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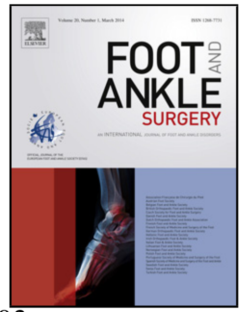

Title: Plantar fasciopathy: revisiting the risk factors

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Title page

Plantar fasciopathy: revisiting the risk factors

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ABSTRACT

Background: Plantar fasciopathy is the most common cause of acquired sub-calcaneal heel pain in adults. To-date, research of this condition has mainly focused on management rather than causal mechanisms. The aetiology of plantar fasciopathy is likely to be multifactorial, as both intrinsic and extrinsic risk factors have been reported. The purpose of this review is to critically reevaluate risk factors for plantar fasciopathy.

Methods: A detailed literature review was undertaken using English language medical databases.

Results: No clear consensus exists as to the relative strength of the risk factors reported.

Conclusions: To-date numerous studies have examined various intrinsic and extrinsic risk factors implicated in the aetiology of plantar fasciopathy. How these factors interact may provide useful data to establish an individuals' risk profile for plantar fasciopathy and their potential for response to treatment. Further research is indicated to rank the relative significance of these risk factors.

KEY WORDS

Fasciopathy; Fasciitis; Plantar; Calcaneal; Risk factors; Genetic

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INTRODUCTION

Similar to other conditions where pathological origin is unclear, chronic plantar heel pain has become a generalized term that includes several pathological conditions that affect the heel.[1] Heel pain may be the result of arthritic, neurological, traumatic, or other systemic conditions, although the overwhelming cause is mechanical in origin.[2, 3]

Plantar fasciitis is a commonly reported cause of plantar heel pain.[4-7] Terminology for this condition is confusing, as a degenerative process of micro tears (fasciosis) similar to tendinosis, a degeneration of collagen in tendons[8], and fascial thickening predominates over inflammatory changes. Similar histopathological changes have been reported in tendon and ligament disorders elsewhere[8] hence redefinition of the condition from plantar fasciitis to plantar fasciopathy (PF) may better reflect the underlying pathology within the fascia, which rarely includes inflammatory cells.

The purpose of this paper is to critically reevaluate risk factors for PF.

BACKGROUND

Relevant clinical aspects of PF are included to help support the discussion of risk factors.

Diagnosis

There is no widely accepted test or ‘gold standard’ for diagnosing PF.[9] Ultrasound can be used to confirm clinical diagnosis and classify the disease pattern. Ultrasound diagnosis of PF includes reduced echogenicity[9] and plantar fascia thickening (>4-4.5mm) at its calcaneal insertion[10-12]. Jeong et al[13] also found discrete fibromata and thickening in those with pure distal (non-insertional disease). More importantly there is disorganization of the normal reflective structure and loss of normal organized ligament architecture.[9]

Classification

Jeong et al[13] examined 125 consecutive feet with symptoms of recalcitrant PF. All had failed to respond to a stepwise conservative management protocol. Disease characteristics were evaluated using diagnostic ultrasound. A high proportion of atypical non-insertional PF was reported.[13] This would not be detected without imaging studies. The use of ultrasound in cases of recalcitrant plantar heel pain that have failed proper first-line management is recommended.[12, 13] It was concluded that ultrasound confirmed clinical diagnosis and classification characteristics as either insertional (proximal), non-insertional (distal) or mixed disease PF.[13]

Clinical picture

Plantar fasciopathy is a clinical diagnosis, characterized by the insidious onset of plantar heel pain after prolonged periods of rest.[2, 6] It is usually worse in the morning after the first few steps or starting to walk after a period of inactivity. Although walking helps initially, the pain can recur with further exertion. Some patients complain of pain on toe extension due to invocation of the windlass mechanism[5]. Pain may worsen towards the end of the day and with increased weight-bearing activity.[14] Although patients exhibit similar patterns of symptoms, the clinical presentation can vary in location, level of pain and duration.[15] Up to one third of patients with PF will present with bilateral symptoms.[16, 17] The condition affects both sedentary[18-20] and athletic individuals[21, 22], including military personnel[23, 24]; as such a diverse patient population is observed. The condition has been reported to peak between 40 to 60 years-of-age.[6]

Prevalence

The prevalence of heel pain in the general population is estimated to range from 3.6% to 7%[20-22], and the disorder has been reported to account for about 8% of all running related injuries.[25, 26] A retrospective review of 1407 patients from an outpatient sports medicine clinic, found that younger athletes had a lower prevalence of PF (2.5%) than older athletes (6.6%).[27] The literature is inconsistent regarding the association between gender and PF. Some studies show an increased prevalence in men[24]; while others show greater prevalence in women.[18, 22] Plantar fasciopathy is commonly described as a self-limiting condition.[5,6] Crawford & Thomson[28] undertook a systematic review supporting this observation. However, PF can be a painful and disabling condition with detrimental effects on health-related quality of life and subsequently be frustrating for patients. There is a higher risk of prolonged symptoms in overweight patients[29], those with bilateral involvement and when there is a long delay before seeking medical attention.[30, 31]

Impact on health

Patients are unlikely to be satisfied with evidence of the self-limiting nature of the condition and most are likely to demand treatment for their symptoms.[32] Irving et al[33] demonstrated that chronic heel pain has a significant negative impact on foot-specific and general health-related quality of life. The degree of negative impact does not seem to be associated with age, sex, or BMI.[33] Physical inactivity is recognized as one of the greatest public health challenges in Western countries.[34] The morbidity of PF can result in immobility and reduced activity levels.[33] Furthermore, patients who develop PF are often overweight and therefore subsequent loss of weight becomes increasingly difficulty due to the pain of everyday weight bearing.[35] The duration of obesity in obese patients may be important to the development of heel pain in such patients. Inactivity and an increased body weight are major risk factors for many diseases such as obesity, cardiovascular disease, diabetes and osteoarthritis making it imperative that treatment for PF is instituted rather than waiting for spontaneous resolution.

Economic burden

Plantar fasciopathy is an important public health disorder due to its frequent occurrence.[5] Researchers have estimated that 10% of people in the USA may present with heel pain over the course of their lives, with 83% of these patients being active working adults.[36, 37] With people working and living longer the age range for this condition may be potentially extending. An estimated one million visits per year were made to physicians and hospital outpatients in the USA for treatment of PF, representing an important economic burden to health services.[37] Frequently, patients do not seek treatment until symptoms are considered chronic. At this point treatment regimens can become costly, as symptoms are recurring, recovery is lengthy and the response to treatment is unpredictable.[31] Furthermore the potential for longer-term health consequences related to immobility such as weight-gain, hypertension, coronary artery disease and non-insulin dependent diabetes in chronic PF exist.

METHODS

The following criteria were used to search the literature:

1. English language human studies.
2. Published after 1988.
3. Electronic databases: Cochrane library, BioMed Central, EMBASE, CINAHL, AMED, Ovid, Swetswise, PubMed, Highwire, SportDiscus, ISI web of knowledge, Science direct, Science citation index, The Lancet.com, BMJ clinical evidence, MEDLINE, Scirus.com, Index to thesis, Controlledtrials.com UK national research register for on-going/ recently completed trials.
4. MeSH terms used alone or in combination: plantar fasciitis, fasciopathy, sub-calcaneal, heel pain, aetiology, risk factors.

5. Search limited to: peer-reviewed journals, systematic reviews/ meta-analyses, cohort studies, case control studies and surveys. Case reports and letter to editors were excluded.
6. Research papers were chosen based upon evaluation of PF risk factors.
7. Series with $n \geq 10$.
8. Results for each risk factor were separable if > 1 discussed.

DISCUSSION

Although PF is the most common soft-tissue cause of heel pain[5, 19] its aetiology is not fully understood.[22, 38, 39, 40, 41] The condition is considered to be multifactorial[6, 17, 20] and numerous risk factors are implicated in its development **(Table 1)**. The evidence supporting these factors is limited and their relative importance is unclear. Several causes have been hypothesized, with the most common being overuse due to prolonged weight-bearing, obesity, unaccustomed walking or running, limited ankle joint dorsiflexion, posterior muscle group tightness and standing on hard surfaces.[2, 5, 19, 20, 32, 40, 41, 51]

The presence of co-existing calcaneal spurs has often been reported[9, 11, 14, 42] but confusion exists as to whether it is a causal or significant association. Some suggest that calcaneal spurs may be an adaptive response to vertical compression of the heel rather than longitudinal traction at the calcaneal enthesis[43]. A study examining prehistoric skeletal remains[44] concludes that plantar calcaneal spurs are a modern phenomenon resulting from long periods of standing, excess weight and associated with lower limb osteoarthritis. Wainwright et al[42] reported a strong correlation with calcaneal spurs over 1mm long and PF and Johal & Milner[45] found a higher prevalence of calcaneal spurs in patients with PF. Further research is warranted to assess whether the association is causal.[44]

Typically PF affects middle-aged or older people, often women more than men. The association of PF with increasing age is consistent with the histopathological findings of degenerative changes within the plantar fascia.[38] These degenerative findings support the hypothesis that PF is secondary to repetitive micro trauma caused by prolonged weight-bearing activities.[52] The constant overload inhibits the normal repair process, resulting in collagen degeneration, which causes both structural changes and perifascial oedema.[1] These changes in turn lead to a thicker heel pad, which has been shown to be associated with pain in individuals with PF.[52] Increasing heel pad thickness leads to a loss of heel pad elasticity; both of these factors are associated with increasing age and increasing BMI.[53, 54] The decrease in elasticity of the fascia seen with increasing age is associated with a decrease in shock absorbing capabilities[54], which may be a result of the degenerative fascia's inability to resist normal tensile loads.

The current literature is inconsistent regarding the association between gender and PF **(Table 1)**. No theories exist hypothesizing the reason for a difference in prevalence between the sexes. This may relate to hormonal differences or structural changes like those seen in tendinopathy or differences caused by genetic variations.

Increased body weight [53] and increased body mass index (BMI)[19, 29] have been shown to be significant risk factors for PF **(Table 1)**. A BMI of more than 30 kg/m^2 having an odds ratio of 5.6 (95% confidence interval, 1.9 to 16.6; $p < 0.01$) compared with a BMI of less than 25 kg/m^2 [19]. Rano et al[29] also concluded that a BMI of 25 (the target for cardiovascular risk) represents a reasonable goal for weight loss that may reduce heel pain. Frey and Zamora [55] demonstrated a 1.4-fold increased probability of PF being diagnosed in an overweight or obese patient.[55] Rome et al[52] suggested that BMI is not related to plantar fasciitis pain in the athletic population, but other factors such as a low oestrogen levels in female athletes which leads to reduced collagen elasticity.[52]

Previous research has suggested that limited ankle dorsiflexion[30], obesity[29] and prolonged weight bearing[19] may increase the risk of PF. Those studies, however, involved the use of univariate analytical approaches and, in some cases, did not include a control group. Riddle et al[19] hypothesized that reduced ankle dorsiflexion is the most important risk factor for development of PF and reported that individuals with $\leq 0^o$ of dorsiflexion have an odds ratio of 23.3 (95% CI 4.3-124.4).[19] Riddle et al[19] hypothesized that increased ankle equinus can result in more compensatory foot pronation and subsequently greater tensile loading on the plantar fascia. Limited ankle dorsiflexion appears to have a biologically plausible explanation for causality. Individuals who spend the majority of the workday weight-bearing and those who are obese also theoretically have increased tensile loads on the plantar fascia compared with those who spend less time weight-bearing and those who have a normal body weight.

It is unclear whether limited ankle dorsiflexion is a cause or a consequence of PF. It is possible that limited dorsiflexion may develop after the onset of the disorder. Theoretically, if PF had caused the loss of dorsiflexion, then the motion on the involved side would have been reduced and the motion on the uninvolved side would not have been reduced. Riddle et al[19] undertook a case-control study where only cases of unilateral PF were used. The uninvolved side was used as the control for ankle joint dorsiflexion. It was found that dorsiflexion on the uninvolved side was also reduced relative to that in the control group.[19] A “dose-response” relationship was found for the risk factor of limited dorsiflexion on the uninvolved side.[19] Thus it was hypothesized that ankle dorsiflexion may have been limited before the onset of the disorder.[19]

More recently Bolivar et al[51] found an association between posterior leg muscle tightness (hamstring as well as triceps surae) and PF in a controlled trial of 100 participants. Labovitz et al[56] and Harty et al[46] also found an association with hamstring tightness and PF. Harty et al[46] concluded that this was found to prolong forefoot loading and through the windlass mechanism might be a factor that increases repetitive injury to the plantar fascia.

The most common cause cited for plantar heel pain is biomechanical stress of the plantar fascia at its enthesis of the calcaneal tuberosity.

Mechanical overload, whether the result of biomechanical faults, obesity, or occupation, may contribute to the symptoms of heel pain.[2] Foot pronation alone, as measured by the Foot Posture Index[57] has also been shown to be significantly greater in patients with chronic plantar heel pain.[16] This is supported by Lee et al[47] who demonstrated a high correlation between arch height (r = 0.642), plantar fascia tension (r = -0.797) maximum rearfoot eversion (r = -0.518). It is hypothesized that a lack of cushioning in a rigid high arched foot may also result in PF but this has not been proven.[5]

Stress shielding (failure of a stress deprived deep structure to heal because of the superficial element bearing most of the load) has been implicated in enthesopathy.[14] It has been suggested that that proximal tendinopathy of the flexor digitorum brevis muscle (which is deep to the plantar fascial ligament) is implicated in the pathology of PF.[14]

Localized nerve entrapment of the medial calcaneal or muscular (first) branch of the lateral plantar (Baxter's) nerve may be a contributory factor to plantar heel pain.[2] The presence of sensory disturbances including radiation of pain is indicative of neurological pathology thereby differentiating it from PF.

Work-related prolonged weight bearing has been reported to be associated with PF.[19, 23, 35, 40]. Riddle et al[19] found a significant association (OR 3.6, 95% CI 1.3-10.0) of the reported cases of PF with time spent working on feet (>80% of work day).[19] There was however, no data presented on the extent and duration of exposure; nor the particular occupations and work histories of the cases and controls.

Inappropriate footwear[5, 19, 48] and rapid increases in activity levels[5, 19] have also been reported as risk factors associated with PF.

In athletes PF is primarily believed to be an overuse injury combined with training errors, training surfaces, biomechanical alignment and muscle dysfunction and inflexibility.[5, 22, 25, 26] Additionally, PF has been associated with individuals engaging in sports involving jumping.[1] Excessive foot pronation can lead to increased plantar fascial tension during the stance phase of running.[25, 26] Furthermore, heel strike during running causes compression of the heel pad up to twice body weight.[5] For athletes with inadequate muscle strength or flexibility and decreased shock-absorbing capabilities, the initiation of a new training program may exacerbate overloading of the plantar fascia.[24] Increases in tensile loading, seen with new increases in running intensity or frequency and changes in general footwear have been associated with overloads of the plantar fascia leading to micro tears.[30] In particular, firm footwear may exacerbate the developing PF in such patients.[23]

These risk factors combine to create a pathological overload of the plantar fascia at its origin, causing micro tears[49] in the fascia that subsequently lead to perifascial odema and increasing heel pad thickness.[20, 38, 52] As these micro tears increase in size, they may coalesce to form a large symptomatic mass causing an increase in heel pad thickness.[52] These changes in fascial thickening[50] (particularly proximal portion), and oedema of the adjacent fat pad and underlying soft tissues can typically be seen on ultrasound or MRI.[1]

To-date no research has considered a possible genetic basis to PF.

Candidate gene variants for tendinopathy (a degenerative process not dissimilar to PF) have been examined and various associations revealed.[58-64]. Some of the candidate gene variants based on tendon studies may also be relevant to ligaments such as the plantar fascia **(Table 1)**. As in tendinopathy[60] a range of candidate gene variants may also contribute to the development of PF. Individuals may possess certain genetic risk factors that predispose them to PF. These genetic factors may interact with other factors (intrinsic and extrinsic) to increase their overall risk profile for developing PF. Research to examine a possible genetic basis for PF may add to our understanding of the intrinsic risk profile for this condition. Furthermore, it may help to predict the patients at risk from developing chronic PF.

Inflammatory disease[65-67] or drug therapy may also be implicated in the development of PF[68] in a few cases that is unresponsive to common conservative interventions.

CONCLUSION

Plantar fasciopathy is a common cause of sub calcaneal heel pain. The condition represents an important economic burden to health services due its potential to become chronic in nature. Studies supporting both intrinsic and extrinsic risk factors suggest complex multifactorial soft tissue pathology. Research to examine a possible genetic basis for developing this condition may advance our knowledge of the intrinsic risk profile, provide a novel and alternative approach to understanding this challenging condition and help rank the significance of risk factors.

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CONFLICT OF INTEREST

I would like to state that there are no conflicts of interest.

Dr Paul Beeson

Table 1: Risk factors for plantar fasciitis

INTRINSIC	EXTRINSIC
Increased age: - Average age at presentation 10 yrs higher than controls who presented for other reasons.[29] - Increased prevalence in older athletes.[27] - Age related degenerative changes may result in fascia's inability to resist normal tensile loads.[40, 41] - Associated with increased heel fat pad thickness & loss of elasticity.[52, 54] - Decreased fascial elasticity associated with decreased shock absorbing capabilities in older patients.[54]	**Physical load on ligament:** - Excessive foot pronation.[5, 16, 19, 25, 29, 53, 57] - Rearfoot eversion + arch height collapse.[47, 49] - Repetitive microtrauma.[52]
Obesity: - Increased BMI[16, 97, 29, 48, 53, 55] associated with increasing heel fat pad thickness and loss of heel pad eleasticity.[54]	**Occupation:** - Prolonged weight-bearing.[3, 5, 19, 35, 40] - Change in walking or running surface.[5]

- Significant positive correlation between BMI and PF thickness causing chronic stretch, overloading & focal pressure of PF.[10, 65, 66]	- Standing on hard surfaces.[32, 40]
Gender: Current literature inconsistent**:** - Increased prevalence in men.[26] - Increased prevalence in women.[14, 18, 29]	**Environment:** - Inappropriate footwear.[2, 5, 19, 48]
Ethnicity: No reported associations.	

Biomechanical dysfunction & anatomical variants:

- Reduced range of ankle joint secondary to tight Achilles tendon strains plantar fascia.[3, 5, 16, 29, 32, 53] Riddle et al[19] considers this the most important risk factor.
- Tightness of posterior lower limb muscles[41, 51, 56] and specifically hamstring tightness.[33, 46]
- Decreased 1st MPJ range of extension due to tight Achilles tendon.[5, 35]
- Flexor digitorum brevis tendinopathy secondary to stress shielding.[14]
- Calcaneal spur. [7, 9, 10, 42-45]
- Plantar fascial thickening [50]

Lifestyle:

Rapid increases in activity levels allied to physical demands of sport or occupation.[5, 19]

Sleeping posture:

Can contribute to posterior leg muscle contraction.[51, 67]

Acquired systemic diseases:

- No association with systemic factors.[1, 5]
- Rheumatoid arthritis.[67]
- Ankylosing spondylitis.[3, 9, 67]
- Diabetes mellitus where micro/macro vascular impairment results in accelerated fasciosis.[9]
- Chemotherapy, retroviral infection & rarely gonococcus & TB.[9]

Sport:

- Overuse injury combined with running surface.[22]
- Poor technique.[26]
- Training errors.[30]
- High intensity.[5]
- Fatigue.[25]
- Repetitive loading.[22]
- Muscle dysfunction and inflexibility.[30, 25]

Major trauma (Laceration/puncture wound, previous foot surgery).

No reported associations

Oestrogen levels:

- Low oestrogen levels in female athletes leads to reducted collagen elasticity.[52]

Vascular perfusion of ligament:

- Reduced vascular supply to plantar fascia & subsequent poor nutrition.[12]

Fluoroquinolone antibiotics:

A tendon (Achilles) association exists[68] but none in ligaments to-date.

Inherited systemic diseases:

No association has been reported.

Genetic:

Potential candidate gene variants (based on tendon studies):

- COL5A1.[58]
- MMP1.[59, 60]
- MMP3.[60, 61]
- MMP8.[60]
- MMP10 & MMP12.[59, 60]
- GDF5.[60]
- TGFB.[62]
- ADAMTS1, ADAMTS2, ADAMTS4, ADAMTS5, ADAMTS15.[63, 64]
- TIMP1, TIMP2, TIMP3, TIMP4.[60]