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Procedure for Evaluation and Treatment of Chronic Plantar Fasciitis

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Abstract

Heel pain, most frequently caused by plantar fasciitis (PF), is a common complaint of many patients requiring specialized orthopedic care and is usually chronic pain under the heel. In this article, we discuss the anatomy of plantar fasciitis and its histopathologic features, factors associated with PF, clinical features, imaging studies, differential diagnosis, and various therapeutic modalities used to treat PF. We review studies conducted by prominent practitioners, with a particular focus on non-surgical treatments. Anti-inflammatory medications, plantar stretching, and orthotics turned out to be top priorities. Corticosteroid injections, nocturnal splints, and extracorporeal shock wave therapy were the next priorities for patients with PF. Surgical intervention should be considered in patient's refractory to the above treatments.

Keywords: Plantar fasciitis; Plantar heel pain; Risk factors

Introduction

Heel pain is a common complaint of foot and ankle exercise, and plantar fasciitis (PF) is the leading cause of chronic pain under the heel in adults and is under professional management in adults accounts for 11-15% of foot conditions requiring [1]. It is estimated that in his lifetime, 1 in 10 will develop her PF. PF is common in obese middle-aged women and young male athletes, and is common in athletes, but not all symptoms require medical attention. In the literature, PF is described as painful heel syndrome, chronic plantar heel pain, plantar fasciitis, runner's heel, and calcaneal periostitis.

Patho-anatomical features

An understanding of topographic anatomy precedes the differential diagnosis of PF. The calcaneus is separated from the plantar skin by a complete honeycomb fiber fat pad that acts as a shock absorber [2].

The posterior calcaneal tubercle has a medial and a lateral process. The medial process connects the flexor digitorum brevis (FDB), the abductor muscle (AH), the medial head of the quadratus plantar muscle (QP), and the medial ligament of the plantar fascia [3].

The plantar fascia or deep fascia of the sole of the foot has a direct fibrocartilage attachment proximally to the calcaneus (the attachment), and its median ligament is constant with the medial and lateral ligaments. Triangular in shape, it develops from the medial process of the calcaneal tubercle, branches distally at the level of the metatarsals into five separate cords, and in the forefoot (via the plantar plate) the plantar skin, the proximal phalanges. adheres to the base of the, the metatarsophalangeal joint (MTP) via the collateral ligament and the deep transverse metatarsal ligament [4].

The skin of the heel is innervated by the medial calcaneal nerve and can cause heel pain when compressed proximally (e.g. tarsal tunnel syndrome). The Boxster nerve (first branch of the lateral plantar nerve) may be compressed between the medial ventral AH and QP muscles [5].

Despite the high prevalence of PF, information on its etiology is still limited and its histologic changes suggest degeneration rather than inflammation. The fascia is usually quite thick and rough. Although these pathological changes are more consistent with fasciitis (a degenerative process) than with fasciitis (an inflammatory process), the literature still accepts a description of fasciitis [6].

Histological evidence suggests that spur formation can occur in loose connective tissue surrounding fibrocartilage that may not be aligned with the direction of pulling, and that its trabeculae form generally perpendicular to its long axis indicates that Furthermore, clinical studies have shown that spur development is independent of medial arch height and can occur after surgical release of the plantar fascia [7].

Factors associated with PF

Identifying factors associated with PF will help identify at-risk individuals and develop new and improved prevention and treatment strategies. Obesity is present in up to 70% of patients with PF [8]. Literature shows a strong association between increased body mass index (BMI) and PF in sedentary populations. Evidence suggests that height, unlike weight, is not related to PF. More specifically, weight gain is associated with her PF, but not necessarily with height loss. Interestingly, there is no correlation between the athlete's PF and weight, height, or BMI [9].

Heel spurs are often considered a risk factor for PF. Recent studies have shown a very important association between the heel spur and PF. There is also a weak association between increasing age, prolonged standing, decreased first MTP joint extension, decreased ankle dorsiflexion, and PF.

According to Kibler et al. Lack of plantar flexor flexibility may contribute to increased fascial stretch. They argue that intense muscle contraction of the plantar flexor causes indirect stretching of the fascia, increasing the risk of developing PF [10].

Some reports suggest that 81-86% of patients with PF have excessive pronation. Although pronation foot posture and gait overpronation are commonly cited as causes of her PF, there is conflicting evidence regarding the association of static foot posture and dynamic foot movement with her PF.

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Clinical features and diagnosis

Diagnosis of PF is usually clinical and rarely requires further investigation. The patient complains of pain on the inside of the heel. This is most pronounced in the first steps after a period of inactivity and usually decreases as activity levels increase throughout the day, but tends to worsen towards the end of the day. Symptoms can worsen after prolonged weight-bearing and are often caused by increased weight-bearing activity. PF is usually unilateral, but is bilateral in up to 30% of cases. Achilles tendon strain is seen in nearly 80% of cases [11].

In some cases, the pain may spread throughout the foot, including the toes. Hypersensitivity may be induced at the medial calcaneal tuberosity and may be exaggerated with dorsiflexion of the toes or tip of the standing toes. The clinical course is that in most patients he resolves symptoms within 1 year.

Imaging studies

Imaging is usually not required to diagnose PF. Imaging can provide objective information in the clinical management of chronic heel pain. This information is especially useful when people do not respond to first-line treatment or when more invasive treatments (such as corticosteroid injections) are being considered [12].

- X-ray of the lateral ankle should be the first imaging test. It is a suitable method to assess heel spur, plantar fascia thickness and fat pad quality. Stress fractures, single-chamber bone cysts, and giant cell tumors are usually identified on plain x-rays.

- Ultrasound is operator dependent, but makes sense when the diagnosis is unclear. In the literature, the normal thickness of the plantar fascia as measured by ultrasound varies within a range (2–3 mm on average). A person with chronic heel pain likely has thickened plantar fascia with associated fluid buildup, and a thickness value greater than 4.0 mm indicates he has plantar fasciitis.

- Plantar fascia thickness has also been used to measure the effectiveness of treatment, and there is a significant correlation between reduction in plantar fascia thickness and improvement in symptoms.

- MRI can be used in suspicious cases where conservative treatment fails or when other causes of heel pain are suspected. Tarsal tunnel syndrome, soft tissue and bone tumors, osteomyelitis, subtalar arthritis, stress fractures.

Treatment

The natural history of PF is often self-limiting. However, the typical healing time for him is 6 to 18 months, sometimes longer, which can lead to patient and physician dissatisfaction [13]. Most experts agree that early detection and treatment of PF shortens the treatment course and increases the chances of success with conservative therapy.

Treatments for PF include rest, heat, ice packs, nonsteroidal anti-inflammatory drugs (NSAIDS), heel pads, magnetic insoles, night splints, walking casts, taping, plantar and Achilles tendon stretching, ultrasound, and steroids. A number of interventions have been described. Injections, extracorporeal shock wave therapy, platelet-rich plasma injections, pulsed radiofrequency electromagnetic field therapy and surgery. Unfortunately, few high-quality randomized controlled trials have been conducted to support these treatments. Overall, conservative management trials are generally recommended before attempting more invasive procedures [14].

Stretching

Stretches can be done in the calf or plantar area. Many authors recommend the use of calf stretching as an intervention for patients with PF. For the calf stretch, stand with your feet facing a wall and your hands outstretched.

According to Porter the dosage of the calf stretch is either he 3 minutes at a time, 3 times a day or he 5 times, 2 times a day at 20 second intervals, both have the same effect. The continuity of connective tissue between the Achilles tendon and the plantar fascia, and the fact that decreased ankle dorsiflexion is a risk factor for developing plantar fasciitis, partially justifies calf stretching [15].

Night splints

The design of the night splint is to keep the patient's ankle in a neutral position overnight and passively stretch the calf and plantar fascia during sleep. To make healing possible. There is moderate evidence that Night Splint helps improve symptoms of her PF, and its use is recommended for 1-3 months and should be considered an intervention for patients whose symptoms persist for more than her 6 months.

Local injection of steroids

Steroid injections are the preferred option when more conservative treatments fail. There is no gold standard for type and dosage of topical corticosteroid injections. We recommend performing steroid injections with precise localization that can be easily achieved using ultrasound guidance. In general, the medial approach is considered less painful than the direct plantar approach. Injection deep into the plantar fascia allows the steroid preparation to spread sufficiently and reduces the risk of fat pad atrophy. We compared the efficacy of corticosteroid injections with plantar stretching and believe that there is no difference between these two methods in terms of patient symptoms at 8 weeks [16].

Injections of corticosteroids have been shown to significantly reduce plantar fascia thickness from 2 weeks to 1 month after treatment. Furthermore, there is a significant correlation between reduced plantar fascia thickness and symptom improvement. The results of a Cochrane review show that corticosteroid injection therapy provides short-term benefits compared to controls, with treatment efficacy not lasting longer than 6 months. Complications of steroid injections are uncommon.

Extra-corporeal shock wave therapy

Shock wave therapy can be high energy or low energy. Deep-tissue cavitation effects have been hypothesized to cause capillary microrupture, leakage of chemical mediators, and enhanced angiogenesis in injured tissue. It is usually administered under intravenous sedation with or without local infiltration anesthesia. ESWT is indicated when other conservative treatments such as stretching, casts, and night splints have failed and symptoms persist for more than 6 months. Since this is a relatively safe procedure, it can be considered before any surgical procedure and may be preferable to try before topical steroid injections. Bilateral cases can be treated with a single anesthesia and full weight bearing can be initiated immediately.

Autologous Platelet Rich Plasma (PRP)

There is growing enthusiasm for the use of growth factors, including pooled blood/platelet concentrates, which, unlike steroids, can stimulate repair processes. Results appear to be comparable to, and sometimes better than, topical steroid injections.

Surgery is usually indicated for refractory cases whose symptoms persist for more than 6 to 12 months despite appropriate conservative management. Prior to surgery, nerve conduction and electromyography

should be considered to determine if the posterior tibial nerve is compressed.

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