MRI of Heel Pain

OBJECTIVE. The purpose of this article is to review the normal anatomy of the posterior ankle and hindfoot and review the causes of heel pain, with attention to the clinical, radiographic, and MRI findings.

CONCLUSION. Heel pain is a common problem that may be due to a variety of soft-tissue and osseous abnormalities. Knowledge of the anatomy of the posterior ankle and hindfoot offers a useful way to approach diagnosing the cause of the heel pain: osseous, ligamentous, tendinous, and other soft-tissue structures. Some of the more common causes include Achilles tendinosis, Haglund phenomenon, and plantar fasciitis. MRI offers superior soft-tissue contrast resolution and can be helpful in diagnosis as well as in presurgical planning.

Heel pain is a common problem that may be due to a variety of soft-tissue and osseous abnormalities. Knowledge of the anatomy of the posterior ankle and hindfoot offers a useful way to approach diagnosing the cause of the heel pain: osseous, ligamentous, tendinous, and other soft-tissue structures. Some of the more common causes include Achilles tendinosis, Haglund phenomenon, and plantar fasciitis. MRI offers superior soft-tissue contrast resolution and can be helpful in diagnosis as well as in presurgical planning.

Radiographs are often obtained to exclude acute causes of heel pain, such as calcaneal fracture; this has been well discussed in other articles and will not be further addressed here [2]. Some other causes of heel pain have characteristic radiographic findings, whereas other causes have nonspecific findings. These will be discussed in this article.

MRI allows superior soft-tissue contrast resolution, is noninvasive, and is widely available. It can be very helpful in equivocal cases of heel pain. Additionally, some causes of heel pain require surgical intervention (such as an Achilles tendon tear), and the information gleaned from MRI can influence presurgical planning. In this article, we first review the normal anatomy of the posterior ankle and hindfoot and then review the causes of heel pain, with attention to the clinical, radiographic, and MRI findings.

Anatomy

Three ligamentous groups support the ankle: the syndesmotic ligament complex, the lateral collateral ligament, and the deltoid ligament. The syndesmotic ligament complex is composed of the anterior and posterior tibiofibular (also known as the anterior inferior and posterior inferior tibiofibular) and interosseous ligaments. The lateral collateral ligament can be subdivided into the anterior talofibular, posterior talofibular, and calcaneofibular ligaments. The deltoid ligament contains the anterior and posterior tibiotalar, tibiocalcaneal, and tibionavicular ligaments.
These ligaments, as is true of ligaments throughout the body, appear as thin linear hypo-intense structures on both T1- and T2-weighted images. Interposition of fat between the ligaments can be seen in normal individuals [3]. The ligaments can routinely be evaluated on three-plane MRI with a slice thickness of 3 mm or less.

Several ankle tendons are present in the posterior ankle and hindfoot. These include the Achilles, posterior tibial, flexor digitorum longus, flexor hallucis longus, and peroneal tendons. Pathology of these tendons has been well described in the radiology literature [3]. We will discuss the most common tendon to be diseased: the Achilles tendon. The Achilles tendon is formed by the junction of the medial and lateral heads of the gastrocnemius tendons joining with the soleus tendon. The Achilles tendon lacks a tendon sheath but is instead surrounded by a peritenon (connective tissue also called the paratenon), which is composed of visceral and parietal layers. The vascular system of the peritenon extends within and outside of the Achilles tendon. Importantly, 2–6 cm proximal to the calcaneal insertion there is an area of diminished blood supply to the tendon, known as the “critical zone.” The poor vascular supply to this area results in slower tendon repair. Subsequently, this is where most Achilles tendon disease occurs [4]. The insertion site of the Achilles is an enthesis and is intimately related to the retrocalcaneal bursa [5]. Posterior to the tendon, there may be an acquired or adventitial bursa, known as the “retro-Achilles bursa.”

The ankle tendons appear as hypointense linear structures on T1- and T2-weighted images. T2-weighted images are helpful for showing intratendinous or paratendinous edema, the hallmark of tendinitis. It is important to note the magic angle effect, which occurs at approximately 55° with the main magnetic vector (B0) in sequences with TEs less than approximately 35 milliseconds and may produce increased signal within normal structures [6]. Careful analysis of the tendon in question in multiple planes and evaluation with prolonged TE sequences (T2-weighted) will help avoid this pitfall. The Achilles tendon has a normal thickness of 6 mm [4]. On axial images, the anterior margin of the Achilles tendon should be concave for most of its course.

The retrocalcaneal bursa should measure less than 1–2 mm in anteroposterior dimension [7]. 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 [4].

The plantar aponeurosis consists of medial, central, and lateral bands. The plantar fascia represents the thickest central band. The plantar fascia is adherent to the underlying flexor digitorum brevis muscle. The plantar fascia originates from the medial calcaneal tuberosity and splits into five bands that insert onto each...
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Osseous anatomy is primarily defined by the calcaneus. The calcaneus is the largest tarsal bone and is designed to bear the weight of the body. Traction trabeculae radiate from the inferior cortex, and compression trabeculae radiate to the anterior and posterior articular facets. The lateral surface of the calcaneus is flat with a central peroneal tubercle where the calcaneofibular ligament attaches. The lateral talocalcaneal ligament attaches anterosuperior to the peroneal tubercle. Medially, the intersosseous and medial talocalcaneal ligaments hold the talus and calcaneus in close apposition. The plantar aponeurosis inserts on the calcaneus along the anterior and plantar aspect. The Achilles tendon inserts along the middle portion of the posterior aspect of the calcaneus.

The calcaneus usually ossifies by the third month of gestation. There is a normal variant in which the calcaneus develops from two ossification centers with an apophysis developing along the posteriorinferior aspect of the calcaneus. This apophysis begins ossification at 4–7 years in girls and 7–10 years in boys. It fuses with the calcaneus by 12–15 years [9]. This can be important to remember in pediatric patients presenting with heel pain.

Causes of Heel Pain

Achilles Tendinosis

Achilles tendinosis is often the common precursor by which most Achilles tendon disease stems and refers to intratendinous degeneration by one of four main mechanisms: hypoxic-fibromatosis, myxoid, lipid, and osseous-calcific [10]. Hypoxic degeneration is the most frequent type seen in ruptured Achilles tendons and is thought likely secondary to the relative hypox-

Fig. 3—60-year-old man with acute onset of posterior heel pain while playing tennis. A, Sagittal T2-weighted image with fat-saturation shows complete discontinuity of distal Achilles tendon (arrowheads), consistent with complete Achilles tendon tear with tendon retraction. Note associated increased T2 signal intensity, which likely represents combination of edema and hemorrhage. B, Axial proton-density image shows residual plantaris tendon (arrow).
vascularity of the 2–6 cm area of the tendon that lies proximal to the calcaneal insertion, known as the “watershed area” or “critical zone” [11, 12]. This type of degeneration often follows multiple symptomatic episodes [12]. Axial MRI shows fusiform thickening of the tendon with loss of the normal concave anterior margin (Fig. 1) but often lacks the internal increased signal intensity seen with other types of tendinosis [4].

The most common asymptomatic type of degeneration is myxoid, whereby mucoid patches and vacuoles intersperse between thinned tendon fibers [13]. These vacuoles then coalesce, resulting in the formation of interstitial tears along the long axis of the tendon. Because of the asymptomatic nature of myxoid degeneration, it is not rare for patients to show an Achilles tendon tear at initial presentation. On MRI, mucoid deposits will show linear areas of increased signal on T1-weighted and proton density sequences. T2-weighted and STIR imaging will show interrupted and irregular areas of increased signal within the tendon [4].

Lipoid degeneration results in fatty deposits within the tendon and is the most age dependent of the four types of degeneration [14]. Interestingly, lipoid degeneration does not alter the structural properties of the tendon and thus, does not predispose the tendon to tear [15, 16]. However, lipoid degeneration is related to xanthofibromatosis, which often shows nodular thickening of the Achilles tendon with low to intermediate signal on all MRI sequences but enhances after the administration of gadolinium [17].

The fourth type of degeneration resulting in Achilles tendinosis is calcific tendinopathy. Although rare, dystrophic calcification of the tendon can progress to the formation of cortical bone and trabeculae within the affected area of the tendon, which distinguishes this type of degeneration on radiography, CT, and MRI [18].

**Achilles Tendon Tear**

Tears of the Achilles tendon often occur in middle-aged men between 30 and 50 years old who engage in leisure athletic activities that involve concentric loading, such as basketball and tennis, resulting in indirect trauma to the tendon [19]. Although most of these types of tears occur in the watershed region of the tendon, more proximal tears at the myotendinous junction can also occur, which are more common in younger men and can result from direct trauma [20]. Systemic diseases, such as rheumatoid arthritis, gout, lupus, and diabetes mellitus as well as fluoroquinolone use have also been implicated in tears of the Achilles [21–23].

In terms of tear severity, the spectrum includes microtears, interstitial tears, partial tears, and complete tears or ruptures [24]. Microtears are not visible on imaging but are often the inciting event that leads to tendinosis [25]. Increased signal on T2-weighted sequences is the MRI finding common to interstitial, partial, and complete tears [26].

Interstitial tears are often the sequelae of myxoid degeneration of the tendon as previously described and are longitudinally oriented [4]. On MRI, linear areas of increased signal are seen on T1-weighted, proton density, and fluid-sensitive sequences, but the surrounding fibers are intact. Treatment involves surgical débridement of the mucoid degenerative center with oversewing of the peripheral fibers of the tendon [27].

Partial tears show heterogeneous high signal intensity on fluid-sensitive MRI sequences, and there is incomplete interruption of the tendon fibers (Fig. 2). Involved fibers can partially retract and display a frayed or cork-screw appearance. Complete tears are identified by their high signal intensity fluid-filled tendonous gap, with either distracted or overlapping fibers (Figs. 3 and 4). Intratendinous or peritendinous edema and hemorrhage are often present in the acute setting [23].

Surgical repair of partial and complete tears is determined using the four-stage Kuwada classification system, which is based primarily on the size of the gap between the retracted ends of the torn tendon: type I, partial tear involving 50% or less of the tendon; type II, complete tear with a gap of less than 3 cm;
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Type III, complete tear with a gap of 3–6 cm; and type IV, complete tear with a gap of greater than 6 cm. Type I and II tears are typically repaired with end-to-end anastomosis, whereas an autogenous tendon graft flap is used for type III tears. Type IV tears usually require a free tendon graft or synthetic graft [28].

Chronic tears can result in varying degrees of muscle atrophy, which can be reversible or irreversible [29]. Reversible atrophy is often seen in the acute setting and is characterized by diffuse edema noted throughout the muscle [30]. Over time, the muscle takes on a more fatty infiltrated appearance, which often signals irreversible atrophy [31]. Of note, the soleus is more susceptible to atrophy than the gastrocnemius secondary to its greater proportion of slow twitch type I fibers [32]. Therefore, imaging protocols should include sagittal views of the distal soleus when evaluating for Achilles tendon injury.

Achilles Peritendinitis and Paratendinitis

Achilles peritendinitis refers to inflammation of the connective tissue peritenon that surrounds the tendon. Clinically, this entity is similar to synovitis or tenosynovitis that is seen in sheathed tendons. MRI shows high T2 signal intensity around the tendon, which is partially circumferential and is most evident along the posterior surface of the tendon (Figs. 5 and 6). However, given that the Achilles tendon lacks a true synovial membrane, the high T2 signal intensity is not as bright as that seen with synovial fluid [4].

A related entity is Achilles paratendinitis, which, although the term is often used interchangeably with peritendinitis, more accurately describes inflammation of the pre-Achilles (Kager) fat pad. Fat-suppressed fluid-sensitive sequences show increased signal and edema with irregularity of the fat pad anterior to the tendon. T1-weighted images show replacement of the normal high-signal-intensity fat (Fig. 7). Both peritendinitis and paratendinitis can be found in conjunction with tendinosis but may also be isolated findings.

Retrocalcaneal Bursitis

Another common cause of posterior heel pain is retrocalcaneal bursitis, which can be a manifestation of Achilles tendinosis but can also be a separate entity. Repetitive trauma is the most common cause and is frequently found in runners. Other causes include rheumatoid arthritis and seronegative spondyloarthropathies [33]. Treatment is usually conservative, although corticosteroid injection is sometimes
used [34]. However, caution should be exercised if there is coexisting Achilles tendinosis because steroids can further weaken an already at-risk tendon, making the tendon more susceptible to tear or rupture [35, 36].

Radiography can show obliteration of the normal retrocalcaneal fat by the enlarged fluid-filled bursa (Fig. 8A). On MRI, a fluid collection between the posterior calcaneus and the insertion of the Achilles tendon can be seen, which shows low signal intensity on T1-weighted sequences and high signal intensity on T2-weighted and STIR sequences (Figs. 8B and 9). Enlargement of the normal bursal sac is the hallmark MRI finding, with values greater than 7 mm cranio-caudal, 11 mm transverse, and 1 mm anteroposterior considered abnormal [7]. Additionally, adjacent soft-tissue inflammatory changes are common.

**Insertional Tendinosis of the Achilles Tendon**

Insertional Achilles tendinosis is one of the main causes of posterior heel pain [37]. Generally, this condition is secondary to chronic...
repetitive trauma with associated microtears due to excessive use of the calf muscles. This condition is most often seen in ballet dancers, runners, and athletes who participate in jumping sports. Additionally, systemic conditions, such as rheumatoid arthritis and seronegative spondyloarthopathies, are also associated with the condition [37]. Inflammation of the peritenon may precede or be associated with insertional Achilles tendinosis. Insertional Achilles tendinosis is also frequently associated with Haglund deformity of the calcaneus.

MRI shows thickening of the tendon at its insertion, with loss of the normal concavity of the anterior margin of the tendon [37, 38]. Intratendinous areas of increased signal may be seen with some edema of the adjacent soft tissue and possibly the adjacent calcaneal tissue at the level of the tendon insertion (Fig. 9).

**Haglund Deformity and Syndrome**

Haglund syndrome, first described by Patrick Haglund in 1927, is not an uncommon cause of posterior heel pain and consists of a constellation of soft-tissue and osseous abnormalities. The Haglund deformity is defined by the presence of a prominent bursal bony projection of the calcaneus, which is generally associated with wearing low-back shoes. The bony deformity, along with the shoes in question, may lead to a mechanically induced inflammation of the superficial bursa, Achilles tendinosis, and retrocalcaneal bursitis. These inflammatory changes in the presence of the bony deformity are together known as Haglund syndrome [9, 39]. Hindfoot varus and pes cavus are predisposing factors [40]. Most important is the presence of chronic stress.

On radiographs, the Haglund deformity is manifested by a bony projection along the superior posterior aspect of the calcaneal tuberosity on the lateral view (the radiographic equivalent of the “pump bump”). Diagnosing an enlarged calcaneal tuberosity is accomplished by drawing parallel pitch lines along the superior and inferior aspects of the calcaneus. The lower pitch line is tangent to the anterior tubercle. The upper pitch line is drawn parallel to the lower pitch line and at the level of the subtalar articular facet (Fig. 10). The calcaneal tuberosity is enlarged if it extends superior to the upper pitch line [41]. Although radiography cannot be reliably used to diagnose Haglund syndrome, loss of the normal radiolucent retrocalcaneal recess is important to note because it indicates retrocalcaneal bursitis [1].

MRI may be required for ambiguous or clinically equivocal cases. If present, a bony bump along the superior posterior corner of the calcaneal tuberosity is well visualized on sagittal T1-weighted images (Fig. 11). T2-weighted images will show excessive fluid in the retrocalcaneal bursa and fluid in the retro-Achilles bursa [4]. The presence of bone marrow edema in the calcaneal tuberosity supports the theory of the condition being caused by chronic repetitive mechanical compression and inflammation [42].

**Plantar Fasciitis**

Plantar fasciitis is the most common cause of inferior heel pain [43]. Plantar fasciitis is most commonly due to repetitive mechanical stress, specifically, prolonged pronation stresses. This produces microtears and inflammation of the fascia and perifascial soft tissues. The condition is commonly seen in runners and obese patients [44]. Additionally, it may be a result of an enthesopathy in association with seronegative spondyloarthopathies, such as ankylosing spondylitis, Reiter syndrome, or psoriatic arthritis [1]. Lateral radiographs often show a calcaneal spur, a very nonspecific finding [3].

MRI findings include plantar fascial thickening, intrafascial edema on T2-weighted images, edema surrounding the fascia on T2-
weighted images (primarily that the proximal aspect of the fascia is edematous), and increased infrasacral T1 signal [44] (Fig. 12). Fascial thickening is defined as greater than 3 mm, with some cases thickening to 7–8 mm [3]. The thickening is usually fusiform as opposed to the focal nodular thickening of the fascia seen in plantar fibromatosis. In addition, the nodular thickening of plantar fibromatosis occurs distal to the calcaneal insertion at the level of the midfoot. Furthermore, marrow edema in the calcaneal tuberosity may be present [45].

Tear of the Plantar Fascia

Rupture of the plantar fascia is typically a sports-related injury that is generally seen in athletes engaged in running and jumping [46]. Additionally, it can be seen in patients with chronic plantar fasciitis receiving local corticosteroid injections [47]. Most cases of plantar fascia rupture involve the proximal fascia near its calcaneal insertion—similar to the local edema seen in plantar fasciitis. Distal (anterior) tears also may occur [37, 45].

Radiography has little role in diagnosing plantar fascia rupture aside from excluding other causes of plantar heel pain. MRI findings are the same as would be seen in other fascial or aponeurosis tears. Acute tears show partial or complete disruption of the normally low-signal-intensity fascia, with areas of edema and hemorrhage best seen on T2-weighted or STIR sequences [1] (Fig. 13). Perifascial fluid accumulation is often also seen. Furthermore, it is important to inspect the flexor digitorum brevis muscle because plantar fascia rupture is commonly associated with tears of the flexor digitorum brevis muscle [45]. Acute and subacute muscle tears show intramuscular areas of increased signal on T1- and T2-weighted images, representing bleeding and edema within the muscle [1]. Less commonly, strains of other plantar muscles may be present, such as the abductor hallucis or quadratus plantae muscles [45].
Calcaneal Stress Fracture

The calcaneus is the second most common site of fatigue stress fractures, after the metatarsal bones. Insufficiency stress fractures may be seen in patients with rheumatoid arthritis or neurologic disorders. Stress fractures generally involve the posterosuperior or posterior aspects of the calcaneus and are usually oriented vertically or perpendicular to the long axis of the calcaneus [1]. Patients with diabetes mellitus are at increased risk of calcaneal insufficiency fractures, particularly those involving avulsion of the posterior process of the calcaneus [48]. Stress fractures of the anterior two thirds of the calcaneus may be more frequent than previously thought [49].

Radiographs are insensitive for acute stress fractures, appearing normal in more than 70% of the cases in one series [2, 50]. Follow-up radiographs are diagnostic in only 50% of cases [51]. When abnormalities are present, a stress fracture will show incomplete linear sclerosis running orthogonal to the major trabeculae, best seen on lateral radiographs.

CT is occasionally used to diagnose calcaneal stress fractures because it will show disruption of the bony cortex with surrounding periostitis. The sensitivity of CT is higher than radiography but poorer than MRI and bone scanning.

MRI shows characteristic findings, including bandlike areas of low signal intensity in the medullary space, surrounding marrow space signal abnormalities with ill-defined areas of T1 hypointensity and T2 hyperintensity that represent medullary edema, and hemorrhage [1, 51, 52] (Figs 14 and 15). Periosteal callus formation starts shortly after the fracture and appears as a hypointense line running parallel to the cortex, which represents the elevated periosteum [48].

Sever Disease (Calcaneal Apophysitis)

Apophyseal injuries are unique to the immature skeleton; traction apophyseal injuries may occur acutely at the site of tendinous insertion. Chronic apophyseal injuries is caused by inflammatory changes at the site of tendinous insertion into a bony prominence. When this occurs at the calcaneal apophysis, the associated eponym is Sever disease, first described by J. W. Sever in 1912.

Several authors have argued that calcaneal apophysitis is primarily a clinical diagnosis [53, 54]. However, foot radiographs are often obtained to exclude other causes. Unfortunately, there are no pathognomonic radiographic findings of calcaneal apophysitis [55, 56]. Nonspecific radiographic features include increased density and greater fragmentation of the apophysis, which may also be seen in healthy children [55].

As might be expected, MRI will show edematous changes within the calcaneal apophysis, possibly extending into the adjacent calcaneal tuberosity (Fig. 16). Furthermore, in equivocal cases, MRI can be helpful in excluding other causes of heel pain.

Os Trigonum Syndrome

The posterolateral talus has a variety of anatomic variants, including a normal tubercle; enlargement of the lateral tubercle of the posterior process of the talus, known as the “Stieda process”; and a separate ossicle, known as an “os trignonum,” which may or may not be fused to the posterior process of the talus. The os trignonum is considered by some to represent a developmental anomaly analogous to a secondary ossification center because it is formed by enchondral ossification within a cartilaginous extension from the talus [57]. The mineralized os trignonum appears between the ages of 7 and 13 years and usually fuses with the talus within 1 year. In approximately 10% of patients, it remains as a separate ossicle [58].

Clinically, the os trignonum is asymptomatic unless impingement occurs; hence, os trignonum syndrome is also known as “posterior ankle impingement syndrome.” Other osseous abnormalities of the posterior talus may also result in posterior ankle impingement syndrome, such as the Steida process [59]. Strenuous activities that result in extreme plantar flexion can cause compression of the adjacent synovial and capsular tissue against the tibia [60]. Thus, the syndrome is often seen in athletes participating in ballet, football, and soccer.

On radiography, the normal os trignon generally shows smooth margins. Repetitive trauma to the os trignon may result in a fracture of the ossicle. On MRI, os trignon syndrome shows abnormal signal intensity in the lateral talus tubercle, the os trignon, or both because of the bone impaction (Fig. 17). Furthermore, inflammatory changes will generally be present in the soft tissues of the posterior ankle, especially in the posterior synovial recess of the subtalar and tibiotalar joints [59]. Inflammatory changes affecting the flexor hallucis longus tendon sheath may variably be present. However, it is important to remember that the flexor hallucis longus tendon sheath normally communicates with the tibiotalar joint in approximately 20% of normal individuals; therefore, fluid in the tendon sheath may be normal. Septations, contour irregularities, and abrupt cutoff of the fluid at the level of the posterior talus are helpful distinguishes in diagnosing tenosynovitis of the tendon sheath [61].

Conclusion

There are a variety of causes of heel pain, and we have reviewed the most common causes. We have primarily reviewed mechanical causes, by far the most common; however, it is important
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to remember that there may be other causes of heel pain, such as osteomyelitis, which will present with a much different clinical picture. Although clinical examination is often useful, imaging the hindfoot is helpful for solving equivocal cases as well as for preoperative planning by showing the exact location and extent of the pathology. MRI has superior soft-tissue contrast resolution to other modalities; therefore, it can usually provide a specific diagnosis.

References

47. Sellman JR. Plantar fascia rupture associated with corticosteroid injection. Foot Ankle Int 1994; 15:376–381
48. Kathol MH, el-Khoury GY, Moore TE, Marsh JL.

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