

Treatments for posterior tibial tendon dysfunction

Posterior tibial tendon dysfunction is a progressive condition that can lead to disability, and therefore early diagnosis is important. Carly Bodill and Michael Concannon discuss the many treatment options available

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Posterior tibial tendon dysfunction (PTTD) is the most common cause of adult-acquired flatfoot, and if patients are not treated effectively they risk progression of the condition and possible severe deformity of the foot (Arai et al, 2007). The progression of PTTD can result in additional pathology, including stretching of the medial ankle ligaments which can result in a valgus alignment of the calcaneus and abduction of the forefoot (Augustin et al, 2003).

Conservative treatment is an alternative pathway to surgical options and can reduce the physical and financial burdens placed on a patient (Alvarez et al, 2006). Conservative management includes education, stretching and strengthening exercises, ankle braces, foot orthotics, exercise modifications, and footwear changes (Ringleb et al, 2007).

The structure and function of a healthy tibialis posterior tendon

The tibialis posterior muscle is the deepest muscle on the back of the leg that originates from deep within the calf region. The origin of the tibialis posterior is from the proximal tibia, fibula and interosseous membrane and its tendon tracks behind the posterior aspect of the medial malleolus and inserts in various places on the plantar medial aspect of the foot. The tendon divides into separate slips, with the main continuation of the tendon attaching to the navicular and to the inferior surface of the medial cuneiform. There are also attachments across the second to fourth metatarsals.

There are more slips to the lateral cuneiform and cuboid described. One of the indicated functions of this muscle is to invert and supinate the foot in gait. Its activity during walking also suggests it may be involved in controlling pronation and distribution of weight through the metatarsal heads but is also recognized as a key stabilizer of the medial longitudinal arch of the foot (Ellis H, 1989; Augustin et al, 2003; Squires et al, 2006; Semple et al, 2009; Drake R et al, 2010; Marieb and Hoehn, 2010).

The posterior tibial muscle affects the function of the subtalar and midtarsal joints during gait, providing an inversion moment at the

subtalar joint. It is the main supinator of the foot during stance (Durrant et al, 2011).

Supination is considered an essential action within the gait cycle as it is observed that those patients with a subtalar joint that remains in its maximally pronated position may be more prone to injury owing to the lack of pronation available for shock absorption (Kirby, 2000). It was recognized as early as 1977 when Root et al (1977) described feet that are unstable, as a result of mid foot pronation, relatively inefficient, resulting in wasted foot joint energy as the foot may struggle to generate a sustained plantar force during gait.

Aetiology and prevalence

PTTD can be described as a sudden or progressive condition which may or may not present with pain and loss function. Although there are numerous aetiological factors that have been described there is no universally accepted cause. There may be a report of an acute traumatic injury or inflammatory synovitis secondary to mechanical overuse or systemic disease. There may also be chronic

Table 1. Causes and risks of posterior tibial tendon dysfunction

Inflammatory	Rheumatoid arthritis Other inflammatory diseases
Mechanical	Tendonopathy/impairment Functional impairment Abnormality of talocalcaneal joint Soft tissue tension/contraction Pre-existing flatfoot Excessive functional pronation
Metabolic	Increased cholesterol Menopause
Others	Infection Ageing process
Non-specific causes	Pregnancy Microvascular impairment Macrovascular impairment
Non-specific factors increasing risk of developing chronic pathology	Increased body mass index Smoking Localized steroid exposure Long-term use of medication Diabetes Hypertension Previous rearfoot trauma/surgery

From: Tao et al, 1999; Kirby, 2000; Bowring and Chockalingam, 2009

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