Office-Based Management of Adult-Acquired Flatfoot Deformity

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KEYWORDS

• Flatfoot • Nonoperative management • Posterior tibial tendon dysfunction
• Medial ankle pain • Hindfoot valgus • Midfoot abduction

KEY POINTS

- Adult-acquired flatfoot deformity is a common problem, caused by dysfunction of the posterior tibial tendon.
- Early in the disease process, patients present with medial-sided pain and swelling, which eventually progresses to lateral-sided pain as the deformity worsens.
- Nonoperative treatment includes nonsteroidal anti-inflammatory drugs, weight loss, and various forms of bracing and immobilization for symptom control.
- If nonoperative management fails or the deformity worsens, patients may be candidates for surgical intervention.

INTRODUCTION

Adult-acquired flatfoot deformity (AAFD) is defined as a loss of the medial longitudinal arch secondary to failure of the posterior tibial tendon (PTT) and posteromedial soft tissue structures.[1] AAFD was initially known as PTT dysfunction (PTTD), because the condition was thought to be solely caused by failure of the PTT. However, over time, it was recognized that dysfunction and attenuation of the surrounding ligaments also occur, leading to a progressive deformity of the foot.[2]

PTT synovitis was initially described in 1955 by Fowler.[3] He is credited with reporting the first series of 7 patients, in which a tenosynovectomy provided pain relief for patients with tenosynovitis. In 1953, Key reported the first case of a partial PTT rupture. He treated this with debridement and noted that the patient was left with 15% disability of that foot.[1,3] Various theories have been described regarding the origin of PTTD, but the most likely cause is repetitive microtrauma to the tendon, which leads to an inflammatory response and then finally tendon disruption.

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FOOT ANATOMY

The bony anatomy of the hindfoot consists of the subtalar joint, the talonavicular joint, and the calcaneocuboid joint. The talonavicular and calcaneocuboid joints together are known as the *transverse tarsal joints*.[1]

The posterior tibialis muscle originates from the posterior aspect of the tibia and interosseous membrane. It runs immediately posterior to the medial malleolus and divides into multiple slips, inserting on the navicular tuberosity, the plantar surfaces of the second through fourth metatarsals, and the sustentaculum tali, cuboid, and cuneiforms.[1,3,4] The PTT functions to invert and plantarflex the foot. It is also the primary stabilizer of the medial longitudinal arch and hindfoot, and is responsible for locking the midtarsal joints to assist in effective gait patterns.[5]

The spring ligament, also known as the *calcaneonavicular ligament*, is crucial in supporting the medial arch, and therefore its failure contributes to the development of AAFD. The ligament runs from the anterior portion of the sustentaculum tali to the plantar medial aspect of the navicular.[1] It is thought to have 2 components: a superomedial band and an inferior band. Together, these act as a cradle for the plantar medial aspect of the talar head. As the spring ligament becomes attenuated, or ruptures, the talus can migrate in a plantar and medial fashion, leading to subluxation of the talonavicular joint.[1,2]

The deltoid ligament is the primary medial stabilizer of the tibiotalar joint. It has multiple components, the most important being the distal portion that will blend with the spring ligament and talonavicular joint capsule. This aspect of the ligament is subject to repetitive stress during midstance.[1] As the foot deformity progresses, increased strain is placed on the deltoid, leading eventually to deltoid incompetence and a valgus deformity of the tibiotalar joint.[3]

BIOMECHANICS AND PATHOPHYSIOLOGY OF PTTD

The gait cycle is divided into the stance and swing phases. The stance phase consists of the heel strike, midstance, heel rise, and toe off. The posterior tibialis muscle is considered a stance phase muscle, and is therefore intimately involved in positioning the foot appropriately for a biomechanically sound gait pattern.

During heel strike, the posterior tibialis will eccentrically contract to slow the eversion of the subtalar joint, which will unlock the transverse tarsal joint complex, allowing collapse of the foot. As the foot progresses to the midstance and heel rise phase, the posterior tibialis will contract, causing inversion of the subtalar joint. The action of inversion of the subtalar joint locks the transverse tarsal joints, creating a rigid midfoot. This rigid midfoot creates a lever, allowing the contraction of the gastrocnemius-soleus complex to propel the foot and body forward.[1,3,5]

Multiple forces are involved in the development of PTTD. As the PTT becomes deficient, a valgus deformity of the hindfoot is noted, which is caused by the excessive eversion forces created by an unopposed pull of the peroneus brevis. During the stance phase, the lack of posterior tibialis contraction and eversion of the hindfoot cause the transverse tarsal joints to remain unlocked. This unlocked position prevents formation of the rigid lever arm, which accelerates the degeneration of the spring ligament.[3]

As AAFD progresses, the talus will become plantarflexed, the calcaneus will internally rotate and evert, and the navicular and cuboid will evert. These changes lead to the pathognomonic hindfoot valgus deformity (**Fig. 1**A) and forefoot abduction (see **Fig. 1**B).[3]