Journal of Bodywork & Movement Therapies 21 (2017) 205–211

Contents lists available at ScienceDirect

Journal of Bodywork & Movement Therapies

journal homepage: www.elsevier.com/jbmt

PREVENTION & REHABILITATION: EDITORIAL

Plantar heel pain

First steps

A patients description of rising in the morning, placing their feet upon the floor, and their first step eliciting pain under the foot (see Fig. 1), usually alerts the health professional to the likely possibility that the diagnosis will be related to the plantar fascia. The pain may subside and return later in the day particularly if the patient has been on their feet, but improves with rest. Heel pain can also present following an increase in weight bearing activity. (McPoil et al., 2008; Martin et al., 2014; Berbrayer and Fredericson, 2014). The diagnosis of Plantar fasciopathies is made by assessing the clinical picture as there is no gold standard test for the condition (Beeson, 2014).

Heel pain affects millions of people worldwide and is divided into two broad categories, posterior heel pain, and inferior (plantar) pain. Posterior heel pain most commonly is related to the Achilles tendon and its increasing prevalence can be associated with the continuation of exercise into older age (Dinneen et al., 2016). This editorial focuses on inferior heel pain, of which Rosenbaum et al. (2014) suggests that 'plantar fasciitis is the most common culprit, accounting for 80% of patients with inferior heel pain,' and is predicted to affect 1 in 10 people in their lifetime. They suggest that mechanical, rheumatologic or neurologic conditions can all present with plantar heel pain. Hossain and Makwana (2011) suggest that pain in the plantar heel area be initially named Heel pain syndrome (HPS) until clarification of the symptoms can establish a firm diagnosis.

Plantar fasciitis, fasciosis or fasciopathy?

Berbrayer and Fredericson (2014) differentiate acute plantar heel pain symptoms produced by inflammation, and, chronic pain in the same region, from degenerative causes. As the clinical course of plantar fasciopathy follows a predictable path they identify the acute phase as lasting 4 weeks, a sub-acute phase which persists from 4 weeks to 3 months and a chronic phase that goes beyond 3 months. Despite inflammation being cited as a primary cause there is little evidence to support the use of NSAID's (non-steroidal anti-inflammatory drugs) in the papers they quote. NSAID's are typically prescribed in conjunction with other interventions as part of a trial of conservative measures, and 76% of patients report improvement with their use, (Wolgin et al., 1994). Lemont et al.'s (2003) view that 'Plantar Fasciitis' is a condition without signs of classic inflammation, swelling, erythema or macrophage infiltration and is characterised, on histological examination, by chronic degenerative tissue changes, and therefore should be renamed plantar fasciosis, is continuing to gain traction (Hossain and Makwana, 2011) and reflects the sea-change reappraisal of the clinical nature of tendinopathies (Maffulli et al., 2003; McNeill, 2015). The use of the term plantar fasciopathy blends the inflammatory and degenerative elements to the condition.

Rosenbaum et al. (2014) still state that NSAID's are an appropriate treatment for HPS, which may reflect the view that there is an inflammatory pathway to plantar fasciopathy (see Fig. 2) as well as a degenerative one. Rose and Singh (2016) appear to agree, they describe; acute events with a sudden onset of pain during sporting activities as possibly being 'plantar fascia tears' and best treated by ice, NSAID's and immobilisation till symptoms reduce, and a stiff soled boot for 6 weeks, or, 'peri-fascial oedema' again best treated with oral NSAID's or even an ultrasound guided steroid injection above the plantar fascia (see steroid injection discussion later).

As HPS involves many different diagnoses (see Table 1.) that require quite different treatment approaches it is clear that an understanding of the structure of the inferior heel area, the biomechanics of the foot, the risk factors associated with injury and an assessment process is imperative if the path to recovery is to be maximised by the treating Clinician.

Anatomy biomechanics and risk factors

The plantar fascia is actually a broad fibrous triangular aponeurosis extending from the medial and anterior undersurface of the calcaneum to the base of the five proximal phalanges, it travels in a constantly present central band, though in some, the medial and/or lateral bands are missing (Hossain and Makwana, 2011). The intrinsic muscles of the foot are compartmentalised by the plantar fascia. Posteriorly it is contiguous with the Achilles tendon and when the calf musculature contracts, the plantar fascia is tightened by this connection. The structure of the stress dissipating fibrocartilage attachment to the medial calcaneal tubercle helps the aponeurosis to withstand significant loads.

The inferior heel is protected by its fat pad, which acts as a shock absorber, the reinforced, fat globule filled honeycomb fat pad is known to degenerate beyond 40 years of age, losing thickness and height, while softening and reducing its protection of the heel (Rosenbaum et al., 2014), not surprising when the heel's fat pad has to dissipate 250% of body weight while running (Rosenbaum et al., 2014). Beeson (2014) describes an increasing thickness of the heel pad, but with the same effect of a loss of

http://dx.doi.org/10.1016/j.jbmt.2016.11.001

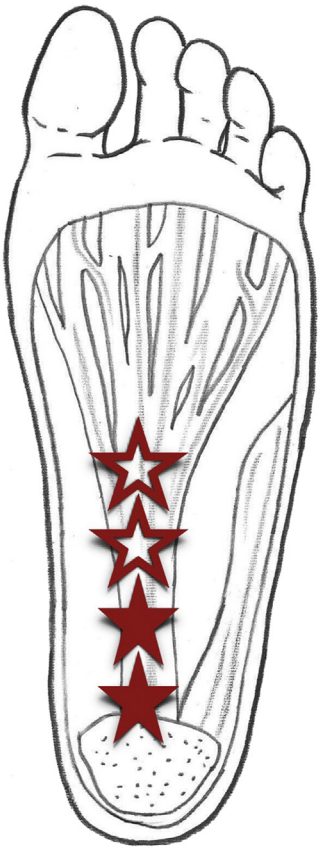

Fig. 1. Area of reported pain of Plantar fasciopathies. The medial calcaneal tubercle is usually painful and the more anterior calcaneal tuberosity (solid stars) may also reported to be tender in insertional Plantar fasciopathies. Pain may also present more distally over the central plantar aponeurosis in non-insertional fasciopathies (Hossain and Makwana, 2011), before it splits into the 5 digital slips (Berbrayer and Fredericson, 2014) Line drawing: Craig McNeill.

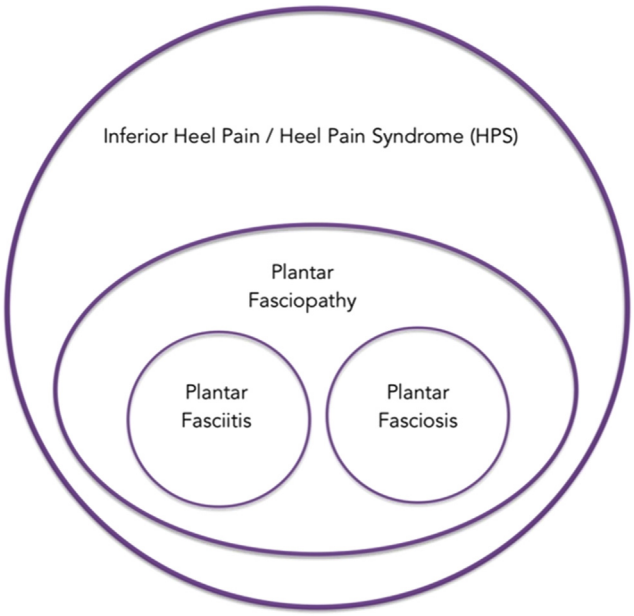

Fig. 2. Nomenclature. (Note; the size of the circle does not represent incidence).

Table 1
Alternate diagnoses to Plantar fascia related causes of inferior heel pain.

Bone
Calcaneal stress fracture
Calcaneal bone marrow oedema
Bone tumour
Osteomyelitis (infective)
Nerve
Tarsal tunnel syndrome
Nerve to abductor digiti minimi neuropathy
Medial calcanea neuropathy
Lumbo-sacral (S1) radiculopathy
Metabolic
Osteomalacia
Paget's disease
Hyperthyroidism
Inflammatory
Seronegative arthropathy
Inflammatory Bowel disease
Gout
Rheumatoid arthritis
Other soft tissue
Heel fat pad bruise
Heel fat pad atrophy
Flexor Hallucis Longus Tendinopathy
Bursitis
Tumours

From Rose and Singh 2016, Hossain and Makwana 2011.

heel pad elasticity. The fat pad of symptomatic feet presents a significantly lower energy dissipation ratio when compared to asymptomatic feet (Wearing et al., 2009). When a patient reports pain on prolonged standing, bilateral pain and night pain, but without first step pain, heel pad atrophy may well be implicated (Martin et al., 2014).

The role of the plantar fascia is to help support the arch of the foot via the skeletal truss formed by the hind foot and forefoot and held along the bottom by the aponeurotic structure. A pes planus foot is predicted to apply greater loads to the plantar fascia and this matches the risk factor of over-pronation in HPS. Beeson (2014) quotes 8 papers linking over pronation as a key risk factor in plantar fasciopathies, at odds with Hossain and Makwana's (2011) view that over-pronation has not been verified as a risk factor, see Table 2. As it is, the gait cycle adds tension to the plantar fascia from heel strike to just before mid stance and the tightening fascia prevents excessive pronation, unless the foot is dysfunctional (Wallden, 2015a). The windlass tightening effect (see Fig. 3) is used for propulsion. The intrinsic muscles of the foot assist in the creation of the desirably timed supinated and stiffened foot, and their loss of function, atrophy or over length reduces movement efficiencies.

A high body mass index (BMI) is a significant risk factor in one major group of patients, the over 40, often female, non athletic individual, and this risk is amplified if there is an increase in weight bearing activity, such as occupational tasks with unsuitable footwear.

Rosenbaum et al. (2014) suggest heel spurs are associated with HPS but do not necessarily cause pain, they state that both heel spurs and pain 'may develop from a common underlying pathologic condition.' It is possible then that excess weight in mature individuals in modern man is an explanation for the increased incidence of calcaneal spurs, however, in the prehistoric record, hunter-gatherers who, in a specific population studied by Weiss (2012), died at significantly younger ages than modern humans, on average at 32.2 years if male and 37.2 years if female. Yet this prehistoric group still presented with 3× more calcaneal spurs than modern populations. This indicates that the increased activity likely required for survival in prehistory had an effect in generating the