial and lateral marginal superficial tracts, inserts onto each proximal phalanx. The plantar fascia plays a significant role in longitudinal arch support.

Plantar Fasciitis

Plantar fasciitis is one of the more common causes of plantar heel pain. Plantar fasciitis is related to microtrauma at the os calcis attachment and can result from repetitive trauma, enthesopathy, pes planus, pes cavus, or heel cord contractures. Stress-related trauma is the most common etiology and usually affects obese middle-age or elderly patients. Imaging-related findings for fasciitis include thickening of the plantar fascia (> 6 mm) at the proximal...
attachment and high-signal intensity on T2-weighted sequences with low to intermediate signal on T1-weighted sequences (Figure 2).6

**Plantar Fascia Tear**

A fascial tear is usually traumatic in etiology with sudden onset and localized tenderness. Fascial tears are commonly seen in running or jumping athletes.7 Imaging findings of acute tears will demonstrate a partial or full-thickness defect of the fascia with focal hyperintense signal on T2-weighted or short tau inversion recovery (STIR) sequences.7 Peripheral fluid-like signal can also be seen (Figure 3).

**Plantar Fibromatosis**

Plantar fibromatosis (Ledderhose disease) is a fibroproliferative disorder in which benign fibrous nodules develop within the plantar fascia.8 Plantar fibromatosis can be associated with many other fibroproliferative disorders such as Dupuytren disease and Peyronie disease.9 Plantar fibromatosis usually involves the more distal fascia and the central or medial bands. Imaging findings of plantar fibromatosis show nodular-thickening of the non-weight-bearing portions of the plantar fascia, which is hypointense on both T1- and proton density (PD)-weighted sequences (Figure 4). Hyperintense signal of the adjacent subcutaneous soft-tissues on T2- or PD-weighted sequences can also be seen.

There are multiple treatment options for plantar fibromatosis, which include surgical resection, radiation, and chemotherapy, used alone or in combination. The treatment is determined based on disease aggressiveness, patient age, and the risk of disability with resection. Surgical resection is the most common treatment; however, recurrence rates are high and can be deforming, even requiring amputation in some cases. If there is a possibility of functional loss, marginal excision and postoperative radiation therapy can be used. In the case of severe neurovascular or significant limb involvement, chemotherapy and radiation are often the sole option for treatment. Chemotherapy alone can also be used in young children to avoid disfiguring surgery and complications of radiation.8

**Calcaneus**

The calcaneus is responsible for significant axial load-bearing forces and is the most commonly fractured tarsal bone, responsible for up to 60% of all tarsal bone fractures in adults.10

**Calcaneal Stress Fracture**

Most stress fractures result from repetitive activity as opposed to direct trauma. Thus, calcaneal stress fractures are common in patients undergoing a
new occupation or repetitive motions (ie, military recruits or runners). Stress fractures are further classified as fatigue fracture (overuse in normal bone) or insufficiency fracture (normal use in abnormal bone).2 Conditions related to insufficiency fractures include those that weaken the bone integrity such as metabolic disorders, inflammatory conditions, bone dysplasias, and neurological disorders.11

Calcaneal stress fractures are a cause of hindfoot pain that is commonly not visualized or radiographically occult, especially in the early stages. Reported radiographic sensitivity for the diagnosis of lower extremity stress fractures ranges from 12% to 56% with a specificity of 88% to 96%. MRI sensitivity and specificity for detecting stress fracture can be as high as 99% and 97%, respectively.12 MRI demonstrates linear hypointense signal in bone marrow on T1-weighted images, which extends to the cortex with surrounding increased marrow signal on T2-weighted and STIR sequences (Figure 5).13 Calcaneal stress fractures are more common in the posterior calcaneus.

Tarsal Tunnel and Nerve Entrapment

The tarsal tunnel is a fibro-osseous canal in the medial aspect of the ankle, which is a common location for compression and entrapment of neurovascular structures. The tarsal tunnel contains the tibialis posterior (TP) tendon, flexor digitorum longus (FDL) tendon, posterior tibial artery/vein, and tibial nerve (yellow bracket), and retinaculum (arrow). Also present are the peroneus longus (L) and brevis (B) tendons in the lateral ankle.

FIGURE 6. Normal tarsal tunnel anatomy. Axial T2-weighted image demonstrating normal tarsal tunnel anatomy including the tibialis posterior (T), flexor digitorum longus (D), flexor hallucis longus (H), posterior tibial artery, posterior tibial vein, and tibial nerve (yellow bracket), and retinaculum (arrow). Also present are the peroneus longus (L) and brevis (B) tendons in the lateral ankle.

FIGURE 7. Varicosities resulting in tarsal tunnel syndrome. Axial PD-weighted fat-saturated image of the ankle with multiple venous varicosities (thin arrows) within the tarsal tunnel, which can result in tarsal tunnel syndrome. Tibialis posterior (T), flexor digitorum longus (D), flexor hallucis longus (H), and the overlying fibro-osseous tunnel forming the tarsal tunnel (thick arrow) are noted.

FIGURE 8. Aneurysmal bone cyst extending into tarsal tunnel. PD-weighted axial image of the ankle with an expansile mass of the talus (solid arrow) with fluid-fluid levels approaching the tarsal tunnel (dashed arrow) causing mass effect and edema (arrowhead).

Baxter Neuropathy

Baxter neuropathy is a syndrome caused by the entrapment of the inferior calcaneal nerve.15 The inferior calcaneal nerve is the first branch of the lateral calcaneal nerve, which if entrapped, can result in chronic heel pain often mimicking plantar fasciitis. Entrapment of the inferior calcaneal nerve occurs at 3 locations in the hindfoot: the medial border of the quadratus plantae...
muscle, along the fascial edge of an enlarged/hypertrophied abductor hallucis muscle, and most commonly at the medial calcaneal tuberosity. Imaging findings are usually related to denervation with increased signal intensity or atrophy of the intrinsic muscles of the foot. Incidental atrophy of the abductor digiti minimi likely reflects a prior clinically missed entrapment (Figure 9) and is not an uncommon finding.16,17

**Tendons**

Tendons normally have a homogeneous hypointense signal on all MRI sequences within the hindfoot.18 The main tendons of the hindfoot include the peroneus longus, peroneus brevis, Achilles, posterior tibial, FDL, and FHL tendons.

**Achilles Tendon**

The Achilles tendon is formed by the commnion of the gastrocnemius

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**FIGURE 9.** Baxter neuropathy. Coronal PD-weighted image of the ankle demonstrating moderate to severe muscle atrophy with fatty infiltration of the abductor digiti minimi (long arrows) compared to the normal abductor hallucis (short arrow) and flexor digitorum brevis muscles (dashed arrow). Incidentally, the central bundle of the plantar fascia is also very thickened, consistent with fasciitis (asterisk).

**FIGURE 10.** Normal anterior linear signal in the Achilles tendon. Axial PD-weighted fat-saturated image of the Achilles tendon demonstrating normal anterior linear signal variations (arrows) that should not be mistaken for a tear/injury.

**FIGURE 11.** Achilles tendinosis. Sagittal STIR image of the ankle demonstrating diffuse thickening of the Achilles tendon (thin arrows) with intermediate intrasubstance signal (asterisk) consistent with non-insertional tendinosis. Fluid-like signal running parallel with, and anterior to, the Achilles tendon (thick arrow) represents inflammation of the paratenon (paratenonitis).

**FIGURE 12.** Achilles tendinosis. Sagittal STIR image of the ankle showing increased signal intensity of the distal Achilles tendon near the insertion on the calcaneus (arrows) representing insertional tendinosis. Longitudinal focus of signal intensity is also noted (arrowheads) representing a partial interstitial tear.

**FIGURE 13.** Achilles tendon disruption. Sagittal STIR image of the ankle demonstrating complete disruption of the Achilles tendon with increased signal of the tendon fibers (thick arrow). Retraction of the fibers is also noted (small arrows).

**FIGURE 14.** Posterior tibialis tendinosis and tenosynovitis. T2-weighted fat-suppressed image of the ankle with thickening of the posterior tibialis tendon (arrow) consistent with tendinosis and increased fluid with the tendon sheath (arrowhead) consistent with tenosynovitis.
and soleus muscles. The tendon inserts at the posterior calcaneus, os calcis. The Achilles tendon normally has a concave anterior and posterior convex contour and has low signal intensity on all sequences. Thickness of the tendon averages 6 mm.\textsuperscript{19,20} A common normal finding is linear or punctate increased signal intensity on low echo time (TE) sequences, usually more anterior within the tendon. This signal represents normal fascicular anatomy but can be mistaken for interstitial tears, therefore knowing this common appearance and location is imperative (Figure 10).\textsuperscript{21,22} Achilles tendinopathy or tendinosis can be insertional or mid-substance. Non-insertional tendinosis is usually acute in onset and often proximal to the retrocalcaneal bursa. This entity usually occurs in older individuals who are less active and overweight.\textsuperscript{23,24} Insertional tendinopathy results from repetitive trauma and micro tears which usually present with weight-bearing pain in less athletic or active individuals and more commonly associated with running and jumping.\textsuperscript{2,23,24} Imaging findings on MR will demonstrate focal or fusiform thickening with diffuse or linear low to intermediate signal on fluid sensitive sequences (Figures 11 and 12). The Achilles tendon is unique as it does not have a true synovial sheath. However, it does have a thin sheath-like structure surrounding the tendon that is separated from the tendon by a lubricating layer of mucopolysaccharides. This structure is called the paratenon. Similar to tenosynovitis, with overuse the paratenon can become inflamed and cause pain. This is called paratenonitis (Figure 11).

Achilles ruptures, or complete tears, usually occur from 25-40 years of age. Activities that require dorsiflexed position while running or jumping are at a greater risk. Complete tears will demonstrate a T2-hyperintense signal defect of the tendon (Figure 13). Partial or complete retraction of fibers can be seen, depending on the degree of tearing. Chronic tendon pathology may lack intrasubstance signal but can be diffuse or focally thickened.\textsuperscript{25}

Tibialis Posterior, Flexor Digitorum Longus, and Flexor Hallucis Longus Tendons

The FDL and FHL tendons course through a shallow groove in the posteromedial aspect of the talus and continue under the sustentaculum tali. On the plantar aspect of the heel, the FHL tendon crosses deep to the FDL tendon at the Master Knot of Henry, before their insertion on the base of the great toe and lesser distal phalanges, respectively.\textsuperscript{26} The TP tendon originates at the posterior tibia, fibula, and interosseous membrane. From its origin it courses along the deep posterior compartment of the lower leg, through the tarsal tunnel, under and around the medial malleolus and into its insertion at the plantar aspect of the navicular, cuneiforms, and metatarsal bases.\textsuperscript{27}

The TP, FDL, and FHL tendons are prone to tendonitis and tenosynovitis, which results in a painful postero-medial heel. These findings are more commonly seen in athletes performing repetitive forceful push off motion with
The forefoot. Tenosynovitis is demonstrated by increased fluid-like signal intensity on T2-weighted sequences distending the tendon sheath (Figures 14 and 15). However, it is important to note that fluid in the FHL tendon sheath may be considered physiologic if similar to the volume of intraarticular fluid, as these structures often communicate.

**Peroneus Longus and Brevis Tendons**

The peroneus longus and brevis tendons course along a groove posterior to the fibula in the lateral ankle and curve anteroinferiorly along the undersurface of the foot. The peroneus longus inserts on the medial cuneiform and the base of the first metatarsal. The peroneus brevis inserts on the base of the fifth metatarsal. The peroneal tendons are also susceptible to tendonitis and tenosynovitis. Repetitive acute tenosynovitis can result in fibrous scar formation in the tendinous sheath, known as stenosing tenosynovitis. Imaging findings of stenosing tenosynovitis will demonstrate an intermediate signal intensity rind surrounding the tendon on both the T1- and T2-weighted sequences (Figures 16 and 17).

**Bursae**

There are two bursae of the hindfoot lying near the insertion of the Achilles tendon to the calcaneus. The retrocalcaneal bursa is located between the Achilles tendon insertion and the calcaneus. The retroachilles bursa (or subcutaneous calcaneal bursa) is situated between the skin and the Achilles tendon. On MRI a normal retrocalcaneal bursa is usually present and measures < 6 mm in the transverse plane and 1 mm in the anterior-posterior dimension. When these bursae become inflamed, they can generally be seen as uninterrupted MRI fluid-like signal in the expected locations of the bursae.

**Haglund Syndrome**

A Haglund deformity is a prominent bursal bony projection of the calcaneus, which can be a normal anatomical structure or associated with other findings. Haglund syndrome is the result of both soft tissue and osseous abnormalities consisting of a Haglund deformity, insertional tendinopathy, and pre-Achilles and/or retrocalcaneal bursitis. This entity is commonly associated with low-back or high-heel shoes. The imaging findings of Haglund syndrome include a prominent posterosuperior tuberosity of the calcaneus with or without increased marrow signal on the fluid-sensitive sequences, fluid-like signal within the pre-Achilles or retro-Achilles bursa, increased signal on T2/STIR sequences in the Kager (pre-Achilles) fat pad, and insertional tendinopathy of the Achilles tendon (Figure 18).

**Retroachilles and Retrocalcaneal Bursitis**

Bursitis in the retroachilles and retrocalcaneal bursa is most commonly a manifestation of Achilles pathology but can also occur as a separate entity. One of the more common causes of retroachilles and retrocalcaneal bursitis is repetitive trauma or friction. Bursitis can also be seen in the setting of rheumatoid arthritis and seronegative spondyloarthropathies. The retrocalcaneal bursa should measure < 1-2 mm in antero-posterior dimension. If enlarged, it may represent disease, especially if surrounding edematous changes are present. The subcutaneous fat should be seen between the Achilles tendon and the skin. If this fat cannot be seen on MRI, a blister or retro-Achilles bursitis may be present.
In particular, retro-Achilles bursitis is distinguished by edematous changes without mass effect on the skin.²

**Plantar Fat Pad**

The heel fat pad is composed of elastic fibrous septae with closely packed fat cells that act as a shock-absorber for the heel. Several plantar fat pad pathologies such as ulcers, abrasions and contusions can be identified with a detailed history and physical examination. However, the use of MRI is sometimes required to provide a differential diagnosis for heel pad abnormalities that cannot be explained clinically. Numerous causes of heel pain can arise from the fat pad, including infection, trauma (rupturing of the septa), neoplasm, inflammatory conditions, and rheumatoid nodules.

Rheumatoid nodules of the heel pad occur in 20% of patients who test positive for rheumatoid factor.² Rheumatoid nodules usually develop on the pressure areas in the heel, but may occur near the Achilles tendon insertion. Imaging findings are related to their histologic composition. Solid nodules are composed of chronic inflammatory cells and usually show decreased signal intensity on T1- and T2-weighted sequences with postcontrast enhancement. Nodules can have central necrosis with increased signal intensity on T2-weighted sequences and peripheral enhancement (Figure 19).³¹

**Conclusion**

Heel pain is a common musculoskeletal complaint for presentation to primary care or a foot specialist, and the specific etiology is often difficult to ascertain clinically. The heel is a complex area to assess with numerous sources of pain. However, compartmentalizing the heel into different anatomic structures and understanding the imaging findings and differential diagnoses for each location will help guide the clinician to a more accurate diagnosis and earlier treatment.

**REFERENCES**