

ATYPICAL TARSAI TUNNEL SYNDROME

Aimee A. Nichols, DPM

Craig A. Camasta, DPM

Perry H. Julien, DPM

Lee Gray, MS, DiPOD

Classical tarsal tunnel syndrome is described as an entrapment neuropathy of the posterior tibial nerve or one of its branches as it crosses the medial retinaculum of the ankle.¹ In tarsal tunnel syndrome, the posterior tibial nerve, the medial and lateral plantar branches, or the calcaneal branch may be entrapped beneath the flexor retinaculum, the fibroosseous tunnel, or the deep fascia along the medial border of the foot. This phenomenon usually results in symptoms of burning, aching, radiating pain along the nerve or nerve branch distribution. To an otherwise healthy, active person, this can be a debilitating problem. Electrodiagnostic testing which identifies prolonged sensory or motor distal latencies can be helpful in making the diagnosis of tarsal tunnel compression. Percussion of the tibial nerve within the tarsal tunnel can reproduce symptoms, the so-called Tinel's sign. A specific cause of tarsal tunnel syndrome can be identified in 60-80% of patients.² Of these diagnosed causes, trauma is the most common. Many other causes have been cited including space occupying lesions, foot biomechanics and position, tarsal coalition, inflammation of the nerve, tenosynovitis, fibrosis, accessory musculature, varicosities and idiopathic causes. Often the diagnosis of tarsal tunnel syndrome is made without identifying the causative factor. Conservative therapy consisting of injections and pain control measures are commonly employed with only palliative efforts. Surgical release of the tarsal tunnel is often utilized alone without addressing the direct causative factor as well. This may lead to variable results or limited success. The purpose of this paper is to present some of the less common causes of tarsal tunnel syndrome and to give the reader some additional insight into other possible etiologies of this condition.

The tarsal tunnel is located posterior to the medial malleolus and is bordered inferiorly by the medial surface of the talus, the sustentaculum tali and the lateral wall of the calcaneus. The flexor retinaculum, or the lacinate ligament, forms the roof of the tarsal tunnel. The flexor retinaculum originates from the medial malleolus and inserts into the posterior inferior aspect of the calcaneal tuberosity.¹ The tarsal tunnel contains the posterior tibial,

flexor digitorum longus and flexor hallucis longus tendons and the tibial nerve and posterior tibial artery. The medial and lateral plantar nerves are the terminal branches of the tibial nerve and this bifurcation occurs within the tarsal tunnel the majority of the time. The fibroosseous space within the tarsal tunnel is finite, and any decrease in this space or relative increase in volume will cause compression of the nerve or its branches.

Isolated fractures of the sustentaculum tali are rare injuries making up less than 15% of all calcaneal fractures.³ The sustentaculum tali comprises the plantar half of the middle facet of the subtalar joint. The deltoid ligament attaches superiorly to the sustentaculum and the flexor hallucis longus tendon courses under the sustentaculum in the groove for this tendon. Due to this strong ligamentous attachment, the sustentaculum rarely migrates in fracture situations. If a fracture of the sustentaculum does occur, it can heal uneventfully or with hypertrophic bone formation. Hypertrophy of bone can also occur at this level secondary to subtalar joint arthritis from previous injury or systemic disease. Regardless of the cause, this hypertrophic bone can cause compression and or irritation of the tibial nerve. In 1995, Myerson and Berger⁴ reported on a patient who sustained an isolated sustentacular fracture that formed a stable non-union. The patient presented with long-standing medial ankle and foot pain with burning and paresthesia over the medial foot, hallux and second digit. Upon CT examination, the patient had a fracture of the sustentaculum tali with migration of the fracture fragment medially and superiorly with compression on the tibial nerve. The patient was treated with surgical removal of the fracture fragment and progressed back to normal activities without symptoms.

Os trigonum syndrome was originally described in 1804 by Rosenmuller as an accessory bone located posterior to the posterolateral tubercle of the talus.⁵ When this posterolateral process is enlarged, it is termed a Steida's process.⁶ This enlarged bone may be completely asymptomatic without causing any posterior ankle joint pain. The incidence of the presence of this bone ranges

from 2.5 to 13%.¹ The flexor hallucis longus tendon courses adjacent to this process and fracture or enlargement of the posterolateral tubercle of the talus can cause chronic inflammation or stenosing tenosynovitis of the FHL. This injury to the tendon can result in a relative increase in the size of the tendon due to edema and fibrosis. This in turn will cause an increase in the volume of the tarsal tunnel which can compress upon the tibial nerve or its branches.

Tendo Achilles lengthening procedures are commonly performed to reduce the amount of ankle joint equinus that is present. This procedure may be performed through an open procedure in which the tendon is incised in either the sagittal or frontal planes or through a percutaneous type of approach. In rare cases, damage to the tibial nerve or its branches can occur due to inaccurate incision placement or dissection techniques.

CASE 1: SUBTALAR JOINT ARTHROSIS AS A CAUSE OF TARSAL TUNNEL SYNDROME

A 19-year-old female presented with chronic pain to her right foot and ankle for 2 years following an inversion ankle injury while playing softball. She was seen at a local emergency room with failure to diagnose any significant injury. She had several local cortisone injections without any relief of symptoms. Pain increased with activity and with length of standing. There was no significant medical history. Her pain was localized to the medial aspect of the right foot and ankle with shooting, stabbing, radiating pain from the medial ankle to the medial arch area.

Hypertrophy of the sustentaculum tali and middle facet of the subtalar joint were palpated upon exam. Pain was reproducible with minimal compression to this area. Epicritic sensation was intact to all areas of the foot.



Figure 1A. Note the presence of osteophytic lipping on the lateral and posterior aspect of the subtalar joint.

Subtalar joint range of motion was somewhat limited but without crepitation. Nerve conduction velocity testing was performed which showed a prolonged latency of the lateral plantar nerve of 4.8ms compared to the medial plantar nerve, consistent with tarsal tunnel syndrome. Radiographic examination showed obvious degenerative joint disease of the subtalar joint with osteophytic lipping of the posterior portion of the joint and along the middle facet (Figure 1). There was joint effusion present and soft tissue impingement along the hypertrophied middle facet of the medial portion of the subtalar joint (Figure 2).

The patient underwent a subtalar joint arthrodesis along with a tarsal tunnel release. A lateral incision was utilized for resection of the subtalar joint. Significant arthrosis was noted within the posterior facet of the subtalar joint. After joint resection was performed, attention was directed medially where a 4 cm curvilinear incision was created just distal and posterior to the medial malleolus, overlying the bony prominence of the middle facet. The tibial nerve was identified and appeared to have a thickened appearance. The nerve was mobilized and retracted. The bony prominence of the middle facet was resected and rasped until an anatomic appearance was obtained. The cartilaginous surface of the middle facet was resected. A 6.5 mm partially threaded cancellous screw was placed from the dorsal talar neck to the calcaneal tuberosity in standard AO technique.

Appropriate dressings were applied and the patient was placed in a below-knee cast, non-weight bearing for 7 weeks. The patient was seen postoperatively at weeks one and seven with no complaints of pain or complications. At week eight, the patient began weight-bearing and stated the sensation to the plantar aspect of her foot was increased from her pre-surgical status. Eight months following surgery, the patient had no complaints of neuritic or arthritic pain at the subtalar joint or medial ankle levels.



Figure 1B. AP radiograph demonstrating hypertrophy and lipping of the middle facet of the subtalar joint. This is the area impinging upon the tarsal tunnel.



Figure 2A. Os trigonum presence on the posterior talus causing inflammation of the long flexor tendon.

CASE 2: OS TRIGONUM AS AN ETIOLOGY OF TARSAI TUNNEL SYNDROME

An 18-year-old collegiate female soccer player presented with a several month history of lateral right ankle instability with medial ankle pain extending to the arch of her foot. This affected her ability to be competitive with soccer. The patient failed conservative therapy consisting of bracing and medication. Upon exam, she demonstrated significant lateral ankle instability, with a positive anterior drawer and talar tilt test, as well as a positive Tinel's sign. Upon radiographic examination, there was the presence of an os trigonum but no other obvious osseous abnormalities (Figure 3). MRI evaluation revealed edema and inflammation of the flexor hallucis longus tendon sheath (Figure 4).

The patient underwent a delayed primary repair of the lateral ankle ligaments, os trigonum excision, and a tarsal tunnel release. The lateral ligaments were augmented through a lateral incision. The os trigonum and the tarsal tunnel were both approached from a medial 7 cm incision following the flexor hallucis longus tendon sheath. The lacinate ligament was incised and the neurovascular structures could be visualized with no apparent pathology. These structures were carefully retracted throughout the remainder of the procedure. The flexor hallucis longus tendon was followed posteriorly and did not have any apparent degeneration present. A significant amount of brownish synovial fluid was noted upon dissection. The prominent os trigonum was easily visualized through the wound and was resected utilizing a rongeur and hand rasp. Attention was then directed distally and the fascial bands surrounding the abductor hallucis muscle belly were



Figure 2B. Fluid in the flexor hallucis longus tendon sheath caused by the inflammatory irritation of the hypertrophies os trigonum.

released. Standard closure was performed and the patient was placed in a below-knee non-weight bearing cast. The patient had an uneventful post-operative course. The cast was removed at 7 weeks and she was placed in a walking brace with protected weight bearing. At nine weeks the patient was asymptomatic and was permitted to begin cycling. Twenty weeks following surgery the patient was back to full activity playing soccer.

CASE 3: TARSAI TUNNEL SYNDROME AS A RESULT OF A TENDO ACHILLES LENGTHENING PROCEDURE

A 60-year-old female sustained a posterior leg injury in 1994 while running. She related a sensation of her leg "ripping" and was diagnosed initially with a partial gastrocnemius tear and was placed in a plaster cast. After 8 weeks of continued pain, the patient was further diagnosed with a partial Achilles tendon tear and sent to a general surgeon. The patient related that this surgeon "split the tendon sheath" to repair the tendon. The patient noted immediate and severe post-operative pain to her medial heel and foot. She indicated a central posterior area of severe pain and a radiating area surrounding it (Figure 3A). The patient was unable to even minimally dorsiflex or plantarflex her foot without excruciating pain. The patient related no improvement in pain throughout the next few weeks of her recovery. Over the next few months, the

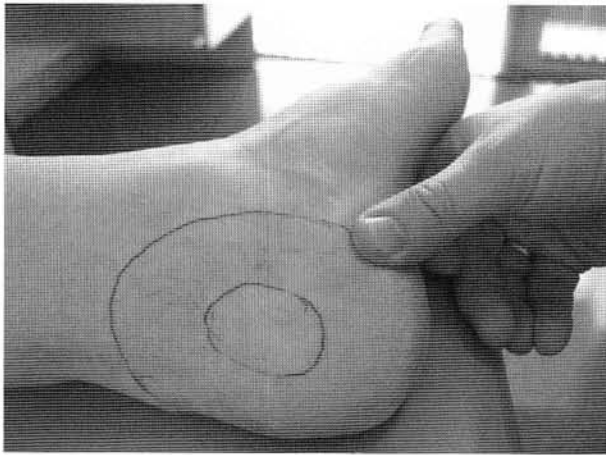


Figure 3A. Central area of excruciating, neuritic pain with surrounding region of radiating pain. Pain reproducible at the medial plantar surface of the calcaneus radiating to the heel and medial plantar arch.



Figure 3B. Well healed appearance of the incision site. The patient is now able to fully dorsiflex her ankle without discomfort.

patient underwent a series of exploratory surgeries and sympathetic nerve blocks with minimal relief. She also endured radiation therapy to her Achilles tendon also without success. She noticed that her foot was beginning to “turn in” and her Achilles tendon was contracting and shortening. The patient underwent a tendo Achilles lengthening procedure and noted that her walking and foot position improved but the pain was increasing to her heel and medial foot. She was next referred to a neurosurgeon who diagnosed damage to her calcaneal branch of the tibial nerve. The patient subsequently underwent a tarsal tunnel release with external neurolysis of the calcaneal branch of the tibial nerve (Figure 3B). At 6 weeks postoperative, the patient was completely pain free and able to dorsiflex and plantarflex her foot fully without symptomatology.

CONCLUSION

In summary, tarsal tunnel syndrome can be caused by a wide variety of conditions and pathology. These causes can be deduced by a careful lower extremity exam and thorough history taking. In addition to standard radiographs, MRI studies can be helpful in evaluating the volume of the tarsal tunnel and locating any sources of inflammation. The tarsal tunnel may not be the only area that needs to be surgically addressed to return the patient to an asymptomatic state without activity limitation.

REFERENCES

1. Downey MS, Sorrento DL. Tarsal tunnel syndromes. In: McGlamry ED, Banks AS, Downey MS, eds. *Comprehensive textbook of foot surgery*, 3rd ed. Baltimore: Williams & Wilkins; 2001. p. 1266-78.
2. Lau JTC, Daniels TR. Tarsal tunnel syndrome: a review of the literature. *Foot Ankle Int* 1999;20:201-9.
3. DeLee JC. Fractures and dislocations of the foot. In: Mann RA, Coughlin MJ, editors. *Surgery of the foot and ankle*, 6th Ed. St. Louis: Mosby-Year Book; 1993. p.1465-703.
4. Myerson MS, Berger BI. Nonunion and fracture of the sustentaculum tali causing a tarsal tunnel syndrome: a case report. *Foot Ankle Int* 1995;16:740-2.
5. Rosenmuller P. (quoted in Holland CT): On rarer ossifications seen during x-ray examinations. *J Anat* 1921;55:235.
6. Shepherd FJ. A hitherto undescribed fracture of the astragalus. *J Anat Physiol* 1882;17:79.

ADDITIONAL REFERENCES

- Burks JB, DeHeer PA. Tarsal tunnel syndrome secondary to an accessory muscle: A case report. *J Foot Ankle Surg* 2001;40:401-3.
- Gordon SL, Matheson DW. The accessory soleus. *Clin Orthop* 1976;97:127-32.
- Hecker P. Study on the peroneus of the tarsus: preliminary notes. *Anat Rec* 1923;26:79-82.
- Perkins JD. An anomalous muscle of the leg: peroneocalcaneus internus. *Anat Rec* 1914;21-5.