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REVIEW ARTICLE

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Plantar heel pain and its 3-mode 4-stage treatment

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Abstract The most common cause for heel pain is plantar fasciitis. The diagnosis can usually be made by clinical examination, but sometimes ENMG (electroneuromyography), ultrasound, and magnetic resonance imaging examinations are helpful. Other reasons for heel pain, e.g., nerve entrapments, atherosclerosis/ischemia, and fat pad degeneration, should be excluded. Plantar fasciitis can also present a symptom of chronic seronegative spondyloarthropathies or reactive arthritis. In the case of common plantar fasciitis, three different modes of treatment can be administered, namely, (1) anti-inflammatory and analgesic treatment, (2) rest and diminution of the strain at the insertion, and (3) maintenance of the tension and flexibility of the soft tissues. A simple four-step treatment plan algorithm, based on symptoms, their duration, and response to treatment, is presented. Operative treatment is seldom needed if the algorithm is correctly followed. Operative treatment is recommended only when the pain remains resistant to conservative treatment after more than 1 year. For operative treatment, partial release of the fascia close to insertion to avoid flat foot and secondary strain on the calcaneocuboid and midtarsal (Lisfranc) joints is our preferred option.

Key words Enthesopathy · Fasciitis · Heel pain · Treatment

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Introduction

More than 200 diseases, abnormalities, or dysfunctions of the human foot have been described (Table 1). Among them, plantar fasciitis is the most common cause of heel pain. Up to 40% of the population suffer from painful feet problems at least once during their lifetime, and more than 10%, at some time during their life, suffer from heel pain that is caused by an inflammation in the proximal insertion of the plantar fascia. Occasionally plantar fasciitis is complicated by nerve entrapment of the nerve branch to the m. abductor digiti minimi and periosteum from the lateral plantar nerve. The etiology of plantar fasciitis remains unclear. Almost all plantar fasciitis patients improve significantly with conservative (nonsurgical) treatment.[1–5] There is no uniform specific treatment plan recommendable for all patients, i.e., treatment plans should always be made individually as they are dependent on symptoms, their duration, and response to various modes of structured treatment, and also the contribution of the patient. Evidence-based knowledge of the treatments is still limited. It is apparent that the patients are often referred for operative treatment before adequate conservative treatment has been carried out.

Anatomy

Plantar fascia is a strong band of fibers that maintain the medial longitudinal arch.[6] A total spontaneous or traumatic rupture of the fascia leads to a flat foot. During walking, when the heel strikes the ground at the first phase of the gait cycle, the foot pronates and the fascia allows flattening of the foot. This stretches the fascia, absorbs part of the shock, and allows the foot to accommodate to irregularities in the walking surface.

The plantar fascia comprises three different parts, the medial, the middle, and the lateral part. The fascia is attached to the tuberosity of the calcaneus. In the calcaneal

Table 1. Some causes of heel pain

I. Bone, cartilage and joints		
	Apophysis calcanealis (Sever's disease)	
	Arthrosis	
	Subtalar instability	
	Stress fracture	
	Ongoing pain after stress fracture	
	Os trigonurum talii posterior syndrome	
	Pes valgus	
	Planovalgus	
	Cavus foot	
II. Soft tissue problems		
	Fasciitis plantaris	
	Subtendinous bursitis (anterior Achilles tendon bursitis, Albert's disease)	
	Subcutaneous calcaneal bursitis (posterior Achilles bursitis)	
	Haglund's deformity	
	Partial or total rupture of Achilles tendon	
	Inflammation of Achilles tendon	
	Peritendinitis of Achilles tendon	
	Synovitis of peroneus, tibialis posterior, or flexor hallucis longus tendons	
	Rupture of peroneus, tibialis posterior, or flexor hallucis longus tendons	
	Tendinosis of flexor hallucis longus	
	Subtalar bursitis	
	Inflammation of short flexor origin	
III. Nervous system		
	Tarsal tunnel syndrome (entrapment of the tibial nerve)	
	Entrapment of the medial calcaneal branch of the tibial nerve	
	Entrapment of medial plantar nerve	
	Entrapment of lateral plantar nerve	
	Entrapment of first branch of lateral plantar nerve to abductor digiti minimi (often connected to plantar fasciitis)	
	N. suralis entrapment or injury (for example after Achilles rupture operation)	
	Neurinoma of medial, lateral, or other plantar nerves	
	Neuritis of the medial, lateral, or other plantar nerve	
	Radiculopathy	
IV. Trauma		
	Soft tissue contusion	
	Stress fracture	
	Acute fracture	
		calcaneus
		posterolateral talar tubercle
		talus
	Wound(s)	
	Plantar fascia rupture	
V. Vascular causes		
	Peripheral vascular disease	
	Thrombosis of deep or superficial veins	
	Arterial thrombosis	
VI. Arthritides		
	Seronegative spondyloarthropathies, e.g.	
		ankylosing spondylitis
		enteropathic spondyloarthropathies
		Reiter's syndrome
		psoriatic arthropathy
	Rheumatoid arthritis	
VII. Other causes		
	Tumors (cysts, osteoid osteoma, osteosarcoma)	
	Infections (soft tissue or bone)	
	Fat pad atrophy	
	Abnormal gait	
	Compensatory stresses	

part, the fibers of the fascia are longitudinal while more distally towards the toes they expand and branch out. The lateral plantar nerve runs under the fascia to the lateral side of the sole, whereas the medial calcaneal branch passes over the insertion (Figs. 1 and 2).

Etiology

Plantar fasciitis can affect patients from childhood to older age, but is most common in middle-aged women and young

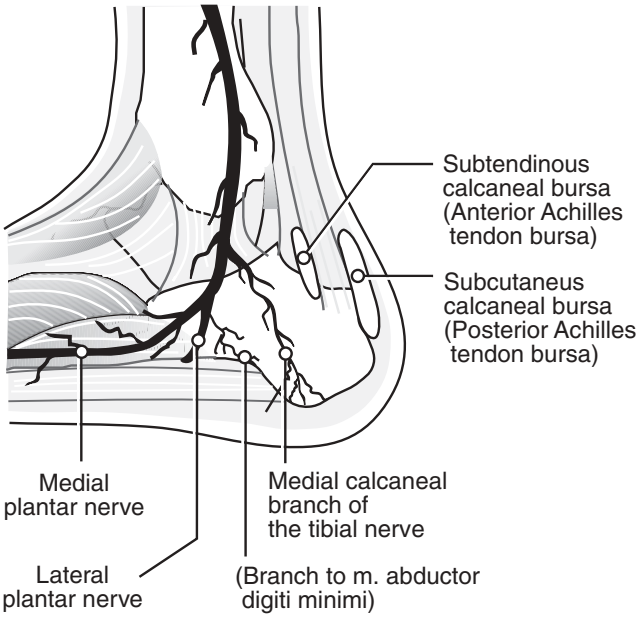

Fig. 1. Medial view of the foot and the tibial nerve with its branches

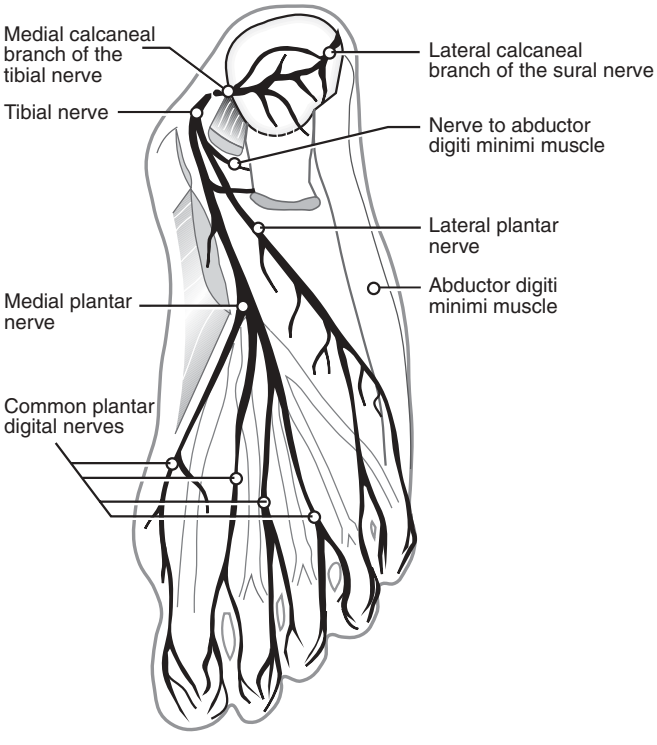

Fig. 2. Plantar view of the foot and its nerves

male athletes. It is also common among professions where the persons have to stand or walk extensively and among overweight persons. Plantar fasciitis is especially common in long-distance runners as a result of overuse.

Many theories have been presented concerning the natural history of the inflammation in the plantar fascia. After a predisposing factor, such as microtrauma or overexertion, the plantar fascia degenerates, its elasticity decreases, and the strain and thus traction of the insertion increases. As the tension at the insertion increases this site is likely to become irritated, resulting in inflammation. In this scenario inflammation following single or repeated trauma is the cause of the heel pain.[7] Biopsies of the irritated fascia display granulation tissue, fibroblast proliferation, and fibrosis. The fascia is normally about 3 mm thick, but in fasciitis more than 15-mm thick fasciae have been reported.[8] The subcalcaneal spur is a result of the inflammation rather than the cause of it. Insertitis leads to growth of new bone in the spur.[9] The spur runs parallel to and actually inside the fascia, and thus does not push out as a spike. A spur is found in 15%–40% of persons with no history of foot pain. About 50% of patients with heel pain do not have a spur.[4,9] The spur (Figs. 1 and 2) can cause entrapments of the branch of the lateral plantar nerve to the abductor digiti minimi muscle. Entrapment, at the site of navicular bone, of the medial plantar nerve may cause similar heel pain to plantar fasciitis.

Periosteal inflammation and bursitis have been presented as alternative causes of heel pain. Also, degeneration of the fat pad and seronegative arthritis can cause inflammation, even though they are considered more as differential diagnostic entities rather than causes for true plantar fasciitis. The thickness of the fat pat decreases after the age of 40 years, which may predispose to plantar fasciitis. There is no consensus on the etiology of this common disorder.

Plantar fasciitis occurs as an enthesopathy in patients with a seronegative arthropathy. In systemic rheumatic diseases enthesitis (insertitis) can occur for endogenous, unknown causes or as a reaction to a triggering infection leading to reactive arthritis. Alternatively insertitis can be part of Reiter's syndrome, psoriatic arthropathy, ankylosing spondylitis, enteropathic spondyloarthropathy, and other chronic seronegative spondyloarthropathies, but seldom of seropositive rheumatoid arthritis.[10,11] Enthesopathy can occur in any insertion of fasciae, tendons, or ligaments. The name refers to the site where the connective tissue fibers enter into bone. In the case of plantar fasciitis, the name of the condition specifies both the site of the problem as well as the structure involved. The most common inflammatory enthesopathies are plantar fasciitis, (peri)tendinitis of the Achilles tendon, and inflammation at the site of the insertion of the joint capsules of the facet joints and of annulus fibrosus of the intervertebral discs in ankylosing spondylitis and related diseases. Enthesopathies start with inflammatory soft tissue involvement, but when such a condition becomes chronic it leads to bone formation. Plantar fascia enthesopathy is usually treated by a rheumatologist and benefits from conservative treatment.[11,12]

Diagnosis

The diagnosis of plantar fasciitis can usually be made by clinical examination. The anamnestic data form the basis for the diagnosis. Pain is often sensed as deep pain and is located in the middle of the heel or somewhat medial to it. Walking, running, standing, or other exertion increases the

Table 2. Nerves of the foot and their most typical impingement sites

Nerve	Site of impingement	Motor distribution	Sensory distribution	Observations
TIBIAL NERVE				
Tibial nerve	Tarsal tunnel tibialis posterior, fl. dig. longus et brevis	M. triceps, plantaris, popliteus		Tinel's sign is positive
– flexor hallucis longus (branches of the tibial nerve)				
– sural nerve	In foot and leg	No motor distribution	Posterolateral skin of heel and cruris	
– medial calcaneal nerve	Heel	No motor distribution	Heel, posteromedially	
– medial plantar nerve	Plantar, navicular bone fl. digitorum brevis, limbricales, I, II	M. abductor hallucis, fl. hallucis brevis	Medial side of plantar foot and toes	
– lateral plantar nerve	Plantar, insertion of the fascia quadratus plantae	M. abductor digiti minimi	Lateral side of plantar foot and toes	
– branch to m. abductor digiti minimi	Calcaneus spur/insertion	M. abductor digiti minimi	No sensory distribution	May be caused by heel spur and fasciitis
COMMON PERONEAL NERVE				
Superficial peroneal nerve	In foot and leg	M. peroneus longus et brevis	Anterior and dorsal leg	
Deep peroneal nerve	In foot and leg	M. tibialis anterior, extensor hallucis proprius, extensor digitorum brevis, etc.	Skin of the first interdigital space Periost of tibia and fibula	
Intermetatarsal nerves (Morton)	Tarsal bones, distal end	No motor distribution	Toes	Most common entrapment in foot

pain. The pain is typically most intensive in the morning when starting to walk, decreases after a while, and increases after exertion. Impingement of sensory nerves causes sharp pain and burning feeling of the area innervated by the nerve (neuropathic pain) and also often causes loss of sensation (hyposensibility). Dysfunction of motor nerve branches causes loss of muscle strength and muscle atrophy.

By inspection, inflammation or swelling is rarely seen in the foot in the heel area. If the foot is swollen, more thorough examinations are indicated.

Tenderness is usually found in the middle of the heel, in the proximal insertion of the plantar fascia. It is best to palpate the fascia when the tendon is stretched with the ankle and toes in dorsiflexion. The pain spot is beneath the anteromedial prominence of the calcaneal tuberosity, with the maximal pain spot often a little medial to the insertion of the fascia. Pain is sometimes located medially from the midpoint toward the insertion of the abductor hallucis muscle or more distally in the fascia.[12] The most intensive pain occurs typically after a period of inactivity, for instance when the patient wakes up in the morning or starts to walk, or also after exertion. The patient feels burning pain along the arch of the foot.

Differential diagnostic disorders are numerous and include infections, bone tumors, nerve entrapment, fat pad degeneration, ischemia, and stress fracture (Table 1). A patient history and a proper physical examination, including a detailed localization of the pain area, are essential for diagnosis. In retrocalcaneal bursitis the pain is located close to the insertion of the Achilles tendon. Achilles tendinitis causes pain and swelling in the tendon, whereas rupture causes local pain at the site of the rupture. In osteochondrosis of the apophysis of calcaneus in children (Sever's disease), pain occurs in the dorsal part of the calcaneus.

Electroneuromyography (ENMG) is used to verify the tarsal tunnel syndrome, although more distal nerve entrapments can also be detected (Table 2). Anatomical variations may make the diagnosis difficult. Therefore, performance and interpretation of the ENMG require experience and knowledge from the investigator. Electroneuromyography is indicated when the patient has typical neuropathic pain, like aching sharp pain that radiates proximally and worsens during night-time, walking, or standing. Loss of sensation or loss of motor function and/or muscle atrophy are definite indications for ENMG. Entrapment of the tibial nerve (the larger of the two terminal branches of the sciatic nerve) in tarsal tunnel syndrome causes burning, unpleasant pain in the foot, and paresthesias in the toes after exertion or when lying on the bed (Table 2). Symptoms get worse during walking or standing, and night pain is common. Tinel's sign is positive, when knocking on the tarsal canal causes pain in the foot. Sometimes the plantar flexion of toes is weak. Entrapment of the medial calcaneal branch of the tibial nerve (Figs. 2 and 3, Table 2) causes pain in the heel (Table 1). The tibial nerve divides into two major plantar branches, the medial and lateral plantar nerves. Entrapment of the medial plantar nerve (Figs. 2 and 3), often seen in long-distance runners, causes loss of sensation of most of the toes (Fig. 4). Entrapment of the lateral plantar nerve (Figs. 2 and 3) causes pain on the lateral side of the plantar foot and toes

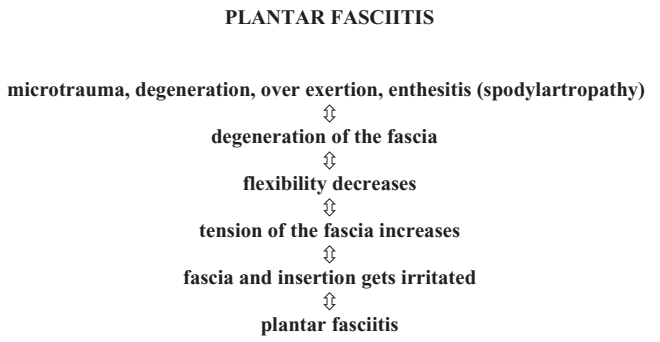

Fig. 3. Pathomechanism of plantar fasciitis (from Tountas and Fornasier[7]; with permission)

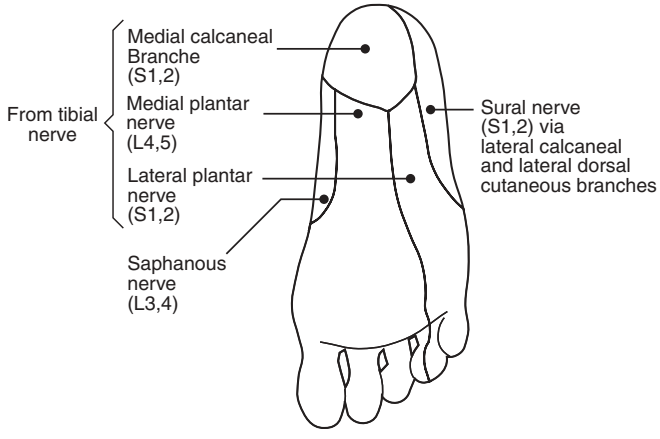

Fig. 4. Denervation of the skin in the foot

(Table 2, Fig. 4). The calcaneal spur can cause an entrapment of the first nerve branch of lateral plantar nerve (to m. abductor digiti minimi) (Figs. 2 and 3) and heel pain. Compression of nerve branch to m. digiti minimi leads to damage of the motor branch to the muscle. This nerve does not have sensory branches to the skin, but some fibers sense pain from the periosteum of calcaneus. According to one theory, the entrapment of this nerve is suspected to be the most common cause of the heel pain, not the fasciitis itself. The role of electromyographic and nerve velocity studies in detection of this nerve-entrapment phenomenon is not well defined. Entrapment of deep peroneal nerve and superficial peroneal nerve (branches of the common peroneal nerve, the smaller of the two terminal branches of the sciatic nerve) cause pain in the dorsal parts of the foot (Table 2). In addition, Fig. 4 shows the area of the sole innervated by the sural nerve, which is mainly formed by a cutaneous branch of the tibial nerve, separating from it as distant as at the back of the knee. Figure 4 also shows the area innervated by the saphenous nerve, which is a branch of the femoral nerve.

Plantar fasciitis can be verified using ultrasound, bone scan, and magnetic resonance imaging (MRI). The calcaneal spur is seen on X-ray. These investigations, however, are seldom necessary and often rather unspecific. More thorough investigations are mostly useful for exclusion of other diseases. Infections cause typical changes in C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and blood count, and microbial cultures of blood and tissue samples are positive. X-ray is indicated to exclude tumors, fractures, and other possible diagnoses. The MRI, however, better reveals both tumors and stress fractures. In the beginning stress fracture is not always seen on regular radiographs, but with the callus formation becomes visible.

Erythrocyte sedimentation rate, CRP, and blood count should be checked to demonstrate the acute phase response/inflammation of plantar fasciitis in context with chronic inflammatory diseases. They actually form part of the hypersedimentation syndromes, in which the ESR can be very high, 50–100 mm/h, without any apparent cause for it (such as angina, tonsillitis, upper urinary tract infection, etc.). Rheumatoid factor is usually negative (thereof "seronegative"). More comprehensive evaluations are seldom needed. Also, patients with spondyloarthropathy may develop a bone spur, which nevertheless is rarely the cause of the pain.

Treatment

A correct diagnosis and analysis of the severity and duration of the problem is essential for good outcome. All patients should be informed of the nature and likely outcome. It is important to carefully explain to the patient that it is not the bone spur that causes the pain, being not the cause but a consequence of the inflammation. By imparting useful information, the surgeon will be able to motivate the patient for successful, but often long-lasting conservative treatment.

Apart from patient education, conservative (nonoperative) treatments can be divided roughly into three groups based on the mode of action: (1) treatment reducing pain and inflammation (e.g., anti-inflammatory and analgesic treatment), (2) treatment reducing the stress of soft tissue (e.g., rest and various supportive orthopedic devices or pads), and (3) treatment to maintain the tension and flexibility of the soft tissues (e.g., various self-administered exercises and physiotherapy). At least one mode of treatment method for each of these three groups should be included in the treatment. Methods for treatment are insoles, good shoes, stretching, physiotherapy, ice/cold, nonsteroid anti-inflammatory drugs (NSAIDs), analgesics, night splint, local steroid injections, and immobilization. There is preliminary evidence for the effectiveness of shock wave therapy.[13–15] The earlier the patient receives treatment, the better the results. In the Cochrane database Crawford et al.[16] found no evidence of beneficial effect of therapeutic ultrasound, low-intensity laser therapy, exposure to an electron-generating device, or insole with magnetic foil.

Therefore, the three different modes of treatment already mentioned can be administered at four different steps according to an algorithm we use in our unit (Fig. 7). All

THREE STEP TREATMENT PLAN OF PLANTAR FASCIITIS

PLANTAR FASCIITIS

STEP 1 **mild symptoms for weeks**	**GOOD SHOES$_1$, INSOLES$_1$** **NSAID 1-2 WEEKS$_2$, ICE, REST$_2$** **STRECHES$_3$**
STEP 2 **severe symtoms** **symtoms for months**	**NIGHT SPLINT$_3$, PHYSIOTHERAPY$_3$** **STEROID INJECTIONS$_2$, LONGER NSAID TREATMENT$_2$**
STEP 3 **Before surgery**	**CAST$_1$, (ESWT$_2$)**
STEP 4 **Symptoms for 12 months or more** **Painful condition that continues after conservative treatment**	**Surgery$_4$**

Fig. 5. The four treatment steps of plantar fasciitis. Target of the treatment is marked with index numbers$_{1,2,3}$. For mild and new symptoms, we recommend the Step 1 program. For the most effective treatment, one method from three different colored treatment targets is included (Treatment target$_1$ = keeps up the flexibility, Treatment target$_2$ = reducing the stress of soft tissue, Treatment target$_3$ = reducing inflammation and pain). For more severe or long-lasting symptoms, Step 2 treatments can be added to the treatment plan. Step 3 treatments should be considered before surgery

patients should receive the treatments of the first step, at least one mode of treatment in each category. If there is severe pain or the pain has lasted for a long time, at least some treatments of step 2 (e.g., night splint to stretch the plantar fascia, corticosteroid injections to diminish inflammation and pain) should be included in the treatment plan. The aim is to maintain the flexibility of the soft tissue, to treat pain and inflammation, and to avoid stress. Step 3 treatments should be tried at least before surgical treatment is considered because ESWT (extracorporeal shock-wave treatment) and casting have almost no complications and some promising results have been reported.

In the treatment of plantar fasciitis with mild symptoms, the first step combines rest with good shoes and insoles, together with stretching exercises for 10 min a day and anti-inflammatory treatment with NSAIDs and ice (Fig. 5).[17] Patients should avoid exertion (stress, strain) of the heel. Viscoelastic insoles or heel cups are recommended. Properly done stretching is important and motivation of the patient essential. Stretching can be instructed by a physical therapist and should be done regularly and daily 2–3 times for a few minutes at time for several weeks (Fig. 6). In a study by Porter et al.,[18] there was no difference between intermittent or sustained stretching, but both sustained and intermittent Achilles tendon stretching exercises were effective nonsurgical treatments for painful heel syndrome. NSAID medication can be used to decrease pain and for their anti-inflammatory effect. Analgesics like the cyclooxygenase-3 selective paracetamol or mild opioids like tramadol or codeine-containing drugs are occasionally used in severe inflammatory enthesopathy of the plantar fascia. Repeated ice/cold treatments can also be recommended.

Step 2 treatments are for patients with severe symptoms or symptoms lasting for months. A night splint is especially effective for a patient having a lot of pain in the morning when starting to walk (Fig. 7). We use it for periods of 4–6 weeks, depending on how the patients respond to treatment. A night splint keeps the plantar fascia stretched and

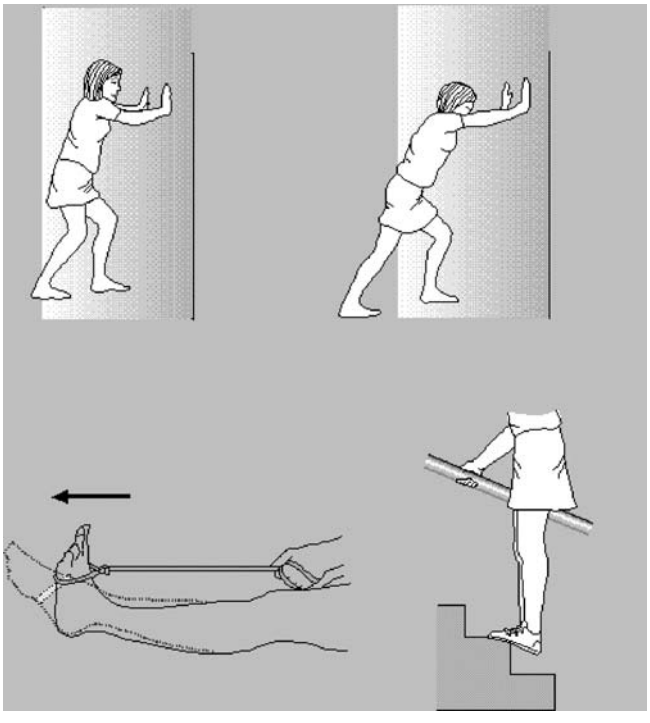

Fig. 6. Regular stretching keeps the inflamed fasciae elastic and decreases tightness, contracture of the fasciae, altered pulling, and so also new microtrauma to insertion/fasciae. Stretching might induce symptoms in the beginning, but it is an essential part of rehabilitation of inflamed fasciae. We instruct our patient to do exercises 3–5 times daily, each exercise 3–5 times for about 10 s each stretch

prevents contractures of the fascia (Fig. 7). Many studies suggest that the night splint is an effective treatment,[19–21] even when compared with corticosteroid injections. Various physiotherapy and foot muscle gymnastics are commonly advocated. Corticosteroid injections can cause fat pad atrophy and in the worst cases may be complicated by infections, osteomyelitis, or iatrogenic rupture of the plantar fascia. There is no evidence that a night splint would

Fig. 7. A night splint keeps the ankle (also the toes) in dorsiflexion. It should be used for 4–6 weeks depending on the patient's symptoms

reduce chronic pain that has lasted for more than 6 months.[16] The general consensus maintains that the earlier splint treatment or physical exercises are started, the better are the results. Corticosteroid injections, e.g., 1 ml of methylprednisolone 40 mg/ml (preferably with local anesthetics), are usually effective, but less successful in chronic cases. The injection can be repeated three times at 2–4-week intervals, but should not be continued if the first injection fails to give any relief. NSAID medication can be supplementary to all the treatments mentioned above. Anti-inflammatory medication can be used as a course for at least 10–14 days and in persistent cases for months.

Step 3 treatment, ESWT, has as yet limited evidence for its effectiveness. Some promising results have been published,[12,14] but some more recent results suggest that ESWT would not be effective.[22,23] However, this treatment does not include complications and can be introduced to patients as one treatment option, at least before considering surgery. Immobilization can occasionally provide relief for the patient. Soft cast, normal cast, or walker orthoses can be used, mainly to help/force the patient to rest. Some studies suggest that casting is effective, even in recalcitrant cases.[9,17,24]

Fasciitis is more common in feet with a tendency for pronation during walking and in feet with a low arch. In these cases an insole that lifts up the arch of the foot can irritate the foot and actually induce fasciitis. In these cases an elevation of the heel in the shoe is recommended.

In inflammatory enthesopathies patients should be directed to a rheumatologist for additional treatment of the general disease. For plantar fasciitis, orthoses and immobilization have not been proven to be as effective for patients with spondyloarthropathies, but soft good shoes and insoles may be helpful. Local corticosteroid injections are effective in these patients and NSAIDs should be used for treatment. Ice/cold treatment can be used, but overuse is avoided and stretching is recommended.[25] If these treatments do not show improvement, a night splint can be added, for example after 6 weeks of primary treatment. Spondyloarthropathy patients benefit from conservative treatment. Operative treatment is not recommended.[25]

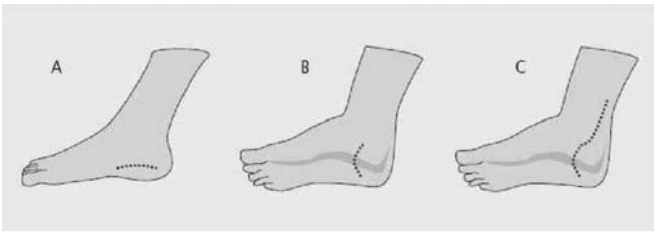

Fig. 8. Different incisions used for surgical treatment of plantar fasciitis

Operative treatment (step 4)

Operative treatment should be considered first after a long (at least 1 year) conservative treatment period. Many methods for surgical treatment of plantar fasciitis have been described in the literature.[4,7,27–29] Unfortunately, however, there are no randomized controlled trials of surgery for heel pain, which makes judgment of the benefits of surgical treatment difficult. Methods used include the release of all soft tissue, release of calcaneal or plantar nerve branch, osteotomy of the calcaneus, extirpation of the spur, or fenestration.[30] Some of these methods can no longer be recommended. The du Vries method includes an incision along the foot on the medial side (Fig. 8a) so that the medial side of calcaneus is exposed and a small, about 1-cm long, piece of the fascia is removed.[31] Some surgeons have used a longitudinal incision, shorter or longer (Fig. 8b,c). When a release of the tarsal tunnel is needed, a longer incision should be used (Fig. 8c). Baxter et al.[1] described a technique where the fascia of the abductor hallucis longus muscle is split in order to enable more space for the lateral plantar nerve. After that a 1-cm long slice of the medial part of the proximal plantar fascia is removed from its insertion to the calcaneus, which enables the release of the nerve. An existing spur is removed. Even if entrapment of the nerve to m. abductor digiti quinti is suspected to be the cause of pain, a nonoperative treatment for at least 6 months should precede any operative treatment.[32] Baxter et al.[1] recommend excising only about one third of the plantar insertion, while du Vries recommends that the whole insertion be released. The excision of the whole insertion can increase the stress of the lateral parts of the foot and cause pain in the adjacent joints, especially in the calcaneocuboid joint and the midtarsal (Lisfranc) joints. Total release may cause flat foot, but this may be avoided by not letting the patient put weight on the foot for some time after surgery. A flat foot, as is well known, changes the whole biomechanics of the foot and may cause a consequent problem.

Endoscopic release of the fascia has been associated with complications. Barrett et al.[28] operated on 652 patients endoscopically, 97% of whom got relief from their pain. However, 53 of these patients had a total of 62 complications. Also, some reports have been published describing a percutaneous fasciotomy.[29]

Patients should be advised of possible complications when considering either open or endoscopic release. We recommend an open procedure, because endoscopic results have been reported to increase the risk of tibial nerve damage. Partial release of the fascia and decompression of the

nerve branch to m. adbuctor digiti minimi and periosteum is an option we currently believe to be the method of choice.[4] There does not appear to be one common treatment of choice that can be agreed on. The operative method has to be chosen after the theory each believes in, and with the incision the surgeon is most comfortable with, until research has produced true evidence. The heel spur theory leads one to choose resection of the spur; the chronic inflammation theory release of the fascia and the nerve entrapment theory brings one to perform neurolysis of the nerve branch. Because we do not think the spur is the true cause of heel pain, we recommend extirpation of the calcaneal spur only if it is considered to grow rapidly thus inducing mechanical discomfort.[1,7] A partial release of the fascia is recommended to avoid flat foot and other changes in foot biomechanics.

Discussion

Plantar fasciitis in Scandinavian countries is often treated by the general practitioner, who might not be so familiar with the disorder. Most often the results of the conservative treatment are good and operative treatment is seldom needed. Operative treatment should be considered only after a long period of conservative therapy of about 1 year. We suggest a four-step plan to help the physician treat his patients effectively. It is still open to debate as to which of the treatments is the most effective. Effective treatment and an organized treatment plan reduce the stress for both the physician and the patient. This treatment plan should be simple enough to help the general practitioner. The earlier the treatment starts, the more effective it is. Several operative methods have been described, of which removal of the spur is debatable. The patient often associates the pain with the spur, and it might be difficult to convince the patient of the real cause of the pain. We do not recommend the release of all soft tissue, excision of calcaneal or plantar nerve branch, osteotomy of the calcaneus, or fenestration. We recommend effective conservative treatment, and surgery only for those few patients who really need it. Excision of the medial third fascia is the recommended option because total fascia release impairs the biomechanics of the foot.[33]

Acknowledgments This work is dedicated to the memory of the Finnish strongman in orthopedics and traumatology, Seppo Santavirta (5 December 1945–22 June 2005), who tragically passed away only a few days before the Midsummer Night.

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