Curr Phys Med Rehabil Rep
DOI 10.1007/s40141-014-0069-7

Plantar Fasciopathy: A Clinical Review

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Abstract Heel pain is the most common type of foot pain. The differential diagnosis for pain in this region is vast, but plantar fasciopathy is thought to be the most common cause for pain in this region. This article will review the anatomy, biomechanics, pathophysiology, history, physical examination, diagnostic imaging, and treatment options available for plantar fasciopathy.

Keywords Plantar fasciitis · Heel · Fascia · Heel spur

Introduction

The foot is a common region of discomfort, with a reported prevalence of 24 % in patients presenting with musculoskeletal pain to physicians [1]. The heel is reported to be the most common region of pain in this group [2]. The differential diagnosis of heel pain includes plantar fascia disorders, traumatic plantar fascia tears, entrapment of the first branch of the lateral plantar (Baxter's) nerve, heel pad atrophy, plantar fibromatosis, tarsal tunnel syndrome, calcaneal stress fractures, plantar vein thrombosis, and rheumatoid nodules [3••, 4].

Disorders related to the plantar fascia are reported to be the most common cause of heel pain, affecting up to 2 million Americans annually [5]. Although plantar fascia disorders are known to occur in active young runners, the peak incidence occurs in individuals between 40 and 60 years of age [6]. The term plantar fasciitis first appeared in the literature in 1975 by Campbell and Inman, and though the term is still used, plantar fasciopathy has supplanted its use by some clinicians and was first described by Lemont et al. [7, 8]. This change in nomenclature was due to the recognition that in a majority of cases, this condition is degenerative rather than inflammatory [8]. This article will review the anatomy, biomechanics, pathophysiology, history, physical examination, diagnostic imaging, and treatment options available for plantar fasciopathy.

Anatomy and Biomechanics

The plantar fascia is composed of collagen fibers arranged mainly in a proximal-to-distal longitudinal direction, with a minority of fibers running in vertical, transverse, and oblique directions [9]. Although sometimes referred to as an aponeurosis, this term is inappropriate as it refers to tissue with a unidirectional arrangement, and fascia refers to tissue with layers running in multiple directions [9, 10].

This article is part of the Topical collection on *Musculoskeletal Rehabilitation.*

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Published online: 23 December 2014

The plantar fascia is well innervated by Pacini and Ruffini corpuscles that are responsible for mechanoreception [11]. A recent study has also noted the presence of hyaluronan in the plantar fascia, allowing the different fibrous bundles to glide over one another [9].

The plantar fascia is composed of three distinctive cords: central, medial, and lateral [3••,9]. The central cord is triangular and thicker at its proximal third, and becomes thinner and wider distally [3••]. It is also the thickest band, arising from the posterior aspect of the medial calcaneal tuberosity and traversing over the plantar surface of the flexor digitorum brevis muscle before dividing into five digitations [9, 11]. The deep branch of these digitations inserts onto the capsule of each metatarsophalangeal joint, and the superficial branches send fibers to the natatory ligament and divide into two sagittal septa that blend and insert on the various structures adjacent to the metatarsophalangeal joints [11]. The central cord of the plantar fascia is thought to be the primary region that becomes degenerated in plantar fasciopathy [3••].

The lateral cord of the plantar fascia originates off the lateral portion of the medial calcaneal tuberosity [11]. It, like the central cord, is thicker proximally and thinner distally [11]. It covers the abductor digiti minimi as it traverses the foot, and is continuous medially with the central cord and laterally with the dorsal fascia [11]. The distal portion of the lateral cord divides into two bands [11]. The medial band inserts onto the planar plate of the third and fourth metatarsophalangeal joints. The lateral band inserts onto the base of the fifth metatarsal, becoming the metatarso-calcaneal ligament [11]. Enthesopathy of the lateral band of the lateral cord of the plantar fascia has recently been reported as a cause of pain at the fifth metatarsal tuberosity [12].

The medial cord of the plantar fascia originates from the medial calcaneal tuberosity and is thin throughout its course. It covers the abductor hallucis muscle and inserts onto the first metatarsophalangeal joint [9]. The medial cord blends medially with the dorsal fascia and laterally with the central band of the plantar fascia [3••, 11]. This band is thought to be the least clinically significant of the three [3••].

Proximally, all three bands of the plantar fascia originate from the medial calcaneal tuberosity in a fibrocartilaginous configuration [13]. Histologically, this region is characterized by the collagenous midsubstance (dense fibrous tissue) that is successively replaced by uncalcified fibrocartilage, calcified fibrocartilage, and finally bone [13]. A connection between the plantar fascia and the Achilles tendon has been noted to be present via a thin layer of periosteal fibers. At a younger age, this connection is thought to be more robust, thinning with age, resulting in the plantar fascia only having a connection with the Achilles paratenon later in life [9].

Hicks, who first described the "windlass mechanism" noted: "The effect was as though a cable had been wound one-quarter of a turn on to the drum of a windlass, the drum of the windlass being the head of the metatarsal, the handle which does the winding being the proximal phalanx and the cable which is wound on to the drum being the plantar pad and the plantar aponeurosis. The effective length of the cable was shortened by, in the case of the first ray, about 1 cm. Actually, the aponeurosis did not shift distally because of its attachment to the calcaneum; instead, it was the windlass which shifted, being pulled 1 cm proximally towards the calcaneum and the arch was thereby made shorter and higher" [14]. Since Hick's description of the plantar fascia, the structure's main role continues to be thought of as a support structure, particularly of the medial longitudinal arch of the foot during the weight-bearing portions of the gait cycle [13, 15]. Without this mechanism, efficiency in the gait cycle would be lost [15]. In addition, increasing tension of the Achilles tendon has been associated with increased strain on the plantar fascia [16, 17].

Pathophysiology

The pathophysiology of plantar fascia disorders has anecdotally been divided into acute, subacute, and chronic phases [18]. The first two phases were originally thought to involve an inflammatory component [18–20]. However, recent research suggests that this condition is predominantly degenerative. Lemont et al. were the first to describe the term plantar fasciopathy [8]. They described myxoid degeneration with fragmentation and degeneration of the plantar fascia and bone marrow vascular ectasia in the plantar fascia of 50 cases undergoing heel spur surgery with the diagnosis of chronic plantar fasciitis. The underlying cause of this degenerative process is thought to be micro tears similar to tendinosis [21••, 22]. This has been supported by imaging findings of a thickened plantar fascia in patients diagnosed with plantar fasciopathy, which can be found in the proximal or distal central cord of the plantar fascia [3••, 23]. Although the exact mechanism upon which the plantar fascia undergoes this degenerative process is unknown, a recent systematic review did associate several risk factors and categorized them as intrinsic and extrinsic (Table 1) [21••]. Familiarity with the risk factors improves the physician's ability to diagnose plantar fasciopathy and council patients on preventative measures.

History and Physical Examination

Figure 1 outlines the clinical approach to diagnosing and treating plantar fasciopathy. Plantar fasciopathy has

Table 1 Intrinsic and extrinsic risk factors for plantar fasciopathy [21••]

Intrinsic	Extrinsic
Higher age	Physical load on ligament
Average age at presentation 10 years higher than controls who presented for other reasons	Excessive foot pronation
Increased prevalence in older athletes	Rearfoot eversion and arch height collapse
Age-related degenerative changes may result in fascia's inability to resist normal tensile loads	Repetitive microtrauma
Associated with increased heel fat pad thickness and loss of elasticity	Occupation
Decreased fascial elasticity associated with decreased shock-absorbing capabilities in older patients	Prolonged weight bearing
Obesity	Change in walking or running surface
Higher BMI associated with increasing heel fat pad thickness and loss of heel pad elasticity	Standing on hard surfaces
Significant positive correlation between BMI and PF thickness causing chronic stretch, overloading, and focal pressure of plantar fascia	Environment
Sex	Inappropriate footwear
Current literature inconsistent	Lifestyle
Ethnicity	Rapid increases in activity levels allied to physical demands of sport or occupation
No reported associations	Sleeping Posture
Biomechanical dysfunction and anatomical variants	Can contribute to posterior leg muscle contraction
Reduced range of ankle joint secondary to tight Achilles tendon strains plantar fascia	Sport
Tightness of posterior lower limb muscles and specifically hamstring tightness	Overuse injury combined with running surface
Decreased 1st metatarsophalangeal joint range of extension due to tight Achilles tendon	Poor technique
Flexor digitorum brevis tendinopathy secondary to stress shielding	Training errors
Calcaneal spur	High intensity
Plantar fascial thickening	Fatigue
Acquired systemic diseases	Repetitive loading
No association with systemic factors	Muscle dysfunction and inflexibility
Rheumatoid arthritis	
Ankylosing spondylitis	
Diabetes mellitus where micro/macro vascular impairment results in accelerated fasciosis	
Chemotherapy, retroviral infection, and rarely gonococcus and tuberculosis	
Major trauma (laceration/puncture wound, previous foot surgery)	
No reported associations	
Estrogen levels	
Low estrogen levels in female athletes leads to reduced collagen elasticity	
Vascular perfusion of ligament	
Reduced vascular supply to plantar fascia and subsequent poor nutrition	
Fluoroquinolone antibiotics	
A tendon (Achilles) association exists but none in ligaments to-date	
Inherited systemic diseases	
No association has been reported	
Genetic	
Potential gene variants (based on tendon studies)—COL5A1, MMP1, MMP3, MMP8, MMP10, MMP12, GDF5, TGFB, ADAMTS1, ADAMTS2, ADAMTS4, ADAMTS5, ADAMTS15, TIMP1, TIMP2, TIMPM3, TIMP4	

BMI body mass index

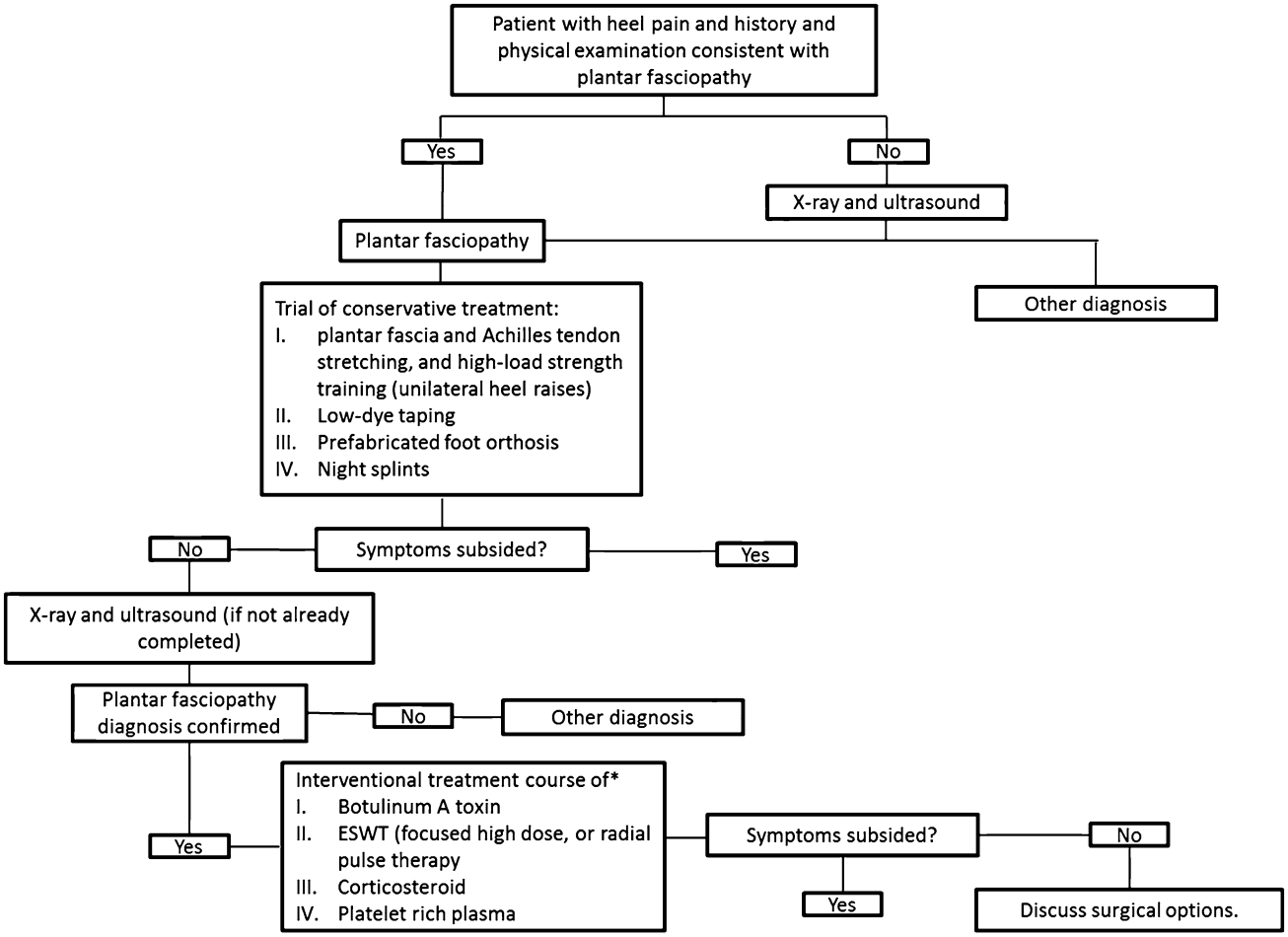

Fig. 1 Algorithm for the evaluation and treatment of patients with plantar fasciopathy. *Option I and II have not been compared directly against each other, but both have stronger evidence than III and IV

traditionally been described as a clinical diagnosis, based on history and physical examination findings [24]. Patients with plantar fasciopathy report the insidious onset of inferior medial heel pain, which is markedly worse with the first steps in the morning and after prolonged weight bearing [4]. Pain in this region has been found to progressively decrease with activity but worsens overall by the end of the day [25]. The majority of patients present unilaterally, but up to 30 % of cases have been reported to involve both feet [6]. Intrinsic and extrinsic factors associated with plantar fasciopathy that can be appreciated on history include increased age, increased body mass index, prolonged weight-bearing activities, inappropriate footwear, rapid increase in activity levels, and sporting factors (overuse, poor technique, high intensity, fatigue, and repetitive loading) (Table 1) [21••].

Although tenderness to palpation of the medial calcaneal tuberosity is a hallmark of plantar fasciopathy, tenderness of the whole plantar fascia may also be seen [4]. Intrinsic and extrinsic factors associated with plantar fasciopathy that can be appreciated on physical examination include reduced ankle joint dorsiflexion, tightness of hamstring muscles, excessive foot pronation, rear-foot eversion, and pes planus or cavus foot deformities (Table 1) [21••]. Provocation of pain occasionally occurs when the patient stands on their toes or with passive metatarsophalangeal joint dorsiflexion due to the windlass mechanism [24]. If reduced range of ankle dorsiflexion is noted on examination, the Skilverskiöld test can be performed, which differentiates between primary contracture of the gastrocnemius muscle itself and of the gastrocnemius–soleus complex [26]. The test is performed by measuring ankle dorsiflexion range of motion with the knee in full extension and in 90° of flexion [26]. An isolated gastrocnemius contracture is noted if ankle dorsiflexion improves with knee flexion and a gastrocnemius–soleus complex contracture if there is no change in ankle dorsiflexion with knee flexion [26]. This decrease in ankle dorsiflexion has been proposed to be a cause or effect of plantar fasciopathy. Interestingly, patients with unilateral plantar fasciopathy were found to have decreased ankle dorsiflexion in the contralateral unaffected side, leading the authors to

conclude that the loss of ankle dorsiflexion range of motion was a cause rather than an effect of plantar fasciopathy [27].

When assessing a patient with medial heel pain thought to be related to plantar fasciopathy, it is important to rule out other potential pain generators in the region. This can be done with history and examination alone in a majority of cases. However, occasionally, additional diagnostic studies are required to rule out other conditions, such as plantar fascia tears, entrapment of the first branch of the lateral plantar (Baxter's) nerve, heel pad atrophy, plantar fibromatosis, tarsal tunnel syndrome, calcaneal stress fractures, and plantar vein thrombosis [3••, 4].

Diagnostic Imaging

On plain radiography, heel spurs are frequently identified in patients with heel pain, but their presence is not specific for plantar fasciopathy [28]. Occasionally, a swollen plantar fascia may be identified on plain radiographs [29]. Although the role of plain radiographs in the diagnosis of plantar fasciopathy is limited, they are essential in the work up of heel pain, as they can identify other conditions such as calcaneal stress fractures or bony erosions of the plantar fascial attachment and Achilles tendon insertion, which would suggest enthesopathy of these structures, leading the clinician to include a seronegative spondyloarthropathy in the differential diagnosis [28].

Diffuse thickening of the plantar fascia is the hallmark feature of plantar fasciopathy on magnetic resonance imaging (MRI). Pathology is suggested by a thickness >4.5 mm [28]. Additional MRI findings that may be seen in patients with plantar fasciopathy include replacement of the normal low signal on T1 images with regions of intermediate signal, linear bands, or lobules of increased signal [28]. As the plantar fasciopathy progresses, the surrounding soft tissue may have increased signal on fluid-sensitive sequences [28].

A recent systematic review found that ultrasound had a comparable accuracy to MRI in diagnosing plantar fasciopathy [23]. In proximal plantar fasciopathy, the most commonly reported sonographic findings include hypoechoic thickening of the plantar fascia (>4 mm), loss of the fibrillar echotexture, and loss of fascial edge sharpness [3••]. Other findings noted on ultrasound include cortical irregularity of the calcaneus, which is often associated with enthesophytes, and perifascial edema (acute cases) [3••]. Plantar fascia thickness of >4 mm can be seen in asymptomatic individuals, so this finding cannot be used to diagnose plantar fasciopathy in isolation [11]. In these cases, the combination of a history and physical examination suggestive of plantar fasciopathy and additional sonographic findings suggestive of plantar fasciopathy increase the specificity of ultrasonography in the diagnosis of plantar fasciopathy [3••]. With distal plantar fasciopathy, fusiform hypoechoic thickening of the distal central cord of the plantar fascia with loss of normal fibrillar echotexture is seen with sonography [3••]. Distal plantar fasciopathy can be differentiated from plantar fibromas, which also present with plantar fascia thickening, by imaging the plantar fascia in a short axis. Thickening of the entire width of the plantar fascia is indicative of plantar fasciopathy, whereas focal thickening is seen in the case of a plantar fibroma [3••].

Treatment

The treatment options for plantar fasciopathy in the literature are extensive. This section will review well-established techniques organized under conservative, interventional, and surgical categories. New and promising treatment options will also be described and noted as such.

Conservative

Stretching is a common treatment option in the rehabilitation phase of musculoskeletal injuries. In plantar fasciopathy, stretching protocols of the plantar fascia and Achilles tendon (calf muscles) have been described [18]. A prospective randomized control trial compared both stretching techniques and found that patients who stretched the plantar fascia had a greater decrease in pain with walking in the morning and a decrease in maximal pain at 8 weeks follow-up [30]. Interestingly, overall pain improved in both groups [30]. No study has examined the combined effect of stretching both regions versus one. Since stretching both the plantar fascia and gastroc-soleus complex has been shown to be efficacious, one might consider having patients with plantar fasciopathy stretch both structures. Porter et al. studied the effect of duration and frequency of Achilles tendon stretching in plantar fasciopathy, and found no difference between the sustained stretch group (3 min per stretch, 3 times daily) and the intermittent group (5 sets, 20 s per stretch, 2 times a day) at 4-month follow-up [31]. Therefore, either of these techniques can be used.

The addition of soft tissue trigger-point manual therapy (gastrocnemius muscles) may be of additional benefit, as this treatment was found to improve outcomes when used in combination with stretching in comparison to the group that only performed stretching at one-month follow-up [32]. Interestingly, a recent article noted improved outcomes at 3 months with high-load strength training versus plantar-specific stretches [33]. In this trial, high-load strength training consisted of unilateral heel raises, and patients progressively increased weight while increasing

sets and decreasing repetitions [33]. There may be a combined effect of plantar-specific stretching and/or Achilles stretching and high-load strength training, but this has yet to be studied.

In clinical practice, taping is often used in conjunction with the treatment options described above [34]. Several different taping techniques are described in the literature [34]. A recent systematic review found low-dye taping to be the most commonly used technique in the included studies [35–39]. With low-dye taping, the aim was to reduce medial heel pressure by lifting the navicular bone [34]. The second most common technique was calcaneal taping, where the calcaneus was repositioned closer to neutral alignment, which theoretically improves biomechanical positioning of the foot [34, 40]. These studies noted a short-term improvement of symptoms with both taping techniques, and suggested that they be used in conjunction with other conservative treatment options [34].

Prefabricated and custom foot orthoses have been studied for the treatment of plantar fasciopathy. One study noted no difference between both orthoses, but found both to be superior to a sham orthosis with regard to function at 3 months, but of no benefit over the sham orthosis at 12 months [41]. Similarly, two other studies also found no benefit between prefabricated and custom-formed orthoses with regard to clinical improvement, but one noted increased compliance with custom-formed orthoses [42, 43]. In contrast, Pfeffer et al. found prefabricated orthoses (heel pad or cup) to be superior to stretching alone or a custom-made orthosis at 8 weeks follow-up [44]. Previous studies have focussed on orthoses that provide heel pad support, but a recent study described the benefit of a new prefabricated foot orthosis (EZStep), which provides heel pad and medial arch support [45]. There does appear to be a role for the use of orthoses in the treatment of plantar fasciopathy, and research suggests that prefabricated orthoses have similar efficacy to custom-formed orthoses.

Night splints are used in the treatment of plantar fasciopathy with the goal of increasing dorsiflexion range of motion at the ankle. The orthoses can be anterior, posterior, or sock type. Batt et al. were the first to describe a benefit of tension night splinting, finding it provided added benefit over ibuprofen use, a heel cushion, and a gastrocnemius and soleus stretching program at 13 weeks [46]. A subsequent, similarly designed larger study found no difference in outcomes between the groups [47]. Barry et al. compared night splinting versus standing gastrocnemius–soleus stretching, and found the former to provide more benefit [48]. An added benefit of nighttime splinting in combination with the use of a daytime foot orthosis was also found at 2 and 8 weeks follow-up [49]. The literature suggests that there is an added benefit with the use of a nighttime splint in patients with plantar fasciopathy.

Interventional

In cases of plantar fasciopathy nonresponsive to conservative measures, extracorporeal shockwave therapy (ESWT) has been used prior to considering surgery [50, 51•]. ESWT is thought to stimulate soft tissue healing in plantar fasciopathy, but the underlying process by which this occurs is not clearly understood. ESWT can be divided into low intensity and high intensity types. A recent meta-analysis reported low intensity ESWT to be more efficacious than high intensity ESWT [50]. Interestingly, another recent systematic review drew the opposite conclusion [51•]. This discrepancy can be explained by the differences in how studies were categorized by the two authors. Speed suggested that a better way to categorize this type of treatment was into focussed ESWT (low dose and high dose) and radial pulse therapy [51•]. Using this categorization, Speed found strong evidence for the use of high dose ESWT and radial pulse therapy [51•].

Corticosteroid injections have been a longstanding treatment option for plantar fasciopathy, and their effectiveness was first described by Furey [52]. There is a clear positive effect with corticosteroid injections in plantar fasciopathy, but the duration of this effect is unclear, with reported durations varying from 4 weeks to 2 years [53, 54]. The main concern with the use of corticosteroids is the risk of plantar fascia rupture [55–57]. In addition to this, a theoretical risk of heel pad atrophy has been suggested, although to date there have not been studies supporting this theory [58, 59•]. Plantar fascia injections traditionally have been performed with landmark guidance. More recently, three different ultrasound-guided techniques have been proposed for plantar fascia injections [59•]. The intrafascial technique has a theoretical risk of plantar fascia rupture [59•]. Superficial plantar fascia injections, where the injectate is delivered between the planar fascia and plantar fat pad, have been noted to have a theoretical risk of heel pad atrophy [59•]. A deep plantar fascia injection, described by McNally and Shetty, theoretically avoids both complications by delivering the injectate deep to the plantar fascia [28]. However, a recent cadaveric study found this technique resulted in limited contact of the injectate with the deep plantar fascia due to the intervening flexor digitorum brevis [59•]. Furthermore, this is close to the location of the first branch of the lateral plantar nerve, which may lead to a higher risk of nerve injury with this approach. Although a recent systematic review and meta-analysis found ultrasound-guided steroid injections to be superior to palpation-guided ones, future studies examining the effectiveness of each of the three ultrasound-guided techniques are warranted [59•, 60].

Botulinum toxin A, first described for use in plantar fasciopathy by Placzek et al., does not have the theoretical risks

associated with corticosteroid injections [61]. The mechanism upon which botulinum toxin A treats plantar fasciopathy has not been studied, but it has been theorized to block the local pain pathway [62]. Palpation-guided and ultrasound-guided (intrafascial) botulinum toxin injections have been found to be more effective than placebo injections at 8 and 12 weeks post-injection [63, 64]. Botulinum toxin has also been found to be superior to a corticosteroid injection at the 1- and 6-month follow-up periods, and other studies found a sustained effect at 1-year follow-up [65–67].

Platelet-rich plasma (PRP) is an emerging option for musculoskeletal disorders. In plantar fasciopathy, PRP is thought to promote healing with the release of several growth factors, but this has not been studied scientifically. Several clinical studies have demonstrated reduced pain following PRP injections for plantar fascia [68–75]. An equal effect was found in comparison to ESWT [76]. A recent systematic review noted that PRP is a promising option in the treatment of plantar fasciopathy, but suggested future studies with a better control group and longer follow-up periods are needed to better assess the role of PRP in the treatment of plantar fasciopathy [77].

Other interventional treatment options with limited evidence include hyaluronic acid injections, micronized dehydrated amniotic/chorionic membrane allograft injections, and radiofrequency ablation of the calcaneal branch of the inferior calcaneal nerve [78, 79]. These options need further investigation to assess their role in the treatment of plantar fasciopathy.

Surgical

Surgery has been a longstanding treatment option in plantar fasciopathy. Initially, patients underwent an open plantar fascia resection with a heel spur resection, but subsequent studies supported the superiority of plantar fascia resection endoscopically without heel spur resection [80]. Although the latter was thought to reduce the risk of infection, wound breakdown, and neurovascular damage, biomechanical studies have suggested that complete release of the plantar fascia may produce dorsolateral midfoot stress that may subsequently lead to foot pain [81, 82]. A partial plantar fasciotomy is another option and can be performed endoscopically or under ultrasound guidance [83, 84]. Interestingly, a recent study compared partial plantar fasciotomy to proximal medial gastrocnemius release, and found that the latter treatment had better outcomes [85].

Conclusion

Plantar fasciopathy is a common condition. Findings suggestive of plantar fasciopathy on history and physical examination include plantar heel pain that is worst during the first few steps in the morning or after prolonged sitting and tenderness to palpation over the medial calcaneal tubercle. Diagnostic imaging is typically not indicated prior to initiating a conservative treatment program in patients with a history and physical examination consistent with plantar fasciopathy. However, if the patient's history and physical examination suggest the possibility of a different diagnosis or if the patient has failed standard conservative treatment measures, then further diagnostic imaging is recommended. A thorough understanding of the scientific evidence supporting various conservative, interventional, and surgical treatment options is critical when formulating an evidence-based approach to the treatment of this condition.

Compliance with Ethics Guidelines

Conflict of Interest S. Rajasekaran and J. T. Finnoff both declare no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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