CHAPTER 7

NEUROSCHWANOMA INDUCED TARSAL TUNNEL SYNDROME

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Tarsal tunnel syndrome is an entrapment of the posterior tibial nerve about the medial aspect of the foot and ankle. This was first described by Kopell and Thompson in 1960.[1] The name tarsal tunnel syndrome first appeared in 1962 in separate report by Keck[2] and Lam.[3] They each described a syndrome created by compression of the posterior tibial nerve in the tarsal canal. This may manifest itself as hyperesthesia, dysthesia, or parasthesia along the course of the posterior tibial nerve, radiating into the plantar aspect of the foot and heel along its terminal branches.[4]

ANATOMY

The tarsal tunnel is a fibrooseous space. It is created by the medial malleolus anteriorly, and the sustentaulum tali and the medial wall of the calcaneous laterally.[5,6] The roof is formed by the flexor retinaculum, a specialized continuation of the deep fascia of the leg.[7] The flexor retinaculum attaches to the medial malleolus and the medial process of the calcaneous.[8] Four compartments are formed within this area. The first compartment contains the tibialis posterior tendon, the second the flexor digitorum longus tendon, the third contains the neurovascular bundle (posterior tibial artery, nerve, and vein), and the fourth contains the tendon of flexor hallucis longus.[6,8]

The posterior tibial nerve is a branch of the sciatic nerve. It gives off its medial and lateral plantar nerve branches in the region of the tarsal tunnel. Up to 7% of the time however, the branching occurs prior to entry into the tarsal tunnel.[9,10] The calcaneal branch is much more variable. It originates above the tarsal tunnel 35% of the time, within the tunnel 34% of the time, and as a branch of the lateral plantar nerve 16% of the time.[10]

The medial and lateral plantar nerves enter the plantar aspect of the foot at the distal aspect of the tarsal tunnel. This occurs when they pass deep to the abductor hallucis muscle. This area between the muscle and the calcaneus is often referred to as the porta pedis.[8]

ETIOLOGY

Tarsal tunnel syndrome has a myriad of origins. In a review of the literature, the largest single origin was noted to be idiopathic. This accounted for 21%[5] to over 50% of the cases.[11] In a smaller series, it was reported that ganglia accounted for 36% of the cases.[12] Other significant causes include trauma, varicosities, heel varus, heel valgus, and fibrosis.[5] Separate reports have also identified neuilemomas,[13] anomalous muscle[14] and proliferative synovitis within the tarsal tunnel in patients with rheumatoid arthritis.[15]

In 1985, Chardoroff and Ball reported on a previously unidentified double crush syndrome of the lower extremity involving tarsal tunnel syndrome and an additional lesion of the nerve.[16] This had been previously reported in the upper extremity in 1973 by Upton and McComas.[17] Although the second lesion may be of mechanical origin, it also may be a generalized disease such as diabetes mellitus, hypothyroidism, or alcoholism.[18]

DIAGNOSIS

The diagnosis of tarsal tunnel syndrome is made primarily through history and physical examination.[5,6,11] The patient may complain of parasthesia or dysthesia in the foot at the plantar aspect of the heel, sole, or the toes. This may be exacerbated with activities. With long standing symptoms, a complaint of severe night pain is common.[11] A positive Tinel's sign is present in the majority of cases. In one study, 100% of their patients were found to have a positive Tinel's sign.[19] Depending on the area of entrapment, the pain may radiate to the heel, toes, or sole of the foot.

A thorough evaluation of the patient should also include elimination of lower back involvement. Any evidence of a stocking and glove neuropathy should create a high index of suspicion for a systemic disease as the culprit and the appropriate laboratory studies should be obtained. The patient should be observed weight bearing to evaluate for any visible deformity of the foot or ankle.

The tarsal tunnel region should be inspected and palpated for any masses, pain or edema which may be suggestive of tenosynovitis or space occupying lesions.

If varicosities are suspected then Perths' tourniquet test may help to establish the diagnosis.[20] Radiographs may be obtained to evaluate for any osseous involvement. Injections of local anesthetic and corticosteroids can aid in diagnosis as well as offering possible therapeutic benefits.[6] MRI may reveal any space occupying lesions. Utilization of MRI in conjunction with the Perths' test has been suggested to aid in the diagnosis of venous insufficiency.[21]

Electrodiagnostic studies (EMG, NCS, Sensory Action Potentials) have also been utilized in the diagnostic armamentarium. The degree of positive results has been noted to vary greatly. Sensory action potentials are thought to be the most sensitive for compression neuropathy with a reported 90.5% sensitivity.[22] A positive result result may help to establish the diagnosis of tarsal tunnel. Due to the high number of false negatives, one should not exclude the diagnosis of tarsal tunnel syndrome in the face of convincing clinical evidence.[5,6,11]

More recently, Dellon has advocated QST (quantitative sensory testing) for the diagnosis of peripheral nerve entrapment. It is claimed to offer earlier recognition and diagnosis of peripheral nerve entrapment. Other advantages touted is the ease of performance, interpretation and reduced pain for the patient.[23]

TREATMENT

Conservative treatment may initially be tried. In cases of varus or valgus deformities, orthoses may be of value. If synovitis is identified as the cause then course of NSAID's or a local corticosteroid injection may prove beneficial. In most cases however, surgical treatment is necessary.[11]

When surgery is indicated, the technique described by Lam is most often utilized.[3] He described a curvilinear incision over the tarsal tunnel. The flexor retinaculum is released and the posterior tibial nerve is identified. Its terminal branches are then traced. Any areas of entrapment are released and any masses are resected. Prior to closure, if a tourniquet has been used it is released to ensure adequate post operative hemostasis has been obtained. The skin is closed without repair of the deep fascia. The patient is placed in a sort leg cast or posterior splint and kept non weight bearing with progression to partial weight bearing over the next 2-3 weeks.

Results of surgical intervention had been varied. Cases with identifiable lesions respond better than idiopathic or traumatic causes.[12,18,24] In cases of a double crush, both lesions need to be addressed.[18] Early intervention also appears to improve the outcome.[12]

Since it has been shown that cases with identifiable lesions respond better, obtaining an MRI prior to any surgical intervention can help one find an identifiable lesion if it does exist. An MRI can also aid in the surgical planning due to the detailed imaging it gives.

CASE PRESENTATION

A 36-year-old white male presented with classic symptoms for tarsal tunnel syndrome. He had seen a neurologist and NCV/EMG studies were indicative of tarsal tunnel syndrome. His neurologist referred him to a podiatrist for further treatment. After a conservative modalities were exhausted and the patient did not obtain any relief, he was referred for surgical release.

When the patient presented to this office, he complained of right foot numbness on the plantar aspect. He described it as a burning pain and related it could be reproduced with pressure to the lower leg. He pointed to an area proximal to his medial malleolus. He related symptoms for about two years.

Exam revealed a slightly cavus foot structure with no gross or obvious orthopedic deformities. His vascular status was intact. On neural exam, he did have pain on direct palpation to the tarsal tunnel area and there was distal tingling on percussion, but there was no specific edema noted and no masses were palpable proximal to his tarsal tunnel area where he indicated. There was also no weaknesses noted in the posterior tibial muscle, the flexor digitorum longus or the flexor hallucis longus and no pain was noted on manual muscle testing.

His NCV studies were reviewed and it did reveal some latency in the right lower extremity. Previous MRI's of his lower back were unremarkable. Since the patient had symptoms consistent with tarsal tunnel syndrome as well as indicative nerve studies, the decision was reached to consider surgical release.

Prior to surgery an MRI was ordered to rule out any other specific underlying etiology for his tarsal tunnel syndrome. The MRI was performed with a 0.2 Tesla open system, coronal and sagittal T-1 weighted images were followed by inversion recovery coronal T-1 mixed and T-2 weighted images. On this MRI the osseus structures appeared intact except for a small amount of sclerotic changes about the talonavicular and subtalar joints. Evaluation of the region of the tarsal tunnel showed evidence for one to two dilated veins (Figure 1A). There was no evidence for ganglion cyst, edema, synovial

hypotrophy, tarsal coalition, or developmental muscle abnormalities in the area. However, there was a small amount of fluid demonstrated in the region of the flexor digitorum longus. The presence of the dilated veins and fluid along the flexor digitorum longus was presumed to be the etiology of his tarsal tunnel symptoms and it was felt that he would benefit from a release if further treatments failed. After only temporary relief from nonsteroidal anti-inflammatories, oral steroid therapy, and orthotics, he wished to proceed with surgery.

The patient was taken to the operating room and under general anesthesia with a pneumonic thigh cuff for hemostasis, a tarsal tunnel release was performed. The dilated veins were visible and were ligated and excised. The flexor retinaculum was released as well as surrounding fascial tissue at the entrance to porta pedis. His incision was closed and he was managed postoperatively in a non-weightbearing posterior splint for two weeks. He was then progressed to a fracture walker. His postoperative course was unremarkable.

At one month after his surgery, he reported occasional shooting pain but was much better than before surgery. He was allowed to return to work and gradually resume normal activities.

The patient continued to improve over the next one to two months but then his original pain and symptoms started returning. At five months postop, he presented to the office with complaints of hallux pain. He related that his pain was better through the tarsal tunnel area but when he tried to plantarflex his hallux, pain radiated proximally. He again pointed to the area proximal to his medial malleolus and related this pain had become more intense. Palpation to this area caused distal dysthesias through the tarsal tunnel and to the plantar hallux. On active plantarflexion of his hallux he experienced fasciculations in the great toe.

At this point, a second MRI was ordered with instructions to review the area more proximal to the tarsal tunnel and hopefully identify the source of this pain and fasciculations. A second MRI was performed with a 0.3 open Tesla unit. The lower extremity was examined in sagittal, axial and coronal planes of T-2 protein density and T-1 weighted contrast. A marker was used to help identify the region of his pain.

This more proximal MRI revealed a mass measuring 10 x 12 x 14 mm. It lay approximately 8.5 cm proximal to the talotibular articulation (Figure 1B). It demonstrated sharp margins and appeared isointense to muscle on the T-1 ranges and hyperintense on the T-2 images. Some complex material could be appreciated within the contents of the mass. The mass lay along the flexor hallucis longus tendon and within the flexor hallucis longus muscle.

Since the mass touched a tendon surface, an MRI contrast study was recommended to further assess. The reason was to see if the internal material enhanced which would indicate a possible synovial sarcoma.

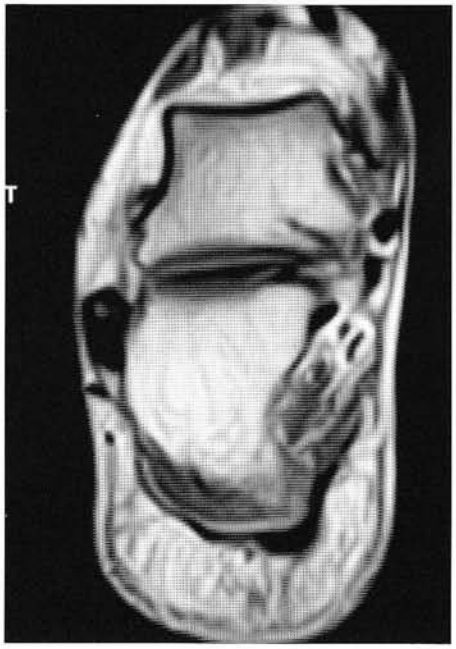

Figure 1A. Coronal view showing the dilated veins through the tarsal tunnel.

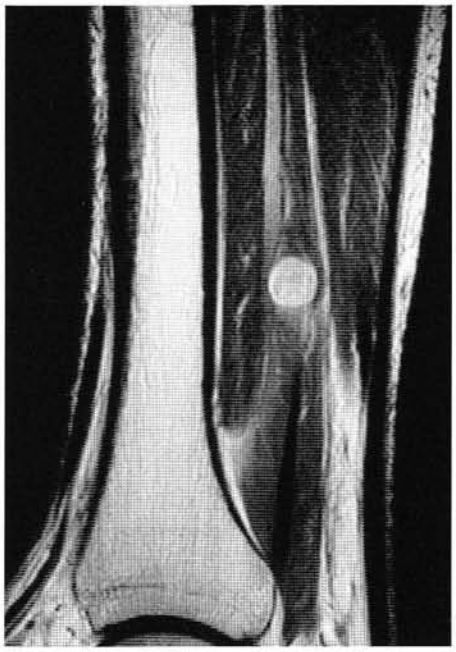

Figure 1B. Sagittal view showing the mass responsible for his symptoms.

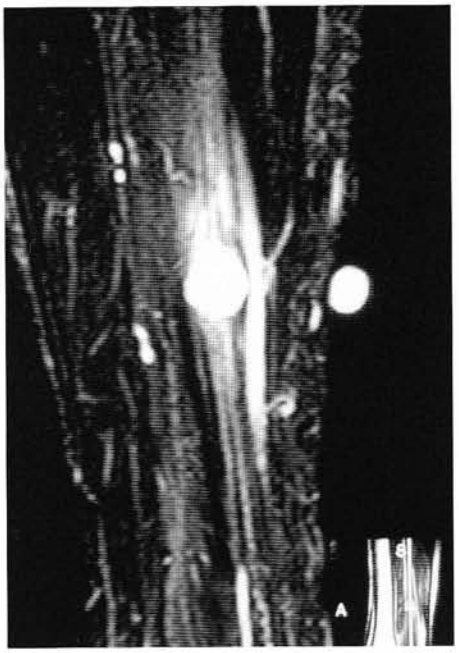

Figure 1C. Gadolinium enhanced sagittal view showing the same mass.

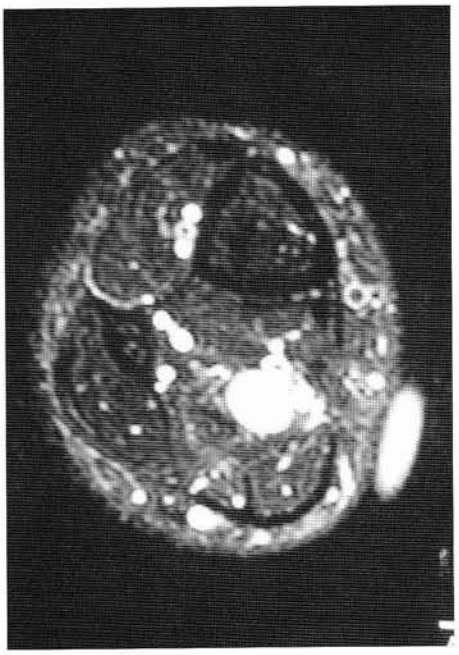

Figure 1D. Gadolinium enhanced transverse view showing the same mass.

A third MRI was then performed on the patient's right lower extremity with gadolinium contrast administered, T-1 weighted fat suppressed axial, coronal and sagittal images were obtained as well as non-fat suppressed T-1 weighted sagittal images through the distal calf. The MRI also noted the lesion along the flexor hallucis longus. The lesion did indeed show contrast enhancement (Figures 1C, 1D). Though this enhancement was histologically nonspecific, it did indicate that the lesion was a vascularized soft tissue mass. Though a benign tumor such as a neurofibroma was possible, sarcoma was included in a differential diagnosis and careful excision on biopsy was recommended.

The patient was then taken back to the operating room and under general anesthesia with a pneumonic thigh cuff, attention was directed to his medial calf at the level of the mass 8.5 cm above the ankle joint. Dissection was carried down through the superficial fascial muscle areas to the posterior tibial nerve and flexor hallucis longus. A firm palpable mass was identified (Figures 1E, 1F). However, this was noted to be within the nerve sheath and not adhered to the flexor hallucis longus muscle and tendon. It was resting in the flexor hallucis longus but was separated by a neural sheath. Loop dissection was then utilized to remove the mass and it was within the sheath and outside the nerve fibers so nerve fibers were not disrupted during the surgery (Figure 1G). The incision was closed in layers and the patient was released again non-weightbearing.

Histological exam did reveal a neuroschwanoma and he presented to the office six days postoperative with no complaints other than swelling through his calf. He returned one week after that and reported 99 percent improvement. He relates his foot felt better than it had in years and he, in his own words, was cured. The patient was allowed to resume normal activities as tolerated and at five months post excision of the neuroschanoma he was still doing well with no recurrence of symptoms.

CONCLUSION

The preceding case is a good example of the double crush syndrome with its two identifiable insults on the tibial nerve. Two known causes of tarsal tunnel syndrome were seen with magnetic resonance imaging. Although a wider field of view in the original MRI may have prevented a second surgery, this case does show the importance in this valuable tool.

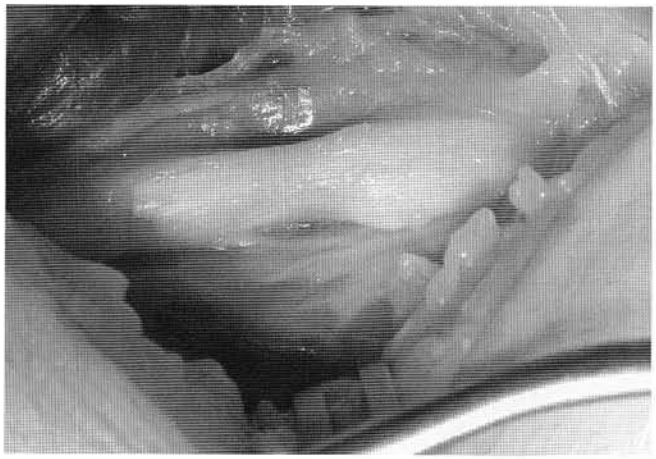

Figure 1E. Intraoperative view of the neural tumor.

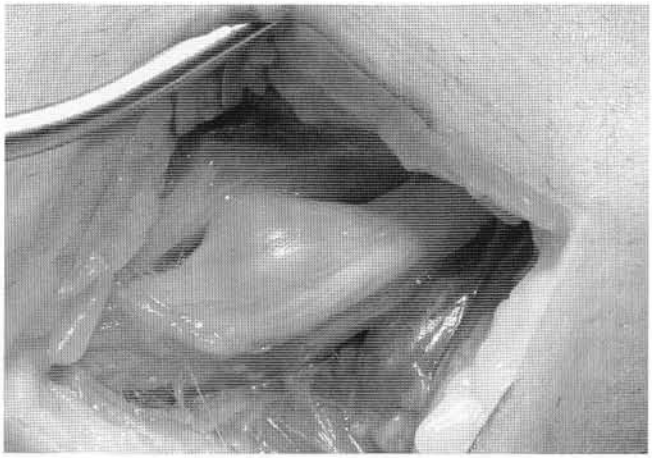

Figure 1F. Intraoperative view of the same tumor exposed from under the perineural sheath.

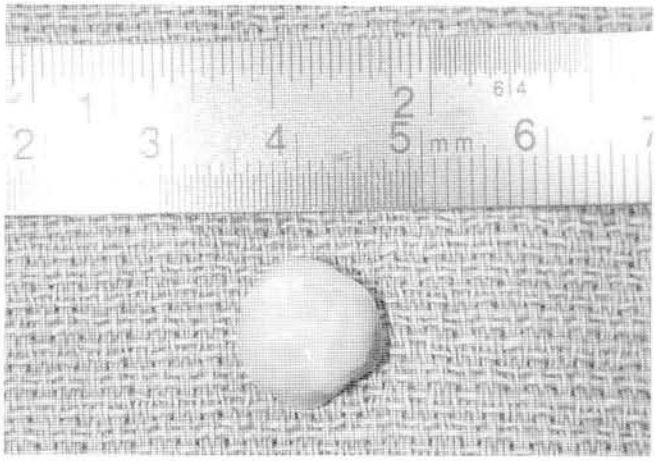

Figure 1G. Neurschwanoma. Note the well encapsulated appearance of the tumor.

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