Tendon Transfers in the Treatment of the Adult Flatfoot

Jonathon D. Backus, MD[a], Jeremy J. McCormick, MD[b,*]

KEYWORDS

• Adult acquired flatfoot deformity • Posterior tibial tendon dysfunction
• Tendon transfer • Tibialis posterior • Flexor digitorum longus • Flexor hallicus longus
• Peroneus brevis • Peroneus longus

KEY POINTS

- Adult acquired flatfoot deformity (AAFD) describes a condition of progressive hindfoot valgus, forefoot abduction, and forefoot varus. It is most commonly caused by posterior tibial tendon dysfunction.
- Patients who have AAFD often complain of posteromedial hindfoot pain, a progressive change in the shape of the foot, difficulty with weight bearing, and gait abnormalities.
- Conservative management consists of nonsteroidal antiinflammatory medications, selective corticosteroid injections, and physical therapy. Patients also benefit from temporary immobilization in a cast or boot, custom foot orthosis, or a custom ankle-foot orthosis or Arizona brace.
- In stage I and II posterior tibial tendon (PTT) dysfunction in which conservative management has failed, many clinicians think that the deformity can be corrected with PTT debridement, tendon transfer, and a medial displacement calcaneal osteotomy. Spring ligament repair may also be indicated in certain cases.
- In more significant stage II deformities, concomitant procedures such as an additional lateral column-lengthening osteotomy and selective fusions may be required.
- In the case of a stage II flexible flatfoot deformity, our preference is to excise the diseased PTT tendon and use a flexor digitorum longus (FDL) transfer through a tunnel in the navicular tuberosity. The FDL tendon is typically long enough to pass through the tunnel from plantar to dorsal and be secured back to itself with nonabsorbable suture.

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[a] Department of Orthopaedic Surgery, Washington University School of Medicine in St Louis, Campus Box 8233, 660 South Euclid Avenue, St Louis, MO 63110, USA; [b] Department of Orthopaedic Surgery, Washington University School of Medicine in St Louis, 14532 South Outer Forty Drive, Suite 210, Chesterfield, MO 63017, USA
* Corresponding author.
E-mail address: mccormickj@wudosis.wustl.edu

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- Patients with stage III and stage IV deformities typically require a hindfoot arthrodesis. A plantarflexion osteotomy or fusion of the first ray may also be required in patients with persistent forefoot varus. Furthermore, there may be a role for tendon transfer in patients who undergo hindfoot arthrodesis.
- The decision to proceed with surgical correction of AAFD should be made after conservative treatment options have failed. Careful preoperative planning is necessary to choose the appropriate reconstructive procedures given a patient's clinical examination, deformity, and radiographic findings.

PATHOPHYSIOLOGY OF ADULT ACQUIRED FLATFOOT DEFORMITY

Adult acquired flatfoot deformity (AAFD) describes a condition of progressive hindfoot valgus, forefoot abduction, and forefoot varus. Although there are several causes of AAFD, posterior tibial tendon (PTT) dysfunction is the most common cause.[1] The posterior tibialis (PT) muscle is a powerful supinator of the subtalar joint, adductor of the midfoot, and serves to assist plantar flexion of the ankle. It has 9 attachments to the medial midfoot that help this muscle statically support the arch of the foot along with the spring ligament and the plantar calcaneonavicular ligament.[2,3] Therefore, the PT muscle has important functions throughout the stance phase of gait and in the overall structure and function of the foot and ankle.

During the early part of stance phase, the PT muscle contracts eccentrically allowing the hindfoot to pronate in a controlled fashion and subsequently unlock the talonavicular and calcaneocuboid joints. This function allows the foot to become supple and adapt to the undulations of the underlying surface that it contacts. In toe-off, the muscle contracts concentrically, supinating the hindfoot and locking the talonavicular and calcaneocuboid joints, which provides a rigid fulcrum for the gastrocsoleus muscle to propel the body forward and begin the swing phase of gait.

In the early stages of PTT disorder, the dysfunctional tendon leaves the spring and calcaneonavicular ligaments unsupported in counteracting the antagonistic hindfoot pronation and forefoot abduction forces of the peroneus brevis (PB). The arch is usually maintained at first; however, with continued PTT dysfunction and the stress of ambulation, the spring ligament eventually fails and the arch gradually collapses.[4] Persistent hindfoot valgus and forefoot abduction eventually result in forefoot varus, which may manifest as dorsiflexion of the first ray or global forefoot varus at the transverse tarsal joint (**Fig. 1**).[5] At first, these deformities are flexible and passively correctable; however, consequent synovitis and scarring lead to the foot becoming fixed in its malpositioned posture.

SYMPTOMS

Patients with AAFD typically complain of posteromedial hindfoot pain below the medial malleolus, a progressive change in the shape of the foot, and difficulty with weight bearing; however, patients rarely report a specific instance in which the foot collapses. Some patients also experience lateral pain near the sinus tarsi if the deformity is such that the lateral malleolus impinges with the calcaneus.[6] Patients often note gait disturbances such as difficulty with push-off, long strides, and an inability to run.[7]

STAGING

Johnson and Strom[8] described the first classification system for PTT dysfunction and AAFD in 1998. This classification is helpful in that it is often used to determine