

Plantar fasciitis

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Heel pain, mostly caused by plantar fasciitis (PF), is a common complaint of many patients who requiring professional orthopedic care and are mostly suffering from chronic pain beneath their heels. The present article reviews studies done by preminent practitioners related to the anatomy of plantar fasciitis and their histo-pathological features, factors associated with PF, clinical features, imaging studies, differential diagnoses, and diverse treatment modalities for treatment of PF, with special emphasis on non-surgical treatment. Anti-inflammatory agents, plantar stretching, and orthosis proved to have highest priority; corticosteroid injection, night splints and extracorporeal shock wave therapy were of next priority, in patients with PF. In patients resistant to the mentioned treatments surgical intervention should be considered.

Key words: Plantar fasciitis, plantar heel pain, risk factors, imaging studies, treatment

INTRODUCTION

Heel pain is a common presenting complaint in the foot and ankle practice, and plantar fasciitis (PF) is the most common cause of chronic pain beneath the heel in adults, making up 11–15% of the foot symptoms requiring professional care among adults.^[1-4] It is estimated that 1 in 10 people will develop PF during their lifetime.^[5] PF, which is more common in middle-aged obese females and young male athletes, has a higher incidence in the athletic population though not all suffering require medical treatment. In the literature, PF has been described as painful heel syndrome, chronic plantar heel pain, heel spur syndrome, runner's heel, and calcaneal periostitis.^[6,7]

Search strategy

Peer-reviewed journal articles that predominantly focus on plantar heel pain are included in this review, which of course does not included non-English language reports. Studies have been identified using the following databases: *PubMed* (from 1980 to 2012), *Ovid Medline* (from 1980 to 2010), *Web of Science* (from 1980 to 2012), *EMBASE* (from 1980 to 2012), *CINAHL* (from 1982 to 2012), *Cochrane Database of Systematic Reviews* (1980 to 2012), *Cochrane Central Register of Controlled Trials* (from

1980 to 2012), and *AMED* (from 1985 to 2012). Using the keywords "heel pain," "painful heel," "plantar fasciitis," and "heel spur" and combining them with search terms: "treatment," and "management," 51 journal articles were identified. Of these, 42 were primary articles while the remaining 9 were review papers.


Patho-anatomical features

The differential diagnosis of PF precedes an understanding of the local anatomy. The calcaneum is separated from plantar skin by a complete honeycombed fibro-fatty fat pad that acts as a shock absorber.

The posterior tuberosity of calcaneum has medial and lateral processes. The medial process gives attachment to the Flexor digitorum brevis (FDB), Abductor hallucis (AH), and the medial head of Quadratus plantae (QP) as well as the central band of plantar fascia.

The plantar fascia or deep fascia of the sole, proximally has a direct fibrocartilaginous attachment to the calcaneum (an entheses), whose central band is constant along with medial and lateral band. It has a triangular shape and develops from the medial process of the calcaneal tuberosity, and diverges distally at mid-metatarsal level into five separate strands, which are attached at the forefoot onto the plantar skin, the base of proximal phalanges (via plantar plate), the metatarsophalangeal (MTP) joints via the collateral ligaments and deep transverse metatarsal ligaments.^[6]

Heel skin is innervated by the medial calcaneal nerve which may present with heel pain if compressed proximally (such as in tarsal tunnel syndrome). Boxter's

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nerve (the first branch of lateral plantar nerve) may be at risk of compression between AH and medial belly of the QP muscle.^[6,8]

Despite the high prevalence of PF, information about its pathogenesis is still limited, and its histological changes are suggestive of degeneration rather than inflammation. The fascia is usually markedly thickened and gritty. These pathologic changes are more consistent with fasciosis (degenerative process) than fasciitis (inflammatory process), but fasciitis remains the accepted description in the literature.^[9]

Histological evidence shows that spur formation can occur in loose connective tissue, surrounding fibrocartilage which may not be aligned with the direction of traction, and spur trabeculae commonly forms perpendicular to its long axis. Additionally, clinical studies have shown that spur development is unrelated to medial arch height and can occur after surgical release of the plantar fascia.^[6,9,10]

Factors associated with PF

Identifying factors associated with PF will help identifying at risk individuals and development of new and improved preventative and treatment strategies. Obesity is present in up to 70% of patients with PF. According to the literatures, there is a strong association between increased body mass index (BMI) and PF in a non-athletic population. The evidence suggests that unlike weight, height has no association with PF. More specifically, increased weight is associated with PF, but not necessarily with reduced height. Interestingly, there is no correlation between PF and weight, height or BMI in an athletic population.^[11]

Heel spurs have commonly been implicated as a risk factor for PF. Current studies demonstrate a highly significant association between calcaneal spur and PF. Besides, there is a weak association between increasing age, prolonged standing, decreased first MTP joint extension, decreased ankle dorsiflexion, and PF.^[10,11]

According to Kibler *et al.*, deficits in flexibility of the plantar flexor muscles may contribute to a greater fascia stretching.^[12] Cheung *et al.* contend that intense muscle contractions of the plantar flexor muscles cause indirect stretching of the fascia, increasing the risk of developing the PF.^[13]

Some reports suggest that 81–86% of patients with PF have excessive pronation.^[14] Despite the fact that the pronated foot posture and over-pronation during gait are commonly cited as causative factors for PF, there is conflicting evidence with regard to the association of static foot posture and dynamic foot motion with PF.^[15,16]

Clinical features and diagnosis

The diagnosis of PF is usually clinical and rarely needs to be investigated further.^[17,18] The patient complains of pain in the medial side of the heel, most noticeable with initial steps after a period of inactivity and usually lessens with increasing level of activity during the day, but will tend to worsen toward the end of the day.^[19] Symptoms may become worse following prolonged weight bearing, and often precipitated by increase in weight bearing activities. Paresthesia is uncommon.^[16] PF is usually unilateral, but up to 30% of cases have a bilateral presentation.^[7] Tightness of Achilles tendon is found in almost 80% of cases.^[2]

Occasionally the pain may spread to the whole of the foot including the toes. Tenderness can be elicited over the medial calcaneal tuberosity and may exaggerate on dorsiflexion of the toes or standing tip toe.^[20] The clinical course for most patients is resolution of symptoms within a year.^[21]

Imaging studies

Imaging studies are typically not necessary for diagnosis of PF.^[9,17] In the clinical management of chronic heel pain, diagnostic imaging can provide objective information. This information can be particularly useful in cases that do not respond to first-line interventions, or when considering more invasive treatments (e.g. corticosteroid injection).

- Lateral radiograph of the ankle should be the first imaging study. It is a good modality for assessment of heel spur, thickness of plantar fascia, and the quality of fat pad. Stress fractures, unicameral bone cysts, and giant cell tumors are usually identified with plain radiography.^[18,19,22]
- Ultrasound examination is operator-dependent, but it proves to be significant when the diagnosis is unclear.^[23,24] In the literature, normal thickness of the plantar fascia when measured in ultrasound varies in range (mean 2–3 mm). People with chronic heel pain are likely to have a thickened plantar fascia with associated fluid collection, and that thickness values >4.0 mm are diagnostic of plantar fasciitis.^[6]
- Plantar fascia thickness values have also been used to measure the effect of treatments and there is a significant correlation between decreased plantar fascia thickness and improvement in symptoms.^[25-27]
- MRI can be used in questionable cases, which fail conservative management or are suspected of other causes of heel pain, such as tarsal tunnel syndrome, soft tissue and bone tumors, osteomyelitis, subtalar arthritis, and stress fracture.^[17,18]

Differential diagnosis

Although PF is the most common cause of chronic plantar heel pain, there are multiple differential diagnoses [Table 1], most of which can be excluded following a comprehensive history and physical examination.

Treatment

The natural history of PF is often self-limited. However, the typical resolution time is anywhere from 6 to 18 months and sometimes longer,^[20] which can lead to dissatisfaction of patient and physician. Most experts agree that early recognition and management of PF leads to short course of treatment and greater chance of success with conservative therapies.^[6]

Numerous interventions have been described for treatment of PF, which include: rest, heat, ice pack, non-steroidal anti-inflammatory drugs (NSAIDs), heel pads, magnetic insole, night splints, walking cast, taping, plantar and Achilles stretching, ultrasound, steroid injection, extra-corporeal shock wave therapy, platelet-rich plasma injection, pulsed radiofrequency electromagnetic field therapy, and surgery. Unfortunately, few high-quality randomized, controlled trials have been made to support these therapies. All in all, a trial of conservative treatment is generally advised before more invasive interventions are attempted.

Stretching

Stretching may be in calf or plantar region. Numerous authors have recommended that calf stretching should be one of the interventions used for patients with PF.^[7,17,18,28] A calf stretch is performed with the patient stands with staggered legs facing toward a wall, with both hands stretched out.

According to Porter *et al.* the dosage for calf stretching can be either three minutes at a time, three times a day or five 20-s intervals, twice daily, as both have the same effect.^[29] The continuity of the connective tissue between the Achilles tendon and the plantar fascia as well as the fact that decreased ankle dorsiflexion is a risk factor in the development of plantar fasciitis provides some justification for calf stretching.^[19]

Table 1: Differential diagnoses of plantar fasciitis

Plantar fascia rupture: Sudden, acute, knife-like pain, ecchymosis, which is more proximal and may be associated with a palpable gap. MRI or ultrasound confirms the diagnosis.
Fat pad syndrome: Atrophy of heel pad, common in elderly and diabetic patients, pain is usually centrally located and is not characterized with morning pain.
Calcaneal stress fracture: Pain with weight-bearing, worsens with prolonged weight-bearing, diffuse heel tenderness
Tumor: Pain is typically aching, constant, nocturnal, and even present without weight bearing and at rest, constitutional symptoms late in the course
Calcaneal bursitis (Policeman's heel): Burning, aching or throbbing type of pain, swelling, and erythema of posterior heel
Boxter's nerve entrapment: Pain is more proximal and dorsal, no sensory disturbance
Medial calcaneal nerve compression: Occurs in tarsal tunnel, positive Tinnl's sign and altered sensation of medial side of the heel.
Seronegative arthropathies: Usually bilateral, history of back pain, urethritis, uveitis, elevated blood inflammatory markers, etc.
Spinal stenosis and L5-S1 nerve root irritation

DiGiovanni *et al.* were the first to publish that tissue specific plantar fascia-stretching exercise is more effective than calf stretching in a randomized clinical trial.^[30] Moreover, it seems that plantar fascia-stretching exercise is more effective than low dose shock wave therapy in acute phase of PF.^[31]

Night splints

The design of night splinting is to keep the patient's ankle in a neutral position overnight, passively stretching the calf and plantar fascia during sleep. There is no difference between the various types of the night splints whose purpose is to allow the fascia to heal.^[32,33] There is moderate evidence that night splints are useful in improving symptoms of PF, which are recommended to be used for 1–3 months and should be considered as an intervention for patients with symptoms greater than 6 months in duration.^[19]

Orthosis

The rationale for use of foot orthoses was to decrease abnormal foot pronation that was thought to cause increased stress on the plantar fascia, but to date based on Ribeiro *et al.* results, the pain reduction mechanism obtained by the use of insoles would be mostly related to its supporting function of the longitudinal arch and not to the overload reduction over the plantar surface.^[34] There appears to be no difference between prefabricated or custom foot orthoses in the results of treatment which is strongly recommended to be used to provide short-term (3 months) reduction in pain and improvement in function.^[35] There is inconclusive evidence with regard the long-term(12 months) use of orthotic devices.^[6,19,36]

Local injection of steroids

When more conservative management is unsuccessful, steroid injection is a preferred option. There is no gold standard regarding the types and doses of local injection of corticosteroids. It is recommended that steroid injection should be performed with precise determination of the location, which can be easily achieved by using ultra-sonographic guidance.^[37] Generally, the medial approach is likely to be less painful than a direct plantar approach. Injecting deep to the plantar fascia ensures adequate spread of the steroid preparation and reduces the risk of fat pad atrophy.^[6,38]

Siavashi *et al.* compared the efficacy of the corticosteroid injection with plantar stretching and believe there is no difference after 8 weeks between these two methods in patient's symptoms.^[39]

Corticosteroid injection has been shown to significantly reduce plantar fascia thickness as early as two weeks and one month following treatment. Additionally, there is a significant correlation between decreased plantar fascia thickness and improvement in symptoms. Results

of a Cochrane review show that corticosteroid injection therapy has short-term benefit compared to control, and the effectiveness of treatment is not maintained beyond six months.^[27,40,41] Complications of steroid injection are not common. Reported complications of palpation-guided steroid injection are plantar fascial rupture, fat pad atrophy, lateral plantar nerve injury secondary to injection, and calcaneal osteomyelitis.^[42,43] However, these complications have not been reported following ultrasonographic-guided injections.^[37]

Extra-corporeal shock wave therapy

Extra-corporeal shock wave therapy (ESWT) can be of high or low energy. It has been claimed that the deep tissue cavitation effect causes micro rupture of capillaries, leakage of chemical mediators, and promotion of neovascularization of the damaged tissue.^[6] It is usually applied under intravenous sedation with or without local infiltrative anesthesia.^[44,45] ESWT is indicated if there is failure of other conservative modalities such as stretching exercises, casting or night splinting, and symptoms lasting for more than 6 months. As this is a relatively safe procedure, it could be considered before any surgical treatment and may be preferable to try before local steroid injection.^[46] Bilateral cases can be treated under a single anesthetic and full weight bearing may be started immediately. Prior steroid injections of over three times appear to be a poor prognostic factor for good recovery following ESWT.^[17] This modality is contraindicated in bleeding diatheses.^[18]

The outcome of ESWT is not dependent on the presence of calcaneal spur where it does not change the radiographic appearance of the spur.^[47]

Presence of calcaneal bone marrow edema on MRI has been found to be a good predictive indicator for a satisfactory clinical outcome following ESWT.^[48]

There are inconclusive data whether to use local steroids or ESWT. Sorrentino *et al.* suggest in patients with idiopathic PF, perifascial edema seems to be a useful criterion to address the therapy to the high resolution ultrasonographic-guided steroid injection treatment, while in cases without edema, treatment could be address to ESWT.^[49] According to Saber *et al.*, both local steroid injection and ESWT are proved to be effective in treatment of PF, but as steroid injection is more cost effective and has more reproducible results regardless of machine or operator, it is preferred. However, ESWT should be considered prior to any surgical treatment for recalcitrant PF.^[25]

Autologous platelet rich plasma (PRP)

There is substantially growing enthusiasm for the use of growth factor containing harvested blood/ platelet

concentrate which, unlike steroids, can stimulate the reparative process.^[50,51] Current studies have revealed that local injection of PRP provides significant relief of pain and improvement of function, and the results seems to be comparable, and sometimes superior to local steroid injection.^[50] However, available data are limited by quality and size of the study, as well as length of follow-up, and are currently insufficient to recommend this modality for routine clinical use.

Surgery

Recalcitrant cases where symptoms persist for more than 6–12 months, even after adequate conservative treatment are usually selected for surgery.^[17] Before surgery nerve conduction and electromyographic studies should be considered to determine if the posterior tibial nerve is compressed.

Open or endoscopic plantar fascia release may be done. Some advantages of endoscopic plantar fasciotomy include: minimal soft tissue dissection, excellent visualization of the plantar fascia, minimal post operative pain, and earlier return to work. However, the American Orthopaedic Foot and Ankle Society recommends that in case of suspected nerve compression, endoscopic release should be avoided.^[52] All in all, still, the procedure of choice is open partial plantar fascia release with simultaneous release of first branch of lateral plantar nerve.^[6] A large cohort study indicates that 70% of patients showed improvement following surgery but only 50% of patients displayed complete satisfaction.

Following complete division of the plantar fascia, the development of pes planus, secondary hallux valgus, or hammer toes are expected, and therefore orthotics are required lifelong post-operatively.^[18]

CONCLUSION

PF is the most common cause of inferior heel pain in adults. The patient usually complains of gradual onset of pain along the medial side of the heel. The pain is worse when arising in the morning which becomes less severe after the few steps. The diagnosis of PF is usually clinical and rarely needs to be investigated by imaging or electromyographically.

In most patients with PF, conservative treatment usually is sufficient. Initially, a period of rest accompanied by anti-inflammatory agents (ice pack/heat, NSAID's), stretching, and an orthosis is recommended. There is no difference in which types of orthosis is used, although plantar stretching seems to be more effective. If the patient remain symptomatic, corticosteroid injection and night splint (especially in patients with symptoms greater than 6 months in duration) may be reasonable. ESWT should be considered prior to any surgical intervention in patients with refractory PF.

In a good majority of the patients, these modalities are sufficient and the patient will become symptom free.

However, if after 6–12 months of conservative treatment, the patient still has sufficient symptoms that interfere with their activities of daily life, surgical intervention should be considered. Moreover, newer treatment modalities such as local injection of PRP which may play more important roles in near future should also be considered.

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