Diagnosis and Surgical Management of Flexor Digitorum Accessorius Longus–Induced Tarsal Tunnel Syndrome

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The flexor digitorum accessorius longus is a rare muscular occurrence in the lower extremity. It has been reported as an etiology of tarsal tunnel syndrome through prior case reports. By means of individual case study, we revisit flexor digitorum accessorius longus as a cause of tarsal tunnel syndrome. This case study discusses diagnosis along with surgical treatment of tarsal tunnel syndrome induced by the presence of flexor digitorum accessorius longus. (The Journal of Foot & Ankle Surgery 46(6):484–487, 2007)

Key words: accessory muscle, flexor digitorum longus, magnetic resonance image, nerve entrapment, tarsal tunnel syndrome, tibial neuropathy

Flexor digitorum accessorius longus (FDAL) is an accessory muscle of the lower leg, the incidence of which has been reported to be 4% to 8% based on cadaveric dissections (1–5), making it the most commonly occurring skeletal muscle anomaly in the medial aspect of the leg and ankle (3). FDAL may have a single or double head (4), and it usually displays a fleshy, tendinous, or aponeurotic appearance as it courses from the leg to the tarsal tunnel. Variable sites of origin have been reported, including the tibia, fibula, deep fascia, transverse intramuscular septum, flexor digitorum longus, flexor hallucis longus, soleus, peroneus brevis, and/or the calcaneus (4). FDAL usually courses through the tarsal tunnel deep to the neurovascular bundle, but it may traverse superficial to the vital structures, and, in such cases, the FDAL is in a position to compress the tibial nerve. After traversing the tarsal tunnel, FDAL travels through the porta pedis to insert into the flexor digitorum longus (FDL) and/or the quadratus plantae (4). Because of its anatomic location, FDAL has been implicated as a cause of tarsal tunnel syndrome (TTS) (6–9).

Identifying the cause of TTS can be a difficult clinical task. Cimino's 1990 review of 186 cases of TTS in 24 different reports revealed the etiology to be idiopathic in 25 (13%) of the cases, whereas there was no mention of etiology in 64 (34%) of the cases (10). In their study of 40 feet in 33 patients with TTS, in which there were 29 feet with electrodiagnostically confirmed nerve entrapment, Frey and Kerr reported that 88% of the involved extremities displayed an inflammatory or mass lesion localized deep to the flexor retinaculum identified by magnetic resonance imaging (MRI) (11). Confirmation of FDAL as the etiology of TTS is often made intraoperatively, although identification of an accessory muscle can be made with MRI (12, 13). In the presence of persistent symptomatology, surgical intervention becomes a strong consideration for the treatment of TTS. Abnormal electroneurodiagnostic studies, as well as MRI findings consistent with inflammatory or mass lesions within the tarsal tunnel, further indicate that surgical intervention may be helpful.

The case described in this report illustrates that the diagnosis of TTS can be made without first establishing an exact etiology of the condition. Furthermore, the case makes clear the importance of having a high degree of suspicion for the presence of accessory muscle when interpreting MRI films as part of the diagnostic work-up. We also describe a method of skeletal muscle and tendon excision that was successful in resolving an FDAL-induced TTS.

Case Report

The patient was a 54-year-old woman with a chief complaint of chronic left heel pain of more than 2 years' duration. Her past medical history included hyperlipidemia and low back pain, and she denied any history of trauma. She complained of pain that radiated along the course of the posterior tibial nerve in the left lower leg but denied any areas of paresthesia. The patient had been treated for heel

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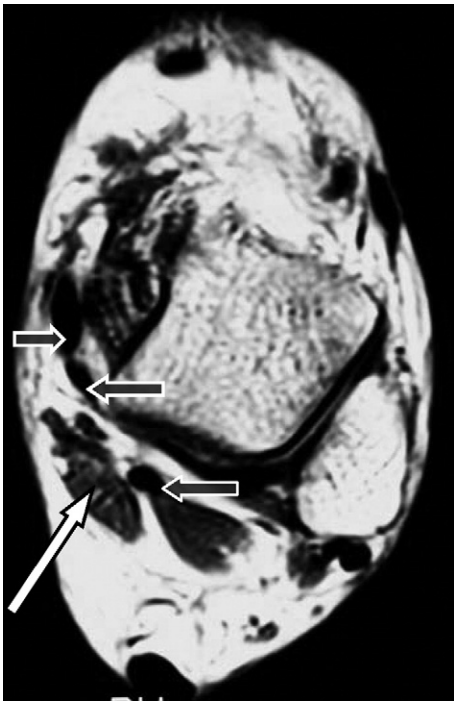

FIGURE 1 Note the accessory muscle belly (*white arrow*) in relation to the tibialis posterior, flexor digitorum longus, and flexor hallucis longus (*black arrows*).

pain approximately 2 years earlier with the use of supportive tapings and foot orthotics. She had also sought pain relief through chiropractic adjustment. These treatment modalities brought limited relief. Physical examination revealed normal, pain-free foot and ankle range of motion in the affected limb. Pain was elicited on palpation of the medial tubercle of the calcaneus, as well as with side-to-side compression of the calcaneus. A positive Tinel sign was elicited on percussion of the posterior tibial nerve. Plain film radiographs of the left foot and ankle were interpreted as normal. Nerve conduction velocities revealed normal amplitude, latency, and velocity on the right lower extremity. Left-sided findings demonstrated absent sensory nerve action potentials in the distribution of the medial plantar nerve. Electromyogram studies were observed to be normal, with no evidence of muscle membrane instability, bilaterally. MRI was also performed (Figure 1), and the initial report made no mention of a space-occupying mass or other irregularities within the tarsal tunnel. Given the clinical findings, duration of symptoms, and failure to alleviate symptoms by conservative management, surgical intervention was performed.

Surgical exposure of the tarsal tunnel entailed use of a curvilinear incision made over the region of the tarsal canal posterior to the medial malleolus, and this incision was carried distally to the level of the abductor hallucis muscle. The dissection was conducted without the use of tourniquet hemostasis. Small-caliber bleeding vessels were either electrocoagulated or ligated. The flexor retinaculum was incised longitudinally, and a large muscle belly was identified just deep to the retinaculum, superficial to the neurovascular bundle (Figure 2). Further dissection revealed a tendon extending from the muscle belly, just proximal to the porta pedis. At this level, the tendon coursed over and then under the posterior tibial nerve, compressing the nerve against the deeper structures. The tendon continued through the porta pedis to enter the plantar vault. When proximal tension was applied to the anomalous tendon, plantarflexion of lesser digits 2 to 5 was observed.

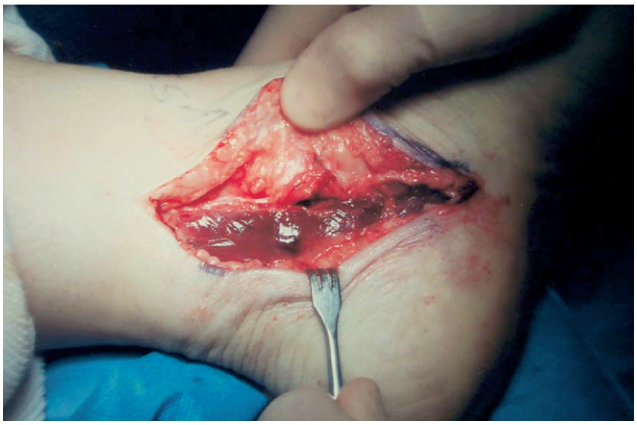

FIGURE 2 After reflection of the retinaculum, the belly of flexor digitorum accessorius longus (FDAL) was identified.

On further inspection, the tibialis posterior, flexor digitorum longus, neurovascular bundle, and flexor hallucis longus were all identified and noted to be in their normal anatomic positions within the canal. The surgeons concluded, based on identification of the normally occurring elements of the tarsal tunnel, that the muscular anomaly was an accessory flexor digitorum longus. Although the definitive origin and insertion of the anomalous muscle could not be determined without more extensive dissection, and given the visible entrapment of the nerve by the FDAL tendon, it was decided not to perform any further dissection. In an effort to eliminate entrapment of the posterior tibial nerve, the tendon of the FDAL was sharply transected at its most distal, grossly visible level (Figure 3). The muscle belly was then transected at the proximal aspect of the wound with the electrosectioning unit to reduce hemorrhage from the highly vascular skeletal muscle.

After removal of the accessory muscle and tendon, the posterior tibial nerve was observed to have several adhesions at the level of the medial malleolus. External neurolysis was performed to free the nerve from the adhesions, after which it was further decompressed distally at the level of the porta pedis by transecting the deep fascial septum between the medial and lateral plantar branches. The medial and lateral branches were observed to be intact with no