Diagnosis and Management of Heel and Plantar Foot Pain

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INTRODUCTION

Heel pain is a common complaint encountered by primary care physicians. Misdiagnosis is not uncommon because of the intricate anatomy of the heel, where many structures lie in close proximity to one another. While there are traumatic (high-energy), infectious, oncologic, vascular and systemic causes of heel pain, this article will focus on the most common ones: repetitive microtrauma and compression of structures within confined spaces. Pathologic causes can be broadly categorized as degenerative, neurologic, or traumatic.

PLANTAR FASCITIS

The plantar fascia is an aponeurosis of collagen fibers that originate from the anteromedial calcaneus, course distally on the plantar foot, and divide into five insertions on the proximal phalanx of each digit.1 The fascia helps maintain the arch of the foot and serves a dynamic function during the gait cycle.1 The most common cause of heel pain, plantar fasciitis is due to inflammation at the calcaneal origin,1,2 resulting in fascial degeneration and microtears.1

Approximately two million people, typically 40 to 60 years old, are treated annually in the United States for plantar heel pain.1,3 Their pain is typically worse with the first steps in the morning and after prolonged standing. It is usually stabbing, non-radiating, and not associated with neurologic symptoms.2 Risk factors for plantar fasciitis include running, flat foot deformity (Pes Planus), professions requiring prolonged standing, obesity, and limited ankle dorsiflexion.1

Patients generally have focal tenderness over the plantar-medial calcaneus1 that worsens with passive toe dorsiflexion and a calf raise. Tightness of the Achilles tendon is common. Imaging is not required for the diagnosis and is recommended only for patients who fail conservative management.

Nonsurgical treatment—successful in greater than 90% of cases—should be attempted for at least six months and may require up to 18 months.1 Conservative measures include non-steroidal anti-inflammatory drugs (NSAIDs), stretching exercises, physical therapy, orthoses (night splints, cast immobilization), cortisone injections, and extracorporeal shock wave therapy, all with varying success.1,2 If conservative therapy fails, surgery may be considered. Plantar fascial release, either open or endoscopic, is the procedure of choice, with success rates of 70-90%.2

HEEL PAD SYNDROME

The heel pad beneath the calcaneus consists of adipose tissue within fibrous septae and allows repetitive load bearing. It is less elastic in elderly and diabetic patients, which leads to inflammation and edema. Diabetic plantar tissue is stiffer than healthy tissue and has a lower capacity to withstand compressive and shear stresses. Heel pad atrophy is often contributory. This diagnosis is more common in obese patients due to higher loads.4

Achilles tendon disorders include a spectrum from chronic degenerative injuries to acute tendon ruptures. Treatment consists of NSAIDs and shoes with adequate heel padding. Corticosteroid injections are contraindicated as they can cause atrophy of the plantar fat.3 Surgery cannot restore the normal architecture of the heel pad. In addition, the plantar skin is prone to wound-healing problems.

ACHILLES TENDON DYSFUNCTION

Achilles tendon disorders include a spectrum from chronic degenerative injuries to acute tendon ruptures. The strongest and thickest tendon in the human body, it is the insertion of the gastrocnemius and soleus muscles onto the posterior calcaneal tuberosity.4 “Achilles tendinitis” is a misnomer as the tendon itself does not undergo inflammation.7 The tendon’s blood supply is provided by a paratenon, a single layer of cells encasing the structure,7 as well as by the musculotendinous junction.4 Plain radiographs can identify both calcific tendinous changes and Haglund deformities (discussed below). Ultrasound is safe, quick and effective but variable operator skills may limit its use in the community.7 MRI provides the most detailed information about the Achilles tendon and surrounding soft tissue and bony structures.7 One classification scheme divides Achilles tendon dysfunction into three categories: peritendinitis, tendinosis, and peritendinitis with tendinosis.9

Peritendinitis, also referred to as paratenonitis, is inflammation of the surrounding paratenon.7 Common in athletes due to poor-fitting shoes, it involves focal swelling, diffuse discomfort, and tenderness to palpation. Conservative therapy includes proper shoes, activity modification, rest, and NSAIDs.7 Surgery involves excision of the thickened paratenon, but is rarely required.10

Achilles tendinosis is a degenerative process related to aging and repetitive microtrauma and microtearing.7 Degeneration usually occurs in the hypovascular zone of the tendon, two to six centimeters proximal to the calcaneal insertion.7 Classically seen in middle-aged men with
increased activity levels, tendinosis occurs gradually. Patients present with pain and nodular thickening of the middle-third of the tendon.\textsuperscript{8} Sharp pain may indicate a partial tendon tear. If detected before tendon rupture, treatment is conservative: activity modification, orthotics, and physical therapy for eccentric strengthening and range of motion exercises.\textsuperscript{7} Corticosteroid injections may further weaken and rupture the tendon, so they are contraindicated.\textsuperscript{8} Surgery is needed in 25\% of cases, for debridement of the degenerative portion of the tendon.\textsuperscript{7}

**Retrocalcaneal Bursitis & Haglund Deformity**

The retrocalcaneal bursa is located between the posterosemiposterior aspect of the calcaneal tuberosity and the Achilles tendon. A Haglund deformity is a prominence of the posterosemiposterior lateral calcaneus. Due to these close anatomic relationships, retrocalcaneal bursitis can be associated with a Haglund deformity and insertional Achilles tenosynovitis.\textsuperscript{11}

Retrocalcaneal bursitis causes pain anterior to the Achilles tendon, just proximal to its insertion. In contrast, Haglund deformity tends to cause pain superolateral to the Achilles insertion. The most common cause is irritation caused by the shoe counter. It is seen in athletes, particularly those involved with uphill running, because ankle dorsiflexion compresses the bursa between the Achilles tendon and calcaneus. One should be wary of a systemic diagnosis, such as inflammatory arthritis, in a patient with bilateral symptoms.

Patients with retrocalcaneal bursitis typically have bogginess along the medial and lateral aspects of the Achilles tendon.\textsuperscript{12} Tenderness with the two-finger squeeze test (medial and lateral pressure anterior to the Achilles tendon above the calcaneus) is a classic finding.\textsuperscript{7} Pain may occur with passive ankle dorsiflexion and resisted ankle plantarflexion as these motions decrease the space available for the bursa.\textsuperscript{12}

Conservative treatment includes ice, activity modification, open-heeled shoes and NSAIDs. Corticosteroid injections should be avoided in the posterior heel as they increase the risk of Achilles tendon degeneration and rupture.\textsuperscript{13} Surgical procedures involve some combination of resection of the calcaneal prominence, retrocalcaneal bursectomy, and Achilles tendon debridement. Surgery is not always curative. In one series, symptoms were completely relieved in only 69.4\% of patients and 14.3\% became worse due to infection, sural nerve injury or painful scar formation.\textsuperscript{14}

**Tarsal Tunnel Syndrome**

The tarsal tunnel is a fibro-osseous space formed by the flexor retinaculum of the ankle, posterior and distal to the medial malleolus. Often compared to carpal tunnel syndrome, it is an entrapment neuropathy of the tibial nerve resulting in pain and paresthesias at the plantar aspect of the foot.\textsuperscript{15} The tarsal tunnel also contains the posterior tibial tendon, flexor digitorum longus tendon, flexor hallucis longus tendon, and the posterior tibial artery and vein. The most frequent causes of this neuropathy include trauma, space-occupying lesions, and foot deformity.\textsuperscript{15} Complainents are often non-specific and poorly localized, making the diagnosis difficult. Pain and paresthesias are intermittent or constant, and frequently associated with proximal or distal radiation. Symptoms worsen with prolonged standing and exercise and are relieved by elevation and rest. Night pain is also a relatively common complaint. Physical examination is often non-specific, but decreased sensation over the plantar foot and a positive Tinel’s sign (paresthesias with percussion over the nerve) are pathognomonic.\textsuperscript{15} Weakness is not common, but when present, indicates severe compression.\textsuperscript{15}

Weight-bearing radiographs evaluate foot deformity and traumatic injuries and

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**Heel pain flowchart.**

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MRI scans may reveal compressive soft tissue masses. Tibial motor and sensory nerve studies (latencies, amplitudes, conduction velocities) are critical in the diagnosis and accurate in 90% of cases. Compression occurs deep to the abductor hallucis where the nerve turns and courses laterally. Patients complain of medial heel pain four to five cm anterior to the posterior aspect of the heel, or just distal to the medial calcaneal tuberosity. Burning pain can radiate into the tarsal tunnel (posteromedial ankle) or distally toward the plantar lateral foot.

Treatment depends on the specific cause of the symptoms. For space-occupying lesions, surgery is often required but can be treacherous due to the close proximity to the neurovascular bundle. Patients with pes planus deformity that stretches the tibial nerve may benefit from custom orthotics. Inflammatory tenosynovitis causing tibial nerve compression may benefit from NSAIDs, rest, and immobilization in a walking boot or cast. Local corticosteroid injections may decrease inflammation, with careful avoidance of intra-tendinous and intra-vascular injections. Electrodiagnostic studies confirm the diagnosis and localize compression in the lateral plantar tunnel or more proximally in the tarsal tunnel, which would affect both the medial and lateral plantar nerves. Rest, ice and NSAIDs should be tried for at least six months. Surgical decompression involves releasing the nerve throughout its course along the medial heel by dissecting the fascia overlying the FDB and quadratus plantae.

BAXTER’S NERVE ENTRAPMENT

Baxter’s nerve is the first branch of the lateral plantar nerve. It courses deep to the abductor hallucis and flexor digitorum brevis (FDB) and superficial to the quadratus plantae along the medial calcaneus. This mixed nerve innervates the quadratus plantae, FDB, and abductor digiti quinti (ADQ); it also supplies sensation to the calcaneal periosteum, the long plantar ligament, and the lateral plantar skin. Compression occurs deep to the abductor hallucis where the nerve turns and courses laterally. Patients complain of medial heel pain four to five cm anterior to the posterior aspect of the heel, or just distal to the medial calcaneal tuberosity. Burning pain can radiate into the tarsal tunnel (posteromedial ankle) or distally toward the plantar lateral foot.

Hindfoot valgus, or pronation, and ankle plantarflexion (Achilles tendon contracture) can increase nerve compression. A recent cadaver study demonstrated that pressure in the lateral plantar tunnel is highest in pronation and plantarflexion. Tinel’s sign is positive if paresthesias are reproduced with tapping over the nerve beneath the abductor hallucis muscle. In chronic cases, patients may have diminished sensation in the lateral plantar foot. In cases of bilateral neurologic findings, spinal pathology and systemic diseases must be ruled out.

As this is primarily a clinical diagnosis, imaging is not helpful. Non-specific atrophy of the ADQ is present in 6.3% of all patients who have an MRI. Electrodiagnostic studies confirm the diagnosis and localize compression in the lateral plantar tunnel or more proximally in the tarsal tunnel, which would affect both the medial and lateral plantar nerves. Rest, ice and NSAIDs should be tried for at least six months. Surgical decompression involves releasing the nerve throughout its course along the medial heel by dissecting the fascia overlying the FDB and quadratus plantae.

CALCANEAL STRESS FRACTURE

The calcaneus consists primarily of less dense cancellous bone, which is susceptible to fractures. After the metatarsals, the calcaneus is the most common location for a stress fracture in the foot. It can occur in athletes participating in sports with repetitive axial loading as well as elderly patients with osteopenia. It is typically an overuse injury that coincides with an athlete changing to a more strenuous exercise regimen. Diffuse heel pain (medial and lateral) worsens with weight-bearing and may progress to being present at rest.

Figure 1. Heel pain anatomy.

**A** - Plantar Fasciitis  **D** - Tarsal Tunnel Syndrome  **G** - Haglund Deformity

**B** - Baxter’s Nerve Entrapment  **E** - Heel Pad Syndrome  **H** - Achilles Tendinitis

**C** - Calcaneal Stress Fracture  **F** - Achilles Tendinitis
The hallmark is a positive calcaneal squeeze test, or pain with compressing the medial and lateral calcaneus.\textsuperscript{23} Depending on the acuity of the injury, there may be swelling and ecchymosis. Radiographs (lateral view) may demonstrate an altered trabecular pattern, but are frequently interpreted as normal. Sclerosis perpendicular to the orientation of the trabeculae indicates a healing fracture.\textsuperscript{24} Persistent pain may necessitate an MRI or bone scan to detect an occult fracture. The former is preferable since it also evaluates soft tissue structures that can cause heel pain.\textsuperscript{24}

If history and physical examination suggest a calcaneal stress fracture but the diagnosis has not been confirmed, patients should decrease their activity level. If symptoms persist and/or the diagnosis is evident on imaging, patients should not bear weight and are placed in a short leg cast. Particularly in the high-level athlete, this treatment should be continued until tenderness resolves.

**Sever’s Disease**

Calcaneal apophysitis, or Sever’s disease, is a common cause of heel pain in the pediatric athlete.\textsuperscript{25} The calcaneal apophysitis is the site of insertion of the Achilles tendon. Causative theories include traction and compression of the growth plate, but there is no consensus.\textsuperscript{25} Patients have posterior heel pain that increases with activity. Examination reveals tenderness at the Achilles tendon insertion, heel cord tightness, and limited ankle dorsiflexion. Diagnosis is primarily clinical and the mainstay of treatment is conservative therapy with rest and NSAIDs. Severe cases may require a short period of immobilization. Both rehabilitation and preventative measures include gascrocneumius/soleus stretching and strengthening.\textsuperscript{25}

**Conclusion**

Heel pain is a fairly common condition due to a variety of etiologies. The appropriate diagnosis and treatment can be difficult because of various bones, nerves and connective tissues confined to small spaces and subjected to weight bearing. An algorithm (Figure 1) offers a logical diagnostic approach and Figure 2 describes the anatomical locations of various etiologies. Regardless of the cause, however, conservative therapies should be attempted for a reasonable period of time before any surgical intervention.

**References**


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