Plantar Fasciitis (Heel Pain)

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal’s medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence.
PLANTAR FASCIITIS (HEEL PAIN)

**Mechanics:** The plantar fascia is a multilayered inelastic fibrous sheet (aponeurosis) composed primarily of Type I collagen. It originates from a bony prominence on the bottom of the heel bone (the medial calcaneal tuberosity) and inserts through several fibrous bands into the plantar plate of the metatarsophalangeal joints, flexor tendon sheaths and the base of the proximal phalanges of the toes. The distal insertion site of the Achilles tendon (heel cord) and the origin of the plantar fascia are close in proximity and the outer layer of the two structures is continuous. Since the plantar fascia originates proximally and inserts distally to the subtalar and midtarsal joints, any functional change in its length can alter the position of the said joints. The plantar fascia is the most important static soft tissue supporter of the medial arch; transecting it in loaded Cadaveric specimens results in a 21% loss of the height of the medial arch [1]. The plantar fascia has an important functional role in the normal gait cycle. In the latter part of the stance phase, the toes dorsiflex. This functionally shortens the plantar fascia causing the calcaneus to rotate inwards (into varus) and the medial arch to elevate (windlass mechanism). The internal rotation of the calcaneus causes a divergence in the joint axis of two of the mid-foot joints (the talonavicular and calcaneocuboid joints), thereby locking the joint complex. The now stable midfoot allows for the smooth transition of forces to occur from the hindfoot to the forefoot. In this manner, the plantar fascia is an important soft tissue stabilizer of the foot with an essential functional role during gait.

**Pathophysiology:** The bottom surface of the heel (calcaneal fat pad) is made up of elastic adipose-tissue-supported spiral fibrous bands (septa) and is the primary structure that cushions the heel from compressive forces at heel strike [2]. The plantar fascia acts as a tie-rod in the foot truss. The primary forces it is subjected to are tensile. The tension is (greatest) at the origin of the plantar fascia near its insertion at the medial calcaneal tuberosity [3]. This is also the location where the plantar fascia commonly becomes swollen and inflamed. It is theorized that the repetitive tensile forces created by walking or standing cause micro-tears in the fascia, leading to acute and eventual chronic inflammation. If the inflammation affects the entire plantar fascia, it is referred to as a plantar fasciitis; however, if it is isolated to the insertion site of the heel, it is called a heel pain (the more common of the two.) MRI studies have demonstrated an increased thickness of the plantar fascia from 3 mm in a normal heel to 7.4 mm in patients with a chronic heel pain [4]. Biopsies of the inflamed area have revealed collagen necrosis (death of some of the fibrous tissue), angiofibroblastic hyperplasia (overgrowth of fibrous tissue and tiny blood vessels), chondroid metaplasia (formation of cartilage tissue) and matrix
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calcification - all microscopic changes typically seen in areas with chronic inflammation and deficient blood flow. On physical examination, the patient often has tightness of the Achilles tendon - this is consistent with the close anatomical relationship of the heel cord with the plantar fascia [5]. It is unclear whether a tight heel cord is a precipitating factor or simply associated with the heel pain [6-9].

In a small percentage of patients, the heel pain is the result of both inflammation of the plantar fascia and entrapment of the nerves in the distal tarsal tunnel. The most common nerve involved in this process is the first branch of the lateral plantar nerve sometimes referred to as Baxter’s nerve. This is a motor sensory branch which supplies sensory nerves to the periosteum of the calcaneus and motor nerves to the abductor digiti minimi muscle.

Etiology: Heel pain may be caused by plantar fascia rupture, fat pad atrophy, stress fractures of the calcaneus, proximal plantar fasciitis, distal plantar fasciitis, plantar fibromatosis, tendonitis of the flexor hallucis longus, tumor of the calcaneus and nerve entrapment, or injury [5]. The most common cause is inflammation of the proximal portion of the plantar fascia. There is no consensus regarding etiology. Snook and Christman wrote, "it is reasonably certain that a condition which has so many different theories of etiology and treatment does not have valid proof of any one cause" [10]. Factors associated with or aggravating this condition are better defined: age, sex, obesity, seronegative inflammatory disorders and activity levels. The average age is 45 years. Prevalence is twice in females vs. males [5]. There may be a history of elevated stress to the foot as a result of increased activities, prolonged standing or weight gain. Physical activity certainly aggravates and can sometimes precipitate the heel pain [11-14]. A retrospective review of injuries among runners cited plantar fasciitis as one of the five most common injuries [14].

Numerous studies have assessed whether or not abnormal foot mechanics predispose an individual to heel pain [5, 7, 9, 12, 13, 15]. Pes planus [16] and pes cavus were the most common foot deformities assessed. A predominance of either deformity has not been identified. Theoretically, both deformities can cause abnormal stresses on the plantar fascia [3, 16-21] and clinical correlations have been suggested [12]. However, a consistent correlation has not been evidenced by retrospective clinical reviews.

Several studies have implicated body weight as a causative factor [8, 10, 22, 23]. Furey [22] identified an increased prevalence of heel pain among obese patients, and Snook and Chrisman [10] noted half of their patients with heel pain were overweight. This makes intuitive sense given that heel pain is
considered an over-use syndrome associated with advancing in age. Increased body mass elevates the forces on the aging soft tissues that are less capable of tolerating the stresses. Weight loss in such patients is often impossible since the painful heel limits their activities.

Factors such as acute injury, the presence of a heel spur, the type of footwear, the walking surface and employment or chronic repetitive activity (other than athletics) have been proposed, but they have not been established as causative [24]. A correlation between prolonged standing or walking has been suggested [8, 9, 25, 26], but direct causation is unsubstantiated since heel pain can occur in all types of patients with varying levels of recreational activities and job requirements. The consistency of the surface on which one walks has not been established as a causative or aggravating factor. This is likely because tension, rather than compression, is the primary force which precipitates injury and inflammation of the plantar fascia. The magnitude of the tensile forces placed through the plantar fascia is related more to the activity level, types of activities, body weight and stability of the medial arch [3, 17-21] rather than the consistency of the walking surface. Although many patients are concerned with the presence of a heel spur, it is not recognized as an etiologic factor. Approximately 50% of patients with heel pain and 16% without have a heel spur [27]. Anatomically, the heel spur is located at the origin of the flexor digitorum brevis muscle, not at the origin of the plantar fascia. Presumably, the same process occurring at the plantar fascia can occur at the origin of the intrinsic muscles of the foot leading to periostitis and new bone formation. Lapidus et al have demonstrated that successful treatment of heel pain is not contingent with excision of the heel spur [8].

Pain at the sites of tendon or ligament insertion (enthesiopathy) is often observed in patients with seronegative arthropathies [5]. Clinical syndromes such as ankylosing spondylitis, psoriatic arthritis, Reiter's syndrome, inflammatory bowel disease and Behcet's syndrome must be ruled out. Associated complaints such as skin lesions, conjunctivitis, arthritis, back pain or abdominal complaints should prompt a referral to a rheumatologist. Screening tests include antigen-B27 (HLA-B27) and sedimentation rate (ESR) can aid in the diagnosis.

Conclusion: The plantar fascia is an inelastic structure that plays an important role in maintaining the stability of the medial arch and midtarsal joints. The primary force placed through the plantar fascia is tension, not compression. It functions as a tie-rod in the foot truss and, during the gait cycle, is under maximal tension in the mid-portion and terminal portions of the stance phase. It is generally accepted that the primary etiology of heel
pain is the result of repetitive tensile forces placed through aging tissue that is no longer capable of tolerating the stresses. While associated factors for this condition have been identified, no one activity is known to be the cause of heel pain.
The windlass mechanism puts tension on the plantar fascia and raises the arch passively.

Le mécanisme de treuil applique une tension sur le fascia plantaire et soulève la voûte plantaire de façon passive.

References

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