CHAPTER 6

TARSAL TUNNEL SECONDARY TO OS TRIGONUM

Marc R. Bernbach, D.P.M.

The correlation between os trigonum and tarsal tunnel syndromes has been previously described. The close relationship between the os trigonum and flexor hallucis tendon can produce inflammation of the flexor sheath. This inflammation of the tendon sheath may result in compression of the posterior tibial nerve within the confines of the tarsal tunnel. A review of the condition, and a case report are presented. Magnetic resonance imaging as an aid in diagnosis is detailed.

In 1962, Keck and Lam separately introduced the syndrome and the terminology of tarsal tunnel. The condition has been described as a compression neuropathy of the posterior tibial nerve, and/or one of its branches. The nerve or nerves are usually affected as they pass inferior to the flexor retinaculum and abductor hallucis muscle belly. Symptoms include radiating pain in the tarsal tunnel region which is aggravated by ambulation or shoe pressure.

TARSAL TUNNEL ANATOMY

The neurovascular structures of the tarsal tunnel pass from the posterior compartment of the leg and course beneath the flexor retinaculum (Fig. 1). This location marks the proximal beginning of the tarsal tunnel. The fibers of the flexor retinaculum run from the medial malleolus to the calcaneus, and are divided into a fan shape with upper, middle, and lower sections. Inferior to the retinaculum, the tibialis posterior tendon, flexor digitorum longus tendon, posterior tibial nerve and vessels, and flexor hallucis longus tendon occupy distinct fibro-osseous compartments.

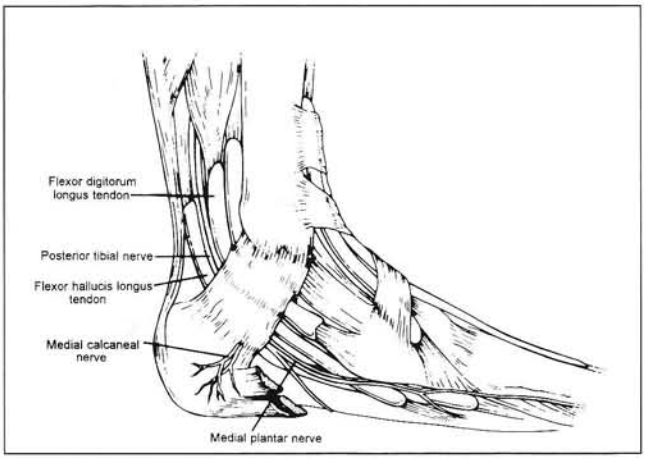

Figure 1. Anatomic relationship of the tarsal tunnel: note the close relationship between the flexor hallucis longus and posterior tibial nerve proximally, as well as the medial plantar nerve distally.

The lateral border of the tarsal tunnel has been described as osseous in its entirety. However, only the middle portion of the lateral wall is actually osseous. The proximal section of the lateral wall consists of the flexor digitorum and flexor hallucis longus tendons, whereas distally, the flexor hallucis longus tendon creates the lateral border for the medial and lateral plantar nerves. Due to the close relationship of the flexor tendons to the nerves, inflammation of the tendon synovial sheath may produce nerve compression and ischemic neuropathy in this confined area.

ETIOLOGY AND DIAGNOSIS

Entrapment of the posterior tibial nerve may occur due to a variety of conditions, including direct trauma, indirect trauma, and space-occupying lesions. Direct trauma can produce an entrapment due to post-traumatic fibrosis. Traumatic causes include ligamentous injury, fractures, chronic pressure from sprains, surgical injury, and tenosynovitis.

Compression neuropathy from a space-occupying lesion or mass may include a varicosity of the posterior tibial veins, ganglion, tumor, fluid retention, osseous prominence, or inflammation. Systemic factors have also been described. Some of these associated with tarsal tunnel are diabetes mellitus, seronegative inflammatory arthropathies, and rheumatoid arthritis.

Symptoms generally exist as poorly localized burning pain, paresthesia, and numbness of the plantar foot and toes. Pain may also concentrate at the plantar medial heel or radiate proximally to the medial calf. The most frequent finding is a positive Tinel's sign with distal paresthesia when the posterior tibial nerve is percussed along its course.

DIAGNOSIS

Diagnosis is often challenging as it requires careful integration of patient history, physical examination, and associated diagnostic studies. These studies may include electrodiagnostic tests, plain film radiographs, magnetic resonance imaging, and diagnostic nerve blocks.

The diagnostic challenge exists since there is no single specific indicator for tarsal tunnel syndrome. Even in the presence of negative electromyography or nerve conduction studies, a posterior tibial nerve compression may exist. The utilization of magnetic resonance imaging can localize lesions within the tarsal tunnel, as well as provide information on pathology in close proximity to the posterior tibial nerve.

CASE REPORT

A 44-year-old white male carpenter presented with burning pain extending from the medial tarsal canal which radiated proximally and distally. There was localized sharp pain on the lateral aspect of the right rearfoot and ankle as well. Several ankle sprains were related subsequent to the initial presentation. Symptoms were aggravated by uneven terrain, prolonged ambulation, and ladder climbing.

Vascular and neurologic examinations of both extremities were grossly within normal limits. The integument was intact without evidence of edema or erythema. Palpation of the sinus tarsi produced mild pain in the right foot, however, there was significant pain with palpation of the tarsal tunnel region. Pressure applied to the posterior tibial nerve produced proximal as well as distal radiation of pain and paresthesia. Pain occurred with palpation of the achilles tendon insertion, as well as the lateral peri-tendinous region. Initial radiographic examination revealed an old postero-lateral process fracture or os trigonum of the right foot.

Motor and sensory nerve conduction studies of the right lower limb were within normal limits. EMG needle examination suggested trace positive sharp waves, however, there was no definitive electrophysiologic evidence for compression of the posterior tibial nerve at the level of the ankle.

Magnetic resonance images of the right ankle were obtained, which revealed a 2 cm fluid collection associated with the flexor hallucis longus tendon, just above the os trigonum (Fig. 2). There

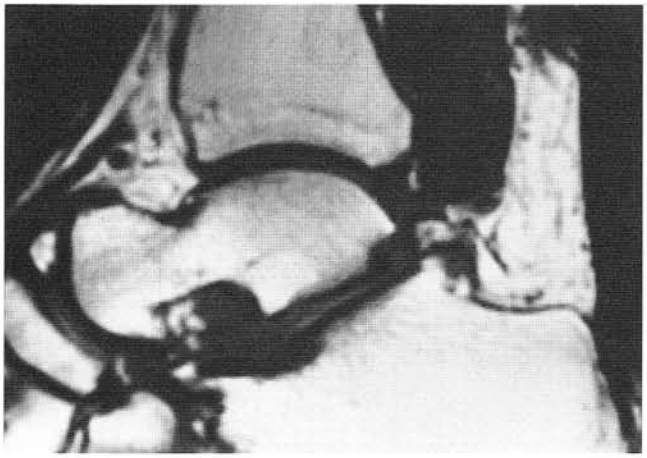

Figure 2. MRI demonstrating fluid collection associated with the flexor hallucis longus tendon just above the os trigonum.

was also fluid within the tarsal tunnel, and fluid in the flexor hallucis longus tendon sheath just distal to the sustentaculum tali (Fig. 3). This was interpreted as tenosynovitis of the flexor hallucis longus tendon.

Figure 3. Notice fluid within the tarsal tunnel and associated tendon sheaths.

As one would expect, a posterior tibial nerve block of the right foot resolved the medial foot complaints and proximal/distal radiation of pain and paresthesia. Accordingly, the lateral rearfoot and tendo achilles complaints remained without effect from the nerve block.

Conservative management over the course of 1.5 years consisted of corticosteroid injections of the tarsal tunnel. A total of three injections were administered four months apart, producing only temporary relief. Additional concurrent therapy consisted of orthotics, physical therapy, and NSAIDs.

Since conservative therapy became ineffectual, a tarsal tunnel release was performed along with excision of the os trigonum through separate medial and lateral incisions. A 7 cm curvilinear peritendonous incision was made on the lateral aspect of the right posterior rearfoot. As the capsular tissue was incised, a copious amount of viscous, cloudy, hemorrhagic synovial fluid exuded from the surgical site. A plano-convex 2.5 x 2.0 x 1.0 cm osseous fragment consistent with an os trigonum was removed. Layered tissue closure was then performed (Fig. 4A, 4B).

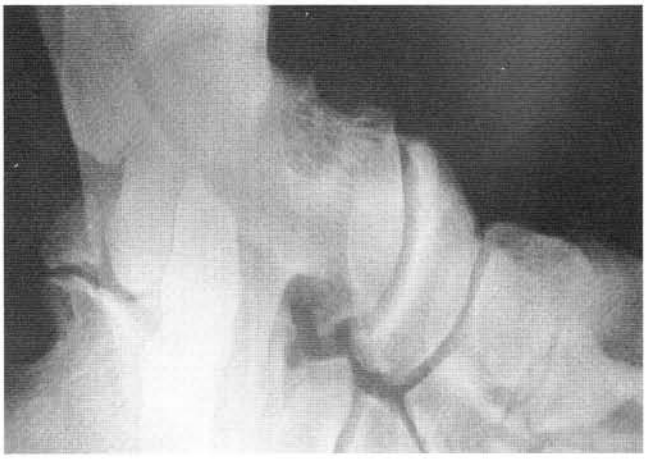

Figure 4A. Preoperative radiograph of hypertrophic os trigonum

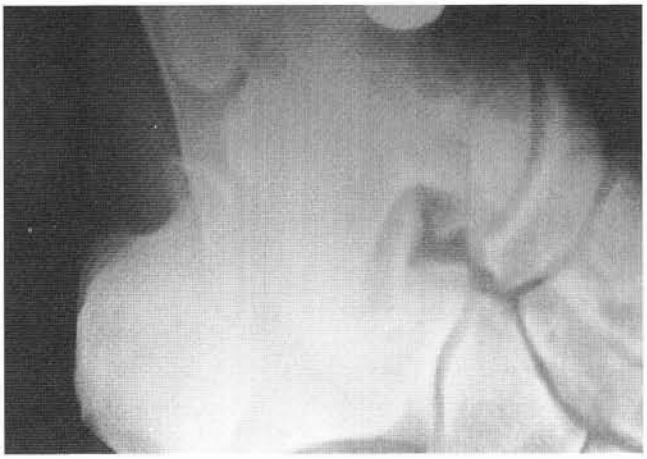

Figure 4B. Post surgical resection of hypertrophic os trigonum.

Following a return to the supine position, a 9 cm medial curvilinear incision was made over the tarsal canal, extending from 4 cm above the medial malleolus to 5 cm below the malleolus. The laciniate ligament above the tarsal tunnel was divided, exposing the tibial nerve and its branches. A viscous, hemorrhagic exudate was found within the tunnel consistent with the fluid from the os trigonum site. The medial and lateral plantar nerves were followed distal to the abductor hallucis muscle belly, into the talocalcaneal tunnel beneath the sustentaculum tali. The flexor retinaculum was not repaired and the subcutaneous tissue and skin were apposed.

Physical therapy and early range of motion were employed postoperatively. At one year follow-up, the patient is asymptomatic with normal ankle, subtalar and midtarsal joint range of motion.

The rationale for performing concurrent procedures was the inflammation of the flexor hallucis sheath occurring in close proximity to the hypertrophic os trigonum. This inflammation produced a space-occupying lesion within the tarsal tunnel and caused a compression neuropathy of the posterior tibial nerve. Isolated performance of a tarsal tunnel release with a coexistent os trigonum syndrome has been described; however, subsequent removal of the os trigonum was required due to a recurrence of symptoms. Surgical excision of the os trigonum alone without tarsal tunnel release would rely on eventual resolution of inflammation. However, ischemic neuropathy would exist until this event occurred.

SUMMARY

Os trigonum syndrome is a condition which produces pain and inflammation of the posterior lateral aspect of the ankle and rearfoot. Flexor hallucis longus tendon sheath inflammation may occur from os trigonum syndrome due to their close relationship. The resultant inflammation of the flexor hallucis longus tendon may produce an ischemic or compression neuropathy of the posterior tibial nerve due to its close proximity to the flexor retinaculum neurovascular compartment (tarsal tunnel). Since the posterior tibial nerve is constricted within the relatively immobile confines of its flexor retinaculum compartment, any inflammation of contiguous tendon structures may produce compression. If conservative care fails, removal of the inflammatory source (os trigonum) as well as posterior tibial nerve decompression is advised.

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