Managing the patient with heel pain

ABSTRACT

Heel pain presents frequently to primary care, commonly affecting athletic and elderly patients. Its presentation can be a common source of confusion for clinicians given the wide variety of differential diagnoses and the similarities in presenting symptoms and signs. This review classifies heel pain according to site of pain and explores the common pathologies clinicians may encounter. A brief summary of common imaging modalities used is provided. The literature is reviewed to guide evidence-based practice and to provide a framework to help clinicians investigate and manage heel pain before onward referral for specialist intervention. A linked article detailing the imaging of heel pain is included in this issue (https://doi.org/10.12968/hmed.2019.80.4.192).

Heel pain is a common presentation to primary care. Its incidence is highest in athletic and elderly populations (Menz et al, 2006). A structured approach is essential in managing such patients, in what can otherwise be a daunting experience given the wide variety of differential diagnoses.

This review discusses a framework for the clinician managing a patient with heel pain (*Figure 1*). Although anatomically the heel is the calcaneus, in clinical practice the term is used more loosely to refer to structures within and beneath the ankle complex. This article describes pathology according to site of heel pain, thus an accurate pain history is essential. Treatment options will be discussed, before briefly outlining findings on further imaging and surgical management – although much of this lies outside the scope of this article.

Inferior heel pain

Pain underneath the heel is the commonest presentation, seen in approximately 15% of adults (Rompe et al, 2007). The pain is often the result of mechanical causes and can be accurately localized – usually described along the plantar fascia but may extend into the midfoot.

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Plantar fasciitis

This is the commonest aetiology of inferior heel pain, affecting 15% of all patients suffering from foot pain (Shrier and Gossal, 2000). The thick plantar fascia extends from the medial calcaneal tuberosity, inserting into the plantar surfaces of the metatarsophalangeal joints, the proximal phalanges and the flexor tendon sheaths (*Figure 2*). It acts as a shock absorber and spring during the gait cycle, akin to a bowstring (Genova and Gross, 2000). It is taut on weightbearing and flexible when non-weightbearing. Hence patients describe plantar heel pain during the first few steps after resting, such as in the morning after waking, and relief through rest or gentle ambulation.

Plantar fasciitis is associated with overuse and overload (Dyck and Boyajian-O'Neill, 2004), thus risk factors include obesity or excessive athletic activity (Gill, 1997). It is frequently seen in middle-aged women (Hyland et al, 2006). Other risk factors include pes planus and pes cavus (flat and high-arched feet), and a tight or short Achilles tendon (Schwartz and Su, 2014).

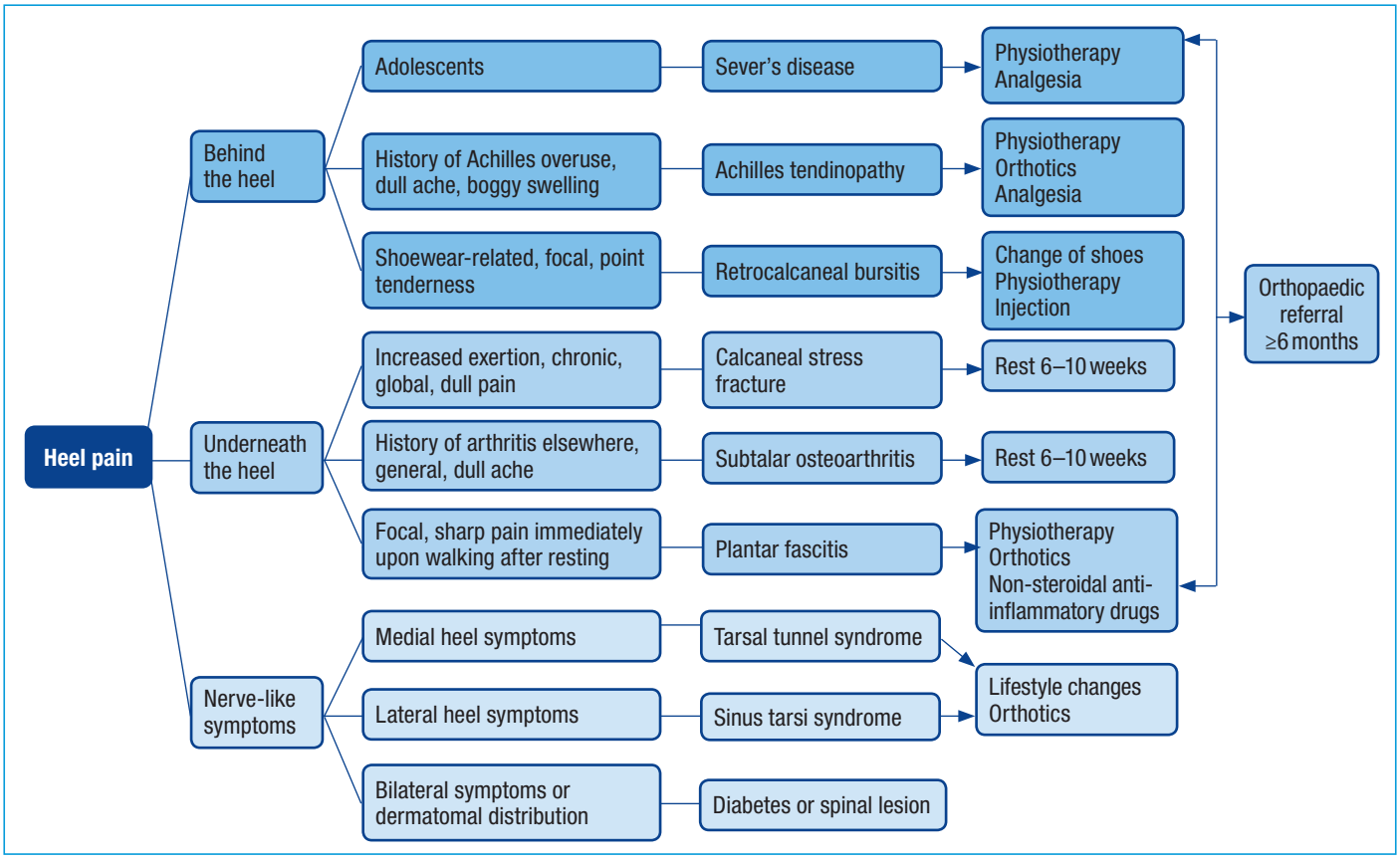
The chronic overload causes microtears in the fascia near its attachment to the calcaneus (Hyland et al, 2006). Repeated trauma during the heel strike phase of gait inhibits repair causing chronic degeneration with laying down of fibrous tissue and sometimes even ossification of its calcaneal attachment (Wearing et al, 2006; Kirkpatrick et al, 2017). Thus despite its name, plantar fasciitis is not an inflammatory process but rather chronic degeneration (it is now named plantar fasciosis or fasciopathy).

Weightbearing radiographs are generally not needed for diagnosis but if performed will show a heel spur in about half of patients suffering with plantar fasciosis. Ultrasound scans performed in patients with plantar fasciosis will show a thickened plantar fascia of >5 mm (McMillan et al, 2009). The presence of calcaneal heel spurs (*Figure 2*) alone does not signify plantar fasciosis, but the coexistence of both calcaneal heel spurs and plantar fascitis indicates greater chronicity of disease (Thomas et al, 2010) and is a risk factor for a longer recovery period. Asymptomatic calcaneal heel spurs are common and do not require referral to orthopaedics.

Examination reveals tenderness at its calcaneal insertion, with pain elicited when the plantar fascia is stressed such as dorsiflexing the great toe (Schwartz and Su, 2014). If a heel spur is present, it may be palpable eliciting tenderness in symptomatic patients.

A conservative approach to plantar heel pain reduces symptoms in 89% of patients after 1 year (Davis et al, 1994). Management is the same even if a calcaneal heel spur is present and should be trialled for at least 6 months

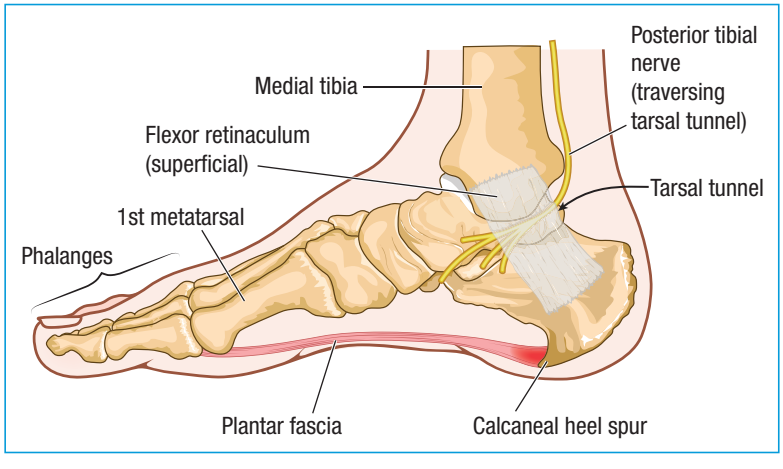
Figure 1. Framework for managing a patient with heel pain.



before referral to orthopaedics. Rest is advised initially, with non-steroidal anti-inflammatory drugs if needed (Hyland et al, 2006). Patients should be referred for physiotherapy. Exercises are targeted at lengthening the Achilles tendon and stretching the plantar fascia, performed three to five times per day. This is the most effective treatment for plantar heel pain (Shrier and Gossal, 2000). Physiotherapy taping of the medial arch or calcaneus is used to reduce the risk of recurrence disease (Hyland et al, 2006). Orthotic in-soles are usually also needed for patients with biomechanical risk factors, e.g. those with flat feet. Patients should be advised to avoid walking barefoot or in flat shoes (which can cause overpronation of the foot stressing the plantar fascia) and weight loss should be advised for those with a high body mass index (Butterworth et al, 2012). During an acute episode, a corticosteroid injection into the point of maximal tenderness can be a helpful adjunct (Ball et al, 2013), but should certainly not be the mainstay of treatment. An increased risk of rupture of the plantar fascia has been reported following a steroid injection; patients should be warned of this before the procedure (Hunt and Anderson, 2009).

If symptoms persist, a referral to orthotics can be considered (Whittaker et al, 2018). Night splints or using either a walking boot or below knee plaster of Paris to offload the fascia for 4–6 weeks can be trialled. This provides sufficient pain control in 90% of patients with persisting symptoms within 3 months (Thomas et al, 2010). Failure of this regimen necessitates referral to orthopaedics.

Figure 2. Medial view of the ankle.

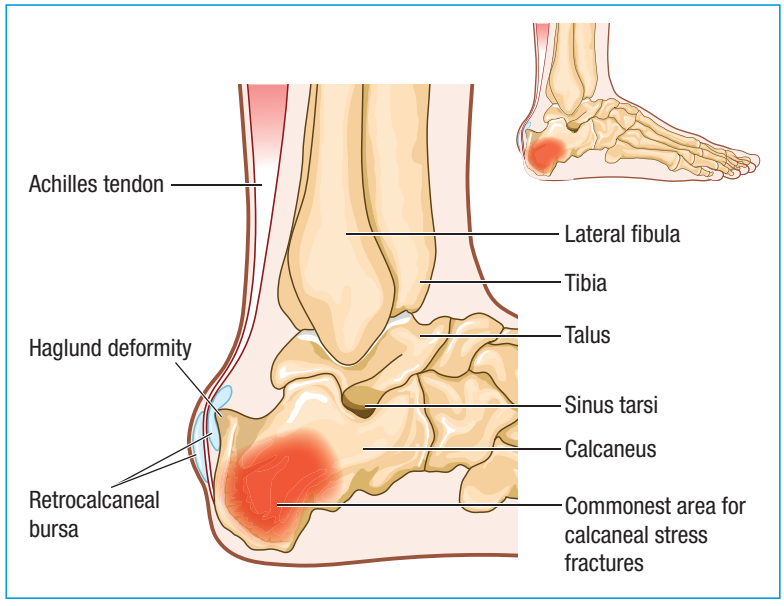


Surgical options include removal of symptomatic heel spurs if present. These can compress the inferior calcaneal nerve (arising from the lateral plantar nerve), or its calcaneal branch (Kirkpatrick et al, 2017). A fasciotomy performed surgically or by coblation using extracorporeal shockwaves may be considered (Thomas et al, 2010).

Calcaneal stress fractures

These are the second commonest stress fracture of the foot (Tu and Bytomski, 2011). Patients describe a dull ache over a period of weeks to months, initially occurring only

Figure 3. Lateral view of the ankle.



after exertion but soon becoming continuous throughout the day. A history of increased load placed across the foot, either through a sudden increase in activity or change in footwear (with less heel support), is almost always stated. The commonest area of fracture is posteroinferior to the subtalar facet joint (Thomas et al, 2010) (*Figure 3*).

Examination often reveals swelling and tenderness globally across the posterior calcaneus. Warmth and erythema is sometimes present, particularly if the bone has recently been strained by activity. The diagnosis is easily differentiated from plantar fasciitis as patients are also tender on mediolateral pressure of the calcaneus – 'squeeze test' (Hunt and Anderson, 2009) – which is absent in plantar fasciitis.

Radiography is usually negative initially but can occasionally show a sclerotic fracture line. Magnetic resonance imaging is a sensitive investigation and can be performed if clinical suspicion is high and radiographs are negative.

Management is almost always conservative; rest and avoidance of activities believed to be causing the pathology. Walking boots or use of cushioned in-soles can be used as adjuncts (Tu and Bytomski, 2011). In recalcitrant cases, complete non-weightbearing is advised, followed by a gradual return to full activity over a period of 6–10 weeks (Hunt and Anderson, 2009).

It should be noted that subtalar osteoarthritis produces symptoms very similar to calcaneal stress fractures – with inferior (and sometimes global) heel pain exacerbated by exertion and relieved by stress. Patients often have arthritis elsewhere. A history of bilateral disease should certainly raise suspicion for an arthritic cause. Active disease may likewise produce warmth, erythema and swelling over the calcaneus. Generalized tenderness is noted, sometimes with focal sharp tenderness over the subtalar area. Patients usually indicate pain over the affected area using the palm of the hand (indicating global disease) rather than with their fingers (indicating focal tenderness). Management is supportive and similar to management of arthritic disease elsewhere; analgesia and rest with lifestyle modification. In advanced cases, patients may be referred to orthopaedics for image-guided steroid injections into the subtalar joint. In severe disease, a subtalar fusion can be considered.

Posterior heel pain

Posterior heel pain is the second commonest presentation of foot pain to primary care (Thomas et al, 2010), and much of its pathology relates to the insertion of the Achilles tendon. Pain is described posterior to the calcaneus and can sometimes extend proximally to include part of the tendon, or even the muscle bulk itself. Many of these patients are successfully managed conservatively and the initial strategies used are similar. Anti-inflammatories should be prescribed with rest from activities exacerbating pain or activity modification suggested, as well as ice compression (Hunt and Anderson, 2009). Thereafter management should be geared towards the suspected cause.

Achilles tendinopathy

The Achilles tendon attaches the two strong plantarflexors to the calcaneus, thus great forces are transmitted along the tendon (*Figure 3*). The tendon depends upon its synovial sheath for blood supply which can occasionally not meet its demands. After prolonged relative ischaemia, collagen fibres within the tendon can begin to degrade and become inflamed (Murrell, 2002). Such mismatch in vascular supply and demand occurs in Achilles overuse or repetitive direct trauma – thus this is often seen in the active, in patients with prolonged Achilles activity (such as wearing high heels) (Tu and Bytomski, 2011) or patients with poorly fitting shoes. This chronic disease process can increase the risk of an acute Achilles tendon rupture (Agyekum and Ma, 2015).

Achilles tendinopathy presents as longstanding dull pain at the Achilles insertion into the calcaneus or in its distal third, associated with exertion and relieved with rest. Any activity recruiting the tendon is painful, thus patients struggle to climb stairs or walk on an incline.

Examination can reveal a boggy swelling of the distal portion of the tendon or sharp point tenderness centrally over the calcaneus at its insertion. Resisted plantarflexion is painful (Hunt and Anderson, 2009). Dorsiflexion beyond neutral is often limited as the Achilles tendon is usually tight in such patients (Hunt and Anderson, 2009); limited passive dorsiflexion beyond neutral is noted with the knee flexed (increased dorsiflexion would be expected) and with fully extended (the Silfverskiöld test). Ultrasound imaging can reveal a thickened tendon but magnetic resonance imaging is the gold standard for showing a degenerate tendon.

Reducing the tensile stress of the tendon should be attempted with heel raises or rocker sole shoes (Thomas et al, 2010). These patients benefit from physiotherapy (exercises to stretch the Achilles tendon). In more resistant cases, there is evidence for trialling extracorporeal shock wave therapy (Thomas et al, 2010) which may stimulate growth of new blood vessels. Corticosteroid injections should not

be trialled because of the increased risk of Achilles rupture post-injection (Tu and Bytomski, 2011). After a trial of at least 6 months of conservative treatment, patients may be referred to a foot and ankle surgeon for consideration of Achilles tendon debridement (from diseased or inflamed tissue) or for Achilles tendon lengthening procedures.

Retrocalcaneal bursitis

Repetitive frictional trauma, such as from shoes, can cause inflammation of the bursa between the Achilles tendon and the calcaneus or skin (Rio et al, 2015) (*Figure 3*). Patients describe acute, severe, sharp pain often related to footwear.

Examination reveals point tenderness, erythema and sometimes mild associated swelling. The pain is exacerbated by either dorsiflexion or plantarflexion as the tendon irritates the inflamed bursa (Agyekum and Ma, 2015).

In longstanding cases, radiographs may reveal a bony growth from the posterosuperior aspect of the calcaneum – a Haglund deformity or 'pump bump' (*Figure 3*) – usually idiopathic with an unclear pathophysiology (Vaishya et al, 2016; Jiménez Martín et al, 2017). Repetitive trauma from this can also be associated with recurrent cases of retrocalcaneal bursitis (Agyekum and Ma, 2015). Signs and symptoms are similar to retrocalcaneal bursitis, except that the bony Haglund deformity may also be felt. The presence or absence of a Haglund deformity is not pathognomonic or specific to a diagnosis of retrocalcaneal bursitis.

Simple lifestyle modification by avoiding shoes that cause irritation – suggesting open-back shoes or any with more padding over the Achilles tendon insertion – may be all that is required. If symptoms persist, a corticosteroid injection into the bursa may be trialled, taking care to avoid the tendon itself (aim into the recess just lateral to the midline of the posterior calcaneus where the Achilles tendon inserts). Achilles tendon stretching exercises should be implemented. After 6 months of conservative therapy, patients may be referred to orthopaedics for consideration of bursectomy and/or resection of symptomatic Haglund deformities.

Calcaneal apophysitis (Sever's disease)

This is inflammation of the calcaneal growth plate and is the commonest cause of heel pain in children between 5 and 11 years of age (Tu and Bytomski, 2011). Repetitive contraction of the Achilles tendon in active children can cause strain on the calcaneal growth plate leading to calcaneal apophysitis. It has also been suggested that rapid pubertal growth may cause temporary strain on the growth plate leading to disease (James et al, 2013). Sever's disease ceases upon fusion of the calcaneal growth plate (Sever, 1912).

Patients describe a gradual onset of pain (Agyekum and Ma, 2015), associated with activity. Examination reveals tenderness over the posterior half of the calcaneus, often with little swelling. Radiographs are frequently normal but occasionally may show a sclerotic calcaneal apophysis.

This is exclusively managed conservatively with symptom resolution subsequently occurring within 2 months (Agyekum and Ma, 2015). Conservative management is initiated as above; sometimes these patients may also benefit from Achilles stretching exercises under the care of the physiotherapists. In more severe cases, heel raises can be initiated for symptom relief. Cast immobilization is rarely needed, generally for non-compliant patients. The authors are unaware of any case requiring surgical intervention.

Neurological causes of heel pain

As with nerve compression elsewhere, paraesthesia is cited (as 'pins and needles' or 'tingling') that may radiate into the appropriate areas of the foot or into the toes. Symptoms worsen as the day progresses, and are relieved by rest or loose fitting shoes (Tu and Bytomski, 2011). Rather conversely peripheral neuropathy is also associated with nocturnal pain that frequently disturbs sleep (McSweeney and Cichero, 2015), possibly as a result of transient recumbent oedema, causing patients to arise and 'shake' off the pain. Risk factors include patients whom have previously suffered hindfoot trauma or undergone surgery to it.

Tarsal tunnel syndrome

The posterior tibial nerve or its branches are frequently affected. It enters the foot posteromedially by coursing through the tarsal tunnel (*Figure 2*); a fibro-osseus tunnel that is covered by the flexor retinaculum of the foot which attaches to the medial malleolus, medial calcaneus and talus posteriorly. Tarsal tunnel syndrome is compression of the nerve in this location which produces pain over the medial ankle with vague paraesthesia sometimes radiating to the great toe. Patients with pes planus are inherently at risk of tarsal tunnel syndrome (Tu and Bytomski, 2011) because of the corresponding flattening of the tunnel. Little may be noted upon examination, but tapping over the tarsal tunnel may reproduce pain and associated paraesthesia (Tinel's sign). Otherwise the nerve may be provoked by plantarflexing and inverting the foot, or with dorsiflexion and eversion (McSweeney and Cichero, 2015). However, the symptoms and signs are frequently ambiguous and nerve conduction studies are required for a formal diagnosis.

Sinus tarsi syndrome

The sinus tarsi is situated over the lateral ankle complex and describes the space between the posterior subtalar joint posteriorly, talus superiorly, calcaneus inferiorly and extends anteriorly to the talonavicular joint (*Figure 3*). It is continuous medially with the tarsal tunnel. Branches of the deep peroneal nerve innervate this space (Choudhary and McNally, 2011) and sinus tarsi syndrome is the commonest neuropathy found laterally over the heel. As with tarsal tunnel syndrome, the biggest risk factor is previous hindfoot trauma, but it can also be seen in patients with rheumatoid arthritis. Pain is again exacerbated on exertion, particularly on uneven surfaces, and relieved by rest. Conversely to tarsal tunnel syndrome, the pain is usually sharp and well localized to the sinus tarsi or inferior pole of the distal fibula. Examination reveals tenderness in the sinus tarsi, generally with little else. Weightbearing radiographs of the foot are often normal.

KEY POINTS

- A structured approach to heel pain aids diagnosis and subsequent management by categorising the location of pain.
- The commonest cause of heel pain is inferior heel pain secondary to plantar fasciitis.
- Conservative management by using physiotherapy and orthotics services is sufficient for the vast majority of patients.
- Conservative therapy should be trialled for at least 6 months before onward referral to orthopaedics.
- If heel pain is bilateral always consider diabetes or a more central (spinal) cause.

Management

Management of both neuropathies follows the same pathway. Conservative measures include lifestyle modification and use of orthotic in-soles (particularly for sinus tarsi syndrome), as well as trial of anti-inflammatories (Tu and Bytomski, 2011). Corticosteroid injections are the next step – although steroid injections for tarsal tunnel syndrome may be trialled in the community the authors recommend all sinus tarsi injections be referred to orthopaedics as radiographic guidance is always required. Resistant cases of tarsal tunnel syndrome should be referred for consideration of decompression, although this is seldom performed (McSweeney and Cichero, 2015).

Peripheral neuropathy rarely ever presents bilaterally. Thus bilateral presentation should raise suspicion for more central nerve compression (particularly with dermatomal distribution of pain; 'radiculopathy'), or indeed systemic disease such as diabetes – the latter should always be investigated in any instance of suspected neuropathy of the foot. Patients with known diabetes presenting with new heel pain require a full re-assessment of their diabetes as this may be an early hallmark for a Charcot joint.

Conclusions

The causes of heel pain are vast but diagnosis can be quickly ascertained with a structured approach. Accurate localization of pain is essential to diagnosis. Examination findings are often ambiguous and radiology can add little to the diagnosis without more specialized imaging, thus history is key. The vast majority of patients can be managed conservatively with lifestyle modification, physiotherapy and orthotics providing the mainstay of treatment. Conservative therapy should be trialled for at least 6 months before considering referral to an orthopaedic surgeon. **BJHM**

Conflict of interest: none.

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