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REVIEW

Plantar Fasciitis in Diabetic Foot Patients: Risk Factors, Pathophysiology, Diagnosis, and Management

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Abstract: Plantar fasciitis (PF) is a common degenerative disorder and a frequent cause of heel pain, mostly affecting patients in their fourth and fifth decades. Diabetic patients are particularly at risk due to the presence of common risks and co-morbidities such as obesity or a sedentary lifestyle. The diagnosis of PF is mainly clinical. Imaging is not recommended for the initial approach. The initial management is conservative and should include physiotherapy, off-loading, stretching exercises, and nonsteroidal anti-inflammatory drugs. Glucocorticoid injections or surgery is an option at a later stage in recalcitrant cases. The overall management of PF does not differ between patients with diabetic foot problems and non-diabetic patients, although the details can differ. This narrative review summarizes the state of the art in terms of the risk factors, pathophysiology, diagnosis, assessment, and management of PF in diabetic patients.

Keywords: plantar fasciitis, diabetic foot, management, epidemiology, diagnosis

Introduction

DM is an epidemic disease that affects 425 million people worldwide and it is predicted to increase by 48% to 629 million patients by 2045.[1] It is associated with a multitude of chronic impairments related to micro- and macroangiopathic involvement and polyneuropathy. These comprise cardiovascular complications, stroke, retinopathy, nephropathy, adhesive capsulitis, Dupuytren's contracture, crystal-induced arthritis, and PF.[2,3] The occurrence of these disorders is associated with the duration and control of DM.[4] Neuropathic complications or previous foot ulcers are associated with an increased prevalence of PF in diabetic patients.[5] However, it remains unclear whether PF is directly imputable to DM itself, its hyperglycemic state, or whether PF only shares common comorbidities and risk factors such as obesity, alcohol consumption, and active smoking. In terms of pathogenicity, metabolic disturbances are known to impair the conformation and mechanical features of tendons,[6] especially in metatarsophalangeal joints, the plantar fascia, and the Achilles tendon, by forming complex biomechanical structures.[7] They also play a role in the pathological distribution of plantar pressure on the foot; a condition that is frequently observed in diabetic patients as they tend to have increased forefoot pressure and, therefore, subsequently develop the corresponding lesions.[8–11]

PF is among the most frequent causes of activity-enhanced foot pain in the adult population. The overall prevalence of this local, non-infectious inflammation is estimated to be 10%. The vast majority of patients are initially assessed by general

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practitioners. To cite an example, in the United States, approximately one million people consult a doctor each year resulting in a final diagnosis of PF.[12] The peak incidence occurs in individuals between 40 and 60 years of age.[13] The occurrence of PF is not associated with participation in sports or with gender. A recent and large cohort study that evaluated the prevalence of PF in 720,000 diabetic patients found that the incidence of PF was 0.85%.[14] More specifically, the incidence of PF was 0.80% in non-diabetic adults, 1.31% in type 2 diabetics, and 0.92% in type 1 diabetic adults. In this survey, the group with type 2 diabetes had a significantly higher incidence of PF than those with type 1 (or no diabetes), while the difference between the non-diabetics and the type 1 diabetics was not statistically significant. This was still the case after adjustment for the BMI and gender.[14] In this short narrative review, we address the state of the art regarding the risk factors, pathophysiology, diagnosis, assessment, and management of PF in adult diabetic patients.

Methods

All of the authors performed a scientific literature search using PubMed and Google (Scholar) for publications regarding PF in adult diabetic patients using the MeSH terms "plantar fasciitis", in combination with "diabetes" or "diabetic foot", in English, French, or German up to 31 July 2019 without restriction of the setting, the medical specialty, or the time. We manually searched the references of the retrieved papers for further reports. We excluded reports with underlying osteosynthesis devices or previous plantar surgeries for any reason.

Epidemiological Risk Factors and the Pathogenesis of Plantar Fasciitis

No exact (single) etiology for PF has been shown, and PF is most likely a multifactorial disease. It has been associated epidemiologically with age, obesity, a sedentary lifestyle, flat (planus) or high-arched (cavus) feet, gastrocnemius contracture, long-distance runners, and individuals who have to stand a lot for their work.[12,15-17] A systematic review of 51 studies found that a BMI > 27 kg/m^2 was the only significant factor associated with PF.[18] Of note, PF has only rarely been associated epidemiologically with systemic rheumatic diseases such as spondyloarthropathy and reactive arthritis of fibromyalgia. Infectious PF, without any other localization of infection in diabetic foot, is very rare.

Some research groups favor a degenerative origin. PF is characterized primarily by degeneration of the plantar fascia as a result of repetitive micro-tears that give rise to a local inflammatory response without systemic repercussions. Shortening of the gastrocnemius-soleus complex may play a crucial role in the development of PF. Other research groups have stressed the apparent involvement of metabolic factors in the pathogenesis of PF, along with mechanical overuse. This could particularly be the case in DM patients. In this view, PF in DM foot is linked to advanced glycation end-products (AGEs).[19,20] Indeed, AGEs induce collagen crosslinking, ultimately leading to altered collagen structures and secondary mechanical dysfunction. In vitro, glycation disrupts the organization of collagen, resulting in an irregular fibril density and morphology. Electron microscopy has revealed a reduced density of tenocytes and fibroblasts along with an increased density of collagen. Of note, AGEs are increased in DM. Their presence has been directly associated with increased plantar fascial or Achilles tendon thickness in diabetic subjects; in contrast to non-diabetic subjects.[21,22] Hyperglycemia itself can modify redox homeostasis, particularly the polyol pathway, thereby leading to cellular edema.[6] DM is also associated with decreased tendon neovascularization.[23] The density of capillaries per unit of surface area is reduced, thereby leading to reduced blood flow.[24] This alteration could reduce vessel and nerve growth. The addition of a sensitive neuropathy and reduced nerve ingrowth decrease the distress signals and ultimately re-promote tendon overuse and damage.

Distal polyneuropathy (DPN) is common in diabetic patients, with a prevalence of at least 50%. DPN is defined as a loss of sensitivity beginning distally in the lower extremities that may also be characterized by pain and significant morbidity. (ref) Half of the patients are asymptomatic and therefore remain at high risk for insensate lesions to their feet. (ref) Assessment of diabetic patients at least once a year for the presence and severity of DPN is recommended. This can be performed using a graduated tuning fork (128 Hz), 10-g monofilament testing, or by the ankle jerk reflex. In the presence of DPN, other causes of neuropathy must be excluded, including vitamin deficiency, renal failure, or thyroid disorders. The precise contribution of DPN in the development of PF has yet to be elucidated as the available data have remained limited. Studies assessing the association have indicated that PF is independent of the presence of moderate to severe DPN.[3,8] Clinicians should, therefore, consider PF even in the absence of clinical DPN.

Other effects of DM, such as hypercholesterolemia, obesity, and hyperuricemia, may also play a role in PF.[6] Obesity in particular contributes by means of two distinct mechanisms. First, the increased weight on the tendons promotes inflammation and structural alterations. Secondly, many adipose tissue hormones, e.g. adipokines, lipocalin-1, serum amyloid A-3, and adiponectin, can disrupt the cellular functions of chondrocytes and tenocytes; thereby potentially leading to tendon disruption.[25,26] The potential role of chronic hypercholesterolemia in tendinopathies remains a matter of debate. Some studies have been able to correlate sonographic or histological tendon alterations with serum lipid levels.[25,26] Small cholesterol particles may accumulate in tendons, thereby eliciting low-grade inflammation and tendon damage.[27] Finally, both asymptomatic hyperuricemia and gout are associated with DM and PF as they share common risk factors.[28] Urate deposition and tophus formation can occur at various sites in the body, including the plantar fascia. Repetitive micro-trauma on these tophi may alter their interaction with the matrix and promote inflammation and consecutive tendon injury.[6,29] Figure 1 provides an overview of the metabolic pathogenesis of PF, especially for diabetic foot.

Pathological Pressure Redistribution in Diabetes and Plantar Fasciitis

Anatomical alteration and pressure redistribution are important factors in the development of plantar fascia and are favored by the presence of neuropathic diabetic foot.

Anatomically, the plantar fascia is composed of three bands of compact connective tissue that arise from the medial calcaneal tubercle and that insert distally into the base of each proximal phalanx. When the plantar fascia is stretched,[30] the strain consolidates the medical longitudinal arch, which then enables the foot to operate as a stiff lever for forward propulsion in conjunction with inversion in the Chopart joint line.[31] This increase in plantar pressure distribution in DM patients has been observed in several studies.[32,33] It appears to be related to altered proprioception that leads to instability between the long extensors and flexors of the toes, thereby causing further protrusion of the metatarsal heads and thus promoting the development of clawed toes.[32] Consequently, the plantar pressure increases under the hallux and the 5th metatarsal head, whereas the pressure under the heel decreases.[34] A vicious circle sets in as the anterior pressure promotes callus formation that, in turn, intensifies the plantar pressure. Not surprisingly, elevated plantar pressure is a major contributor to plantar ulcers in diabetic and PF patients.[5,35,36]

Diagnosis of Plantar Fasciitis (in Diabetic Foot)

PF is essentially a clinical diagnosis, relying on pain localized in the inferior heel with a distinct area of tenderness that is exacerbated by walking, while it wanes with resting. The pain usually dissipates to a certain degree over the course of the day, although it can flare up after periods of prolonged standing. Upon physical examination, passive dorsiflexion of the ankle and toes (windlass test) usually

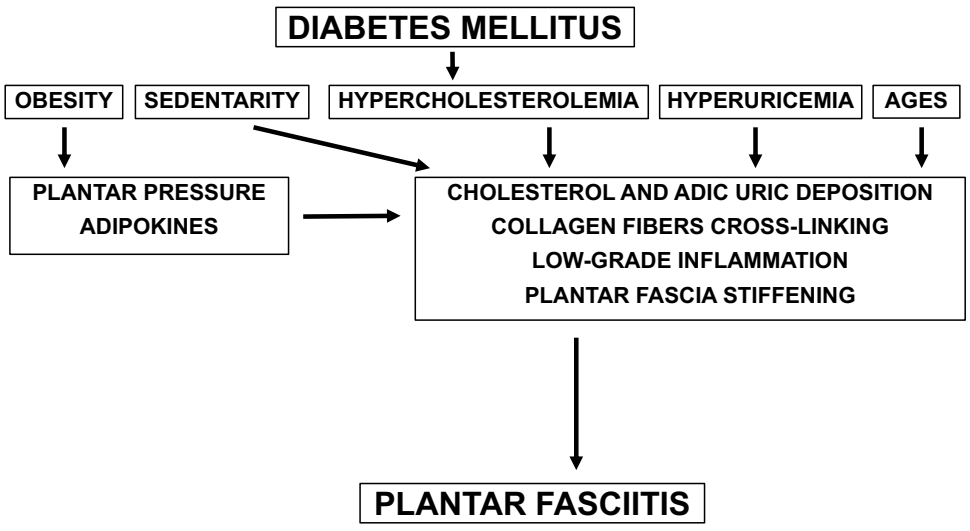

Figure 1 Proposed pathogenic mechanisms for the development of plantar fasciitis in diabetic patients.
Abbreviation: AGES, Advanced glycation end products.

induces pain. Furthermore, physicians should actively examine the gastrocnemius-soleus complex. A lack of ankle dorsiflexion beyond 10° reflects shortening of the gastrocnemius-soleus complex, which is associated with PF. The differential diagnosis is extensive, but it typically comprises neurological causes such as progressive polyneuropathic pain; or compression of the first branch of the lateral plantar nerve ("Baxter neuropathy"). Soft tissue involvement can indicate retro-calcaneal non-infectious bursitis, Achilles tendinopathy, or plantar fascia rupture. Heel pain originating from the bone typically includes a history of contusion, fracture, or Haglund's syndrome, which is an entity defined by the presence of insertional Achilles tendinopathy, retrocalcaneal bursitis and calcaneal prominence (Haglund deformity).As for all diabetic foot problems, an underlying osteomyelitis should be ruled out (at least clinically), especially when the skin is altered, chronically ulcerated, and/or inflamed.[37] By contrast, acute or chronic diabetic foot ischemia (without visual soft tissue necrosis) is unlikely to occur exclusively at the plantar fascia.

Blood tests are not decisive, as inflammatory markers would only be increased by concomitant systemic inflammatory disease, but not by a local (mechanically triggered) inflammation. Imaging is not required for the initial assessment of PF, although it can be helpful to rule out alternative etiologies of heel pain, especially in diabetic foot.[38] Sometimes, in refractory and atypical cases, a plain X-ray may be required. Plantar fascia thickness at its calcaneal origin is suggestive of PF, with a sensitivity of 85% and a specificity of 95%.[39] Plain X-rays can also reveal calcaneal spurs. Although international experts have questioned the diagnostic value of calcaneal spurs for the primary diagnosis of PF,[40] they might nonetheless be indicative of PF severity.[41] In addition, conventional X-rays may be helpful to detect a (stress) fracture. Sonographic findings of PF comprise fascial thickening, hypoechogenicity at the calcaneal insertions, and the loss of fibrillar structures. Ultrasound may also be valuable for distinguishing PF from diabetic foot infections or tumors. The MRI characteristics of PF include thickening of the plantar fascia and increased signal on delayed T2 sequences. Out of all of the available imaging modalities, MRI is considered to be the most sensitive technique for diagnosing PF.[42,43]

Management and Therapies

In 2010, the American College of Foot and Ankle Surgeons released an update regarding PF management. These guidelines favor a three-tiered approach[44] and they are recommended for all PF patients, including those with diabetic foot problems. According to these recommendations (Figure 2), first-line treatment of PF is usually managed by a general practitioner or a diabetologist, and consists of physiotherapy, stretching exercises, over-the-counter arch supports, and oral nonsteroidal anti-inflammatory drugs. The second line includes steroid injections, orthotic devices, immobilization, off-loading, and eventually night splints. The third tier should be considered after six to twelve months of persistent PF and it comprises extracorporeal shockwave therapy (ESWT), physiotherapy, or (as a last resort) surgical intervention such as elective plantar fasciotomy.

Exercises

These recommendations are clearly of overall benefit to DM patients, and those with diabetic foot problems in particular.[45,46] Exercise therapy has an important role in the overall management of diabetic foot disorders. It improves the functional capacity[47,48] and endothelial functions while it reduces oxidative stress and inflammatory responses.[49,50] Walking and stretching can often be performed in

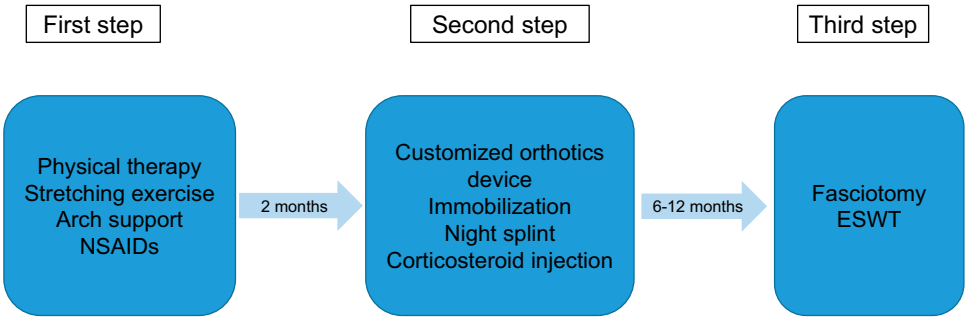

Figure 2 Proposed 3 steps approach for the management of plantar fasciitis.
Abbreviations: NSAIDs, nonsteroidal anti-inflammatory drugs; ESWT, extracorporeal shockwave therapy.

conjunction. Moreover, the foot joint range of motion (ROM) is often altered in diabetic patients due to plantar fascia stiffness, which can ultimately give rise to a rigid foot.[51] Home exercise program should be proposed to diabetic patients in order to improve the ROM to prevent the development of diabetic foot complications related to the alteration of plantar pressure distribution. Indeed, a study have shown that self-care exercise can prevent the reduction of ROM in diabetic patient with and without neuropathy. Pedobarographic assessment should also be performed before and after the occurrence of PF in diabetic patients[52]. Another aim of stretching exercises is to reduce plantar fascia (and Achilles tendon) tension by decreasing the anatomical arch deformation and foot pronation while increasing ankle dorsiflexion. A small RCT with 19 diabetic foot patients showed that only professional active and passive ROM exercises significantly reduced plantar pressure.[53] Likewise, regular exercise at home may also reduce the risk of foot ulceration and promote the functional independence of diabetic patients. Stretching techniques can also influence the outcome. Manual stretching appears to have only a limited beneficial effect on pain and foot function, as suggested by a recent systematic review.[54] The benefit of manual stretching can be seen in the first four months, in contrast to active exercises performed by the patient themselves with professional guidance.[54] The ideal frequency and number of prophylactic or therapeutic stretching for PF remain unknown and could be highly individual. The majority of PF cases can be managed conservatively, as only 10% require surgery.[55,56]

Medical Therapies and Related Outcomes

Systemic oral nonsteroidal anti-inflammatory drugs (NSAIDs) at standard doses should be considered in the absence of any contraindications, despite the limited available evidence. They can always be administered concomitant with exercise. While topical NSAIDs probably do not influence the inflammation, they could provide a degree of relief of local superficial pain. In the area of PF, we are aware of only a single small RCT of NSAIDs with 29 patients, which found non-significant improvement of pain with NSAIDs after 2 and 6 months of treatment, thus suggesting that NSAIDs could possibly result in a degree of pain relief and reduced disability in patients with PF.[55] The duration of treatment with NSAIDs should not exceed two to three weeks to avoid side effects, especially when there is no clinical benefit. The initial therapy also comprises rest and ice packs, as both of these can temporarily reduce pain. Of note, the application of heat appears to result in less of a benefit compared to ice.[57]

Local subcutaneous glucocorticoid injections should be considered if the disability persists after a month of conservative PF therapy. They usually contain a mixture of a long-acting glucocorticoid and a local anesthetic, and they lead to a rapid, albeit short-term, pain relief. A combination of glucocorticoid and lidocaine appeared to be superior to lidocaine only in an RCT involving 106 patients. However, the benefit of such a combination for refractory PF disappeared after 3 months compared to lidocaine only.[58] Repetitive steroid injections can lead to heel-pad atrophy and rupture of the plantar fascia.[59,60] Glucose monitoring is recommended after glucocorticoid injection, and a temporary treatment adjustment may be required. A significant degree of clinical improvement is frequently observed during the first six weeks of conservative therapy. When there is a clinical response, the therapy should be pursued until the symptoms disappear. In the absence of success with this treatment, the patient should be referred to an orthopedic surgeon specialized in foot and ankle surgery, or to a rheumatologist with experience with PF.

Casts, Devices, and Immobilization

There are various types of foot orthoses, such as custom-made, prefabricated, or medial arch supports. Prefabricated orthoses are usually part of the first-line therapy, whereas custom-made or medical arch supports are considered to be a second-line therapy. Over-the-counter arch supports can generally be purchased at pharmacies. However, their clinical benefits remain controversial. Compared to custom-made orthoses, over-the-counter arches have been associated with early withdrawal due to lack of compliance, persistent pain, or an inability to tolerate the device (50). A recent meta-analysis included twenty studies with a total of eight types of foot orthoses for PF.[61] The authors found that there was no difference in pain improvement or function between prefabricated, custom-made, or sham orthoses.[61] Night splints are part of the second-line therapy. Their positive impact in PF remains to be proven, however. Another recent RCT (40 patients) showed no improvement with night splints compared to a home exercise program without night splints.[62] Finally, cast immobilization for 4–6 weeks, or the use of a short-leg walking boot, have been suggested after failure of the first-line therapy. The level of evidence remains low, however, mainly due to the lack of RCTs.[63] Symptomatic improvement with the second-tier approach should be

observed within 2 to 3 months, and it usually happens in up to 90% of patients.[57,64]

When Everything Fails

The third (and last) therapeutic line is reserved for clinical failures after six to twelve months of first- and second-line therapies. Its options are ESWT and surgical plantar fasciotomy. Patients considered for surgery account for only 1 to 2% of all PF cases.[65,66] They should ideally be evaluated by an orthopedic surgeon with expertise in plantar foot surgery. Various surgical techniques have been reported in the literature: endoscopic plantar fasciotomy, open plantar fascia release, open or percutaneous plantar fasciotomy, median calcaneal nerve neurolysis, and calcaneal osteotomy with resection of parts of the fascia.[67,68] These options are the same when DM patients are involved. There has been a lack of RCTs to date comparing the efficacy of these various surgical procedures. The reported success rates vary between the studies, and they typically range between 50 and 90%.[67,68] Currently, most experts tend to opt for an endoscopic minimally invasive surgical approach as opposed to a large open surgery, as a minimally invasive approach generally leads to a faster recovery while providing a similar degree of clinical pain relief after one year.[69] The use of therapeutic ultrasound during surgery does not appear to offer any additional benefit. Rather, it in fact correlates with an increased risk of complications,[44,70-72] and the exact reasons for this remain unknown. The potential complications of PF surgery comprise iatrogenic calcaneal fracture, infection, posterior tibial nerve injury, and flattening of the longitudinal arch. Diabetic patients tend to have more complications, namely surgical site infections, and especially in their feet.[73]

ESWT is the second option in third-line therapy for PF. It is non-invasive and it is based on shock waves produced by electromagnetic, electrohydraulic, or piezoelectric techniques. The wave transmission through pain receptors is thought to elicit a healing process by means of the release of growth factors that ultimately favor neovascularization.[74,75] The level of evidence is low, and no recent studies have reported a significant clinical benefit of this method in PF treatment.[76–78] An elevated BMI is an established risk factor for PF.[79] Weight loss may, therefore, represent an interesting approach to resolve PF. Indeed, researchers have shown that a decrease in the BMI after bariatric surgery can lead to a reduction in the frequency of clinical PF episodes.[80] Other techniques for refractory PF are botulinum toxin injection, topical steroids, radiotherapy, cryosurgery, and local platelet-rich plasma injection. None of these have been shown to clearly exhibit efficacy.[81–84]

Rarity: Infectious Plantar Fasciitis in Diabetic Foot

Classical PF is a symptomatic, non-infectious, and local inflammation. Theoretically, diabetic foot plantar fascia can also be infected, although it is encountered very seldom in everyday clinical activities. We only found three papers in our literature search that explicitly described this clinical entity.[85–87] Usually, this form of diabetic foot infection is moderate to severe, because infectious fasciitis tends to spread within a few days, if not hours. Likewise, the infection would not be limited to the plantar fascia, but also exhibit abscesses, plantar phlegmon, or cellulitis. The origin of diabetic foot plantar infections could be local trauma or injections for conventional PF. By contrast, the classical combination of ischemia and/or chronic neuropathic pressure ulcers, possibly with prior hyperkeratosis, as we have frequently encountered in the forefoot or the calcaneus, is not likely to be the origin. In infectious diabetic foot PF, a combined surgical and medical approach is mandatory from the start.

Conclusion

Adult diabetic patients are more at risk of PF than non-diabetic patients. The current evidence for management of PF is based mainly on retrospective and heterogonous studies and it does not exclusively address diabetic patients. The modalities for management of PF in terms of diagnosis and therapy are the same for diabetic and non-diabetic foot problems, whereas the differential diagnosis of PF in diabetic patients is more extensive. It is important to follow the current three-tiered approach of the American Guidelines,[44] although each management should be individually tailored to the patient. None of the various diagnostic and therapeutic options is superior to another, and this also holds true for the diabetic population. Clinicians in charge of diabetic foot patients should be familiar with PF.

Abbreviations

AGEs, Advanced glycation end-products; BMI, Body mass index; ESWT, Extracorporeal shockwave therapy; MRI, Magnetic resonance imaging; NSAIDs, Nonsteroidal anti-inflammatory drugs; PF, Plantar fasciitis;

ROM, Range of motion exercise; RCT, Randomized controlled trial; DM, Diabetes mellitus.

Author Contributions

All authors contributed to data analysis, drafting or revising the article, gave final approval of the version to be published, and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that there are no conflicts of interest regarding the publication of this article.

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