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Letter to the Editor

Re: Adult Acquired Flatfoot Deformity Following Tibialis Posterior to Dorsum
Transfer: A Case Report.
FAI, 29(3): 351–3, 2008

Dear Editor,

We read with interest "Adult-Acquired Flatfoot Deformity Following Posterior Tibialis to Dorsum Transfer: A Case Report" and were pleased to note further literature that suggests that the action of peroneus brevis is not largely responsible for the adult acquired flat foot deformity which develops as part of posterior tibial tendon dysfunction. We would like to point out however, that there are several other reports in the literature which were not mentioned in the discussion.

Tibialis posterior dysfunction in a patient without functioning peroneii was reported previously in ***Foot & Ankle International*** as a case report.[3] In that case report, the authors suggested that the adult acquired flat foot developed in patients when the tendoachilles had an everting action across the subtalar joint due to pre-existing mild pes planus. Furthermore, the same group presented a group of patients with anterior transfer of tibialis posterior who, at mid term follow-up, had no evidence of pes planus on Harris-Beath pressure mats despite having grade 4 or better power of eversion. Their conclusion was, that provided patients have no pre-existing pes planus, anterior transfer of tibialis posterior will not lead to a change in foot shape.[5,6] They also noted that the patients' ability to evert their foot had not produced an adult acquired flat foot. This conclusion is different to that of Mizel et al, who presented a series of patients with anterior transfer of tibialis posterior who had not developed an acquired pes planus and in whom the peroneii were not functioning.[4] In that study, the authors concluded that imbalance between the peroneal tendons and the dysfunctional tibialis posterior tendon, is responsible for the adult acquired flatfoot deformity seen with posterior tibial tendon dysfunction. Clearly there is more literature to support the hypothesis of Omid, Thordarson, and Charlton and their case report would have been strengthened by referring to it.

There is also another mechanism, by which the action of triceps surae can be contributary to the development of an adult acquired flatfoot, and this is related to the strain an individual with pre-existing pes planus and tight gastrocnemii places on their medial arch. Gastrocnemius tightness is often seen in patients with pes planus and the limitation of ankle dorsiflexion produced by gastrocnemius tightness is accommodated by the increased dorsiflexion at the transverse tarsal joint which remains unlocked due to the subtalar joint remaining pronated.[2] Over time, the spring ligament and plantar aspects of the naviculo-cuneiform and tarso-metatarsal ligaments become attenuated and increased strain is placed on the tibialis posterior tendon, leading to pathology.[1] This would suggest that care should be taken, to ensure that patients with foot drop do not develop gastrocnemius tightness, as it could be that over time, compensatory increased dorsiflexion through the mid foot, will lead to an acquired flat foot deformity.

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We would like to thank Drs. Kohls-Gatzoulis and Sakellariou for their interest in our article. Each of the additional articles that they have cited above does add to our hypothesis that the unopposed peroneal muscles are not the sole cause of the adult acquired flat foot deformity in patients with posterior tibial tendon dysfunction. While each of the articles they have cited represents patients with a different clinical scenario than ours, they do reinforce the theory that the unopposed peroneus brevis, while clearly a contributing factor to the flatfoot deformity in patients with posterior tibial tendon dysfunction, is not the sole source of this deformity. It has also been our clinical experience that patients with posterior tibial tendon dysfunction usually have a pre-existing flatfoot deformity. Thus, the gastroc–solus can act as a chronic deforming force in these patients when the heel is in valgus. While they do cite a cadeveric, biomechanical study that demonstrated increased stress across the midfoot with increased tension in the gastroc-solus, our patient did not have a gastroc-solus contracture. They did cite additional studies that demonstrated the converse clinical situation to our paper, that some patients did not develop a flatfoot deformity despite functioning peroneal muscles. In hindsight all these papers should have been cited in our discussion and we thank Drs. Kohls-Gatzoulis and Sakelllariou for their interest in reinforcing the theory that the peroneal muscles do not cause a flatfoot deformity in isolation.

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