Review Article

Chronic Plantar Fasciitis is Mediated by Local Hemodynamics: Implications for Emerging Therapies

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Abstract

Plantar fasciitis (PF) is a common, disabling condition affecting millions of patients each year. With early diagnosis and timely application of traditional nonsurgical treatments, symptoms generally resolve over time. However, despite adequate treatment, 20% of patients will experience persistent symptoms. In these patients, minimally invasive therapies that augment local hemodynamics to initiate a regenerative tissue-healing cascade have the greatest potential to resolve long-standing symptoms. We performed a narrative review based on a best evidence evaluation of manuscripts published in Medline-indexed journals to determine the mechanisms involved in soft tissue injury and healing. This evaluation also highlights emerging minimally invasive therapies that exploit these mechanisms in recalcitrant PF.

Keywords: Heel pain, Hemodynamics, Minimally invasive, Plantar fasciitis

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Introduction

Plantar heel pain is the most common reason for visits to foot and ankle specialists, accounting for 1-2 million annual visits in the US alone.[1,2] Plantar fasciitis (PF) is the predominant diagnosis,[3] a condition characterized by degeneration of the plantar fascia and perifascial structures with isolated inferior heel pain, particularly with the first steps of the day and after prolonged sitting.[4] Approximately 1 in 10 people will be diagnosed with PF during their lifetime,[5] with women aged 40-60 years most commonly affected.[6] Risk factors include limited ankle dorsiflexion, flatfoot deformity, obesity, and prolonged work-or activity-related weight bearing.[6-8] PF negatively impacts health-related quality of life[9] and is responsible for a significant societal economic burden, with almost 300 million dollars annually spent on physician visits and treatments.[10]

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With early diagnosis and timely application of traditional nonsurgical treatments such as activity modification, gastrocnemius and plantar fascia-specific stretching, anti-inflammatory medications, and/or shoe inserts, the prognosis is favorable with approximately 80% of patients achieving symptom resolution within 1 year.[11-13] Symptoms may persist in some patients despite increasingly aggressive therapies such as corticosteroid injections, night splints, or cast/boot immobilization. Since the etiology of these recalcitrant cases is controversial and likely multifactorial, numerous therapies with various mechanisms of action have been attempted, although none have an ideal efficacy and safety profile. Conventional treatments for chronic PF may be misdirected, while therapies which augment local hemodynamics, thereby initiating a regenerative tissue healing cascade, have the greatest potential to resolve long-standing symptoms.

We performed a narrative review based on a best evidence evaluation of manuscripts published in Medline-indexed journals to determine the mechanisms

involved in soft tissue injury and healing. This evaluation also highlights emerging minimally invasive therapies that exploit these mechanisms in recalcitrant PF.

Mechanism of plantar fascia injury

The plantar fascia connects the medial calcaneal tuberosity to the proximal aspect of the phalanges, plays a major role in supporting the medial longitudinal arch, and aids in dynamic shock absorption.[14] The term plantar fascia is actually a misnomer since this structure is not a facial layer, but a tendinous aponeurosis that shares histological and mechanical traits with tendons and ligaments.[15] Therefore, it is relevant to compare the etiology, pathophysiology, and treatment of PF to the analogous processes which occur in degenerative diseases of other tendons (tendinosis).

It is generally believed that PF is initiated by excessive tensile strain within the fascia during repetitive loading producing microscopic tears and an acute inflammatory response. Macrophages, lymphocytes, and plasma cells infiltrate the calcaneal enthesis causing tissue destruction. If the insult persists, and the reparative process is unable to keep up with the ongoing mechanical demands, then the attempted healing instead produces immature vascularization and fibrosis.[16] Tissue degeneration (fasciosis), rather than inflammation, then becomes the cardinal pathologic feature. A number of studies have analyzed tissue samples from patients with chronic PF and have found no evidence of inflammation.[16-19] Similarly, histological analyses of surgical biopsies of tendons affected by "tendonitis" have revealed no evidence of inflammatory cell invasion.[20-24]

Plantar fascia thickening[25] and loss of normal tissue elasticity[26] play an important role in the genesis of persistent inferior heel pain. There is a fivefold increase in the mechanical stiffness of the plantar fascia in pathologic feet compared with healthy ones. This increased stiffness results in higher tissue hydrostatic pressure within the plantar tissues during loading, consequently acting on the external surface of blood vessels to reduce the flow cross-section area local blood supply.[27] This cascade of events has been hypothesized to cause tissue necrosis and replacement with undifferentiated scar tissue and is the presumed reason for lack of response to traditional nonsurgical therapies.[28]

Once damaged, the biological and biomechanical properties of connective tissue are never completely restored. Healing times in chronic tendinopathies are often prolonged since local blood flow is only about one-third of that delivered to the muscles.[29] Moreover, augmenting the local blood flow has been shown to hasten regeneration of damaged connective tissue.[30] The proximal plantar fascia is relatively hypovascular, as it is perfused by only a few vessels with none at its insertion onto the calcaneus — the point of maximal tenderness in chronic PF. These findings form the rationale for our hypothesis that exogenous stimulation of local circulation via increasing blood flow, angiogenesis, or by direct application of blood-derived products may hasten healing with chronic PF.

Treatment decision-making

Treatment decision-making for chronic PF is challenging given that there are dozens of available treatment options with no clear gold standard. Unfortunately, the effectiveness of continued conservative treatment for inferior heel pain diminishes if symptoms have not resolved with 3-6 months of standard treatment. In these cases, patients must consider more invasive options to achieve satisfactory symptom relief. Treatment decision-making is further complicated by the lack of evidence for emerging therapies.

Given the compelling evidence that chronic subcalcaneal heel pain stems from a degenerative process, it is imperative that the rationale for use of currently available therapies be reevaluated. For example, corticosteroid injections are extensively utilized in patients with chronic inferior heel pain. However, given the absence of a measureable inflammatory response, the utility of corticosteroid injections appears to be misdirected and may partially explain their limited effectiveness in recalcitrant cases.

The reluctance to adopt noninvasive or minimally invasive therapies for chronic PF was evident from a survey of 84 orthopedic surgeons who were asked to provide treatment recommendations for a hypothetical patient presenting with PF resistant to 10 months of nonoperative management.[31] Over 50% of surgeons chose surgery (gastrocnemius recession or open plantar fasciotomy) as their preferred next step in management despite only moderate patient success rates, extended recovery times, and potential complications such as nerve injury, plantar fascia rupture, medial longitudinal arch destabilization, and altered loading patterns.[32-38] Consideration of emerging treatments focused on augmenting local hemodynamics, which is hypothesized to play a major role in lingering PF cases, is warranted to fill the therapeutic gap between ineffective conservative care and invasive surgical options in the patient with chronic PF refractory to conventional therapy.

Minimally invasive therapies for chronic PF

Given the growing evidence for the role of local hemodynamics in chronic PF, therapies intended to stimulate angiogenesis and/or improve local circulation

have recently been explored with promising results. Tendinosis forms as an imbalance between the demands that are placed on a tendon and its ability to remodel. Thus, the key to successful treatment may hinge on augmenting local circulation, which may revert a chronic lesion to an acute lesion by stimulating the inflammatory phase of the healing cascade.

Extracorporeal shockwave therapy

Extracorporeal shockwave therapy is a minimally invasive treatment for recalcitrant PF. The mechanism of action remains unknown, although the current opinion is that focused application of shock waves to injured soft tissue disrupts scar tissue and induces revascularization, releases local growth factors, and recruits stem cells; all of which enhance the intrinsic healing process.[39] Success rates have ranged from 34 to 88% in well-selected patients.[40] A meta-analysis of randomized placebo-controlled trials demonstrated that the odds of achieving clinical success was more than twice as high with high-energy extracorporeal shockwave therapy vs sham therapy through 12 weeks.[41] Through 6-12 months follow-up, extracorporeal shockwave therapy yields similar improvements in foot pain and function compared to autologous condition plasma[42] and endoscopic plantar fasciotomy,[43] and superior improvements versus conservative care in patients with chronic PF.[42] However, over the long-term, extracorporeal shockwave therapy has been shown to be no more effective than plantar fascia-specific stretching[44] and some studies have reported marginal improvement above that of sham treatment.[45] Consequently, extracorporeal shockwave therapy remains a controversial therapy for recalcitrant PF. Diverse treatment protocols are thought to confound patient outcomes including different therapeutic doses, applicator positions, and use of local anesthesia.[46-48]

Radiofrequency microtenotomy

The radiofrequency microtenotomy technique has been applied to chronic tendinopathies for over a decade with promising results. Radiofrequency microtenotomy stimulates angiogenesis in the avascular, fibrotic fascia, which promotes secretion of fibroblastic growth factor, vascular endothelial growth factor, and vascular cells. Histologic evaluation of treated tendons shows an early inflammatory response, with extensive proliferation of vascular cells and new blood vessel formation by 28 days.[49] Patients with chronic isolated Achilles tendinopathy refractory to nonoperative treatment rated their clinical outcome with radiofrequency microtenotomy as good or excellent with an average of 2.5 years follow-up.[50] Several case series have reported promising outcomes in recalcitrant PF,[51-56] with the open method more efficacious than the percutaneous technique.[56] No comparative studies have been performed to date with radiofrequency microtenotomy and follow-up data is limited to 1 year or less in all studies.

Platelet-rich plasma (PRP) injections

PRP is an autologous biological blood-derived product that contains high concentrations of platelet-derived growth factors. When used for the treatment of PF, 20-30 ml of the patient's blood is drawn and spun down, which yields 3-5 ml of platelet-rich substrate. Soft tissue healing is thought to be stimulated via enhanced fibroblast migration and proliferation, up regulated vascularization, and increased collagen deposition.[57,58] In one case series of 50 patients with refractory PF injected with PRP, mean pain severity improvement was 45% at 6 months with almost two in three patients reporting satisfaction with the treatment.[59] Other case series with no more than 1-year follow-up have reported similar outcomes with patient satisfaction rates of 79-96%.[60,61] To date, no controlled studies of PRP in chronic PF have been performed.

Micromobile compression (MMC)

MMC is a noninvasive technology that augments circulation through the deep veins of the leg via cyclic pressure pulses to the plantar venous plexus. The platform for the MMC is an orthotic that provides cyclic compression to the arch of the foot when the user is non-weight bearing. Dohm *et al*.,[62] reported that MMC augmented blood flow velocity 12-fold above resting levels in the posterior tibial vein and fourfold above resting levels in the popliteal vein of healthy adults. Charles and colleagues[63] compared MMC and below-knee graduated compression stockings on peak venous velocity at the popliteal vein. MMC yielded a four fold increase in peak venous velocity vs no change for compression stockings. It was subsequently hypothesized that since compression of the deep venous plexus increases perfusion through the plantar fascial microvasculature, this technology may have potential in the treatment of patients with PF.

MMC for the treatment of PF has been attempted so far in one patient. A 39-year-old otherwise healthy woman (body mass index of 23 kg/m^2 had a 2-year history of inferior heel pain. The patient complained of moderate/severe inferior heel pain and stiffness on the first steps of the morning, after prolonged sitting, and with ambulation > 30 min. She rated her pain as 8/10 with ambulation. She had tried activity modification, anti-inflammatory medications, physical therapy, orthotics, soft tissue manipulation, night splint, and multiple corticosteroid injections all without relief. Physical examination revealed a tight heel cord with dorsiflexion limited to neutral with the knee extended

and tenderness to palpation at the insertion of the PF. Plain X-rays demonstrated a small plantar heel spur but no arthritis. Magnetic resonance imaging (MRI) was used to exclude other potential causative factors such as calcaneal stress fracture and tumor. Ultrasound examination revealed thickening of the plantar fascia (7 mm) and mild retrocalcaneal bursitis. MMC was applied for 2-3 h/day, 5-6 days/week, for 2 months. No concomitant therapies were administered during this period. Pain severity during ambulation decreased to 6 over the first week, 4 at 1 month, and 4 at 2 months. During the 2-month treatment period, the patient was able to gradually increase her physical activity from walking 2-4 miles, two to three times per week to walking/jogging 3-5 miles, four to five times per week. She did not experience discomfort or side effects from the use of MMC. A randomized controlled study of conservative care, with or without MMC, is planned to begin enrollment in early 2015 to further evaluate the potential of MMC for PF.

Conclusion

PF is a misunderstood condition with little consensus regarding optimal treatment practices. Chronic PF is not associated with inflammation, but instead with tissue degeneration. Classical treatments including stretching, shoe inserts, and anti-inflammatory medications are effective in 80% of cases. Refractory cases have traditionally been treated with surgery although iatrogenic risks prevent widespread utilization. The emerging minimally invasive therapies have the potential to speed recovery in patients with chronic PF and may serve as an alternative to surgery for patients refractory to traditional nonsurgical treatments. Research efforts should thus concentrate on these emerging minimally invasive therapies that promote tissue regeneration by augmenting local hemodynamics.

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