

# Hypertrophic Sustentaculum Tali Causing a Tarsal Tunnel Syndrome: A Case Report

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*A case report of tarsal tunnel syndrome caused by a hypertrophic sustentaculum tali is presented. This is the first reported case secondary to this etiology. Complete resolution of the patient's symptoms has been obtained through resection of the hypertrophic anatomy. The authors also discuss possible etiologies of tarsal tunnel syndrome. (The Journal of Foot & Ankle Surgery 40(2):110-112, 2001)*

Key words: hypertrophic sustentaculum tali, tarsal tunnel syndrome

**T**arsal tunnel syndrome (TTS) is used to describe an entrapment neuropathy of the tibial nerve or one of its branches within the tarsal tunnel. Keck and Lam independently described this condition in 1962 (1, 2). Usual clinical symptoms include pain, paresthesias, and/or numbness and tingling along the medial aspect of the ankle and plantar foot (3). Various space-occupying lesions, metabolic abnormalities, trauma, systemic disease states, biomechanical abnormalities, ankle and foot deformities, and idiopathic causes are known to cause TTS (1-6).

Diagnosis of tarsal tunnel syndrome is often difficult due to the commonly vague and diffuse symptoms. Common differential diagnoses include: plantar fasciitis, heel spur syndrome, peripheral vascular disease, interdigital neuroma, longitudinal arch sprain, sciatica, metatarsalgia, rheumatologic diseases, peripheral neuritis, or drug toxicity (6). Therefore, a thorough history and physical exam, including various imaging techniques, are used to establish the diagnosis of TTS. Electrodiagnostic studies may help support the diagnosis in up to 80% of cases (3).

In our case, a hypertrophic sustentaculum tali (HST), acting as an intrinsic space-occupying lesion, resulted in

decreased volume within the tarsal tunnel and compression on the posterior tibial nerve. We present the first reported case of TTS secondary to this anatomical cause.

## Case History

A 52-year-old male was referred by his family physician for treatment of recalcitrant, inferior heel pain. The presenting complaint was burning and numbness to the medial aspect of the right ankle and plantar foot for the past year. The pain was exacerbated by long periods of standing and was worse when first arising after long periods of rest. The patient's job required standing on hard floors. Following long periods of weightbearing, the patient walked with a limp because of pain.

Prior treatment included steroid injections, strappings, over-the-counter arch supports, and physical therapy. Failure of these modalities prompted referral to the authors.

His past medical history was noncontributory. Vascular examination of the lower extremity was normal. There was no edema or erythema medially or plantarly. Sharp/dull and vibratory sensations were intact. Percussion of the tibial nerve at the tarsal tunnel elicited pain and numbness at the medial ankle and medial plantar right foot. An antalgic gait was present. Posterior tibial tendon strength was normal. Ankle dorsiflexion of the right foot was 0°, and 10° on the uninvolved side. Range of motion of the subtalar and midtarsal joints was normal. Pain was noted in the right calf and plantar fascia with initiation of the windlass mechanism. Palpation of the medial band of the fascia elicited pain at the right plantar medial heel. Initial radiographs revealed an enlarged sustentaculum tali (Fig. 1) and a well-defined plantar calcaneal spur. Differential diagnosis included heel spur secondary to

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**FIGURE 1** A hypertrophic sustentaculum tali is first identified on this Harris-Beath view.

plantar fasciitis, entrapment or neuroma of the medial plantar nerve, and tarsal tunnel syndrome of the right foot.

Initial treatment included strapping, stretching, and rest. At 1-week follow-up, the patient complained of continued pain and burning to the medial ankle and plantar right foot. A 1-month course of diclofenac sodium with misoprostol gave the patient temporary relief; however, the original symptoms returned, in spite of continued use of the medications. A diagnostic nerve block to the tarsal tunnel was performed with marcaine and provided substantial relief for 2 days. The patient was then sent for an EMG study, which came back as normal. The patient was placed in a below-the-knee cast for 1 month. While he was in the cast, the symptoms were significantly decreased. Soon after removal, the patient experienced pain in the medial ankle and plantar foot and had pain with eversion of the foot.

An MRI reconfirmed that there was a hypertrophic sustentaculum tali (Fig. 2). There were no other pathologic findings in the tarsal tunnel. It was felt that the HST was acting as a space-occupying lesion and causing compression on the posterior tibial (PT) nerve. Therefore,



**FIGURE 2** MRI confirms the presence of a hypertrophic sustentaculum tali, with the neurovascular bundle in close contact.

the patient was scheduled for surgery to debride the hypertrophic sustentaculum tali, which would decompress the PT nerve.

The surgical approach was made through a 12-cm retromalleolar incision overlying the course of the tibial nerve at the level of the sustentaculum tali. Dissection was carried down to the compartment of the tarsal tunnel that houses the posterior tibial artery, nerve, and vein. The PT nerve was identified and traced distally through the tarsal tunnel. Upon dissection, it was noted that there was pressure placed on the posterior tibial nerve by the sustentaculum tali, resulting in a deviation through its course in the tarsal tunnel. Clinically, there was no hypertrophy or other abnormality to the PT nerve itself.

The posterior one third of the sustentaculum tali was resected. No other pathologic changes were found. A stirrup style splint was applied following routine closure. The patient was instructed to be nonweightbearing on the affected extremity for 3 weeks postoperatively. Range-of-motion exercises were initiated twice a day, on postoperative day 18. He returned to normal activities during postoperative week 4 with only mild pain to the inferior heel. In the 7th postoperative week, he had returned to work with resolution of all preoperative symptoms. At 13

months after surgery, the patient remained pain free and was enjoying his normal activities.

## Discussion

The exact etiology of tarsal tunnel syndrome is often difficult to ascertain due to the intimate relationship of the anatomical structures within the tarsal tunnel. Therefore, clinical and anatomical considerations are of the utmost importance (3, 5, 6). The diagnosis is often difficult due to concomitant pathologies that mimic some of the clinical signs and symptoms. Therefore, all possible etiologies should be explored before a final diagnosis is made.

Due to the vague and diffuse symptoms of our patient, it was difficult to determine whether he was suffering from tarsal tunnel or heel spur syndrome. In plantar fasciitis, there is usually more heel pain that is localized to the origin of the plantar fascia. However, TTS usually presents with the feeling of burning, tingling, and numbness at the medial aspect of the heel and plantar arch (1–6), and was present in our patient. Also, with plantar fasciitis the pain usually presents in the morning and may improve with stretching and exercise. In comparison, tarsal tunnel pain usually worsens with increased exercise or prolonged walking, as was the case with our patient. A positive Tinel's sign is commonly found in TTS and not HST. The diagnostic block relieved the symptoms and thus was helpful in diagnosing the TTS. Although EMG studies were normal, this does not rule out TTS, as they are usually found to be supportive, but not diagnostic (3).

Ruling out a coalition was also important in confirming our diagnosis, as this may also cause a TTS (1–6). Radiographs did not show any signs of a medial talocalcaneal bar, and this was confirmed intraoperatively. MR imaging can be a very valuable tool in helping to make the diagnosis of TTS, especially when a space-occupying mass or lesion is suspected (5, 6). Since the sustentaculum tali forms part of the superior border of the tarsal tunnel, it seems logical that an alteration of the normal anatomy of

this structure may cause TSS. There is one reported case of TTS caused by a fracture of the sustentaculum tali that migrated superiorly to cause tibial nerve impingement (4).

It is unclear to us why this patient exhibited symptoms rather late in life. We would have expected symptoms earlier in view of the anatomic abnormality. However, it may have presented late because of the minor but cumulative trauma to the nerve. We were able to identify the HST on MRI, although exact volumetric parameters are not yet established. In our case, the HST acted like an intrinsic space-occupying lesion, thus, decreasing the volume within the tarsal tunnel and causing compression of the posterior tibial nerve. Furthermore, with weight-bearing and ambulation, the posterior tibial nerve was tethered over this anatomic abnormality and may have increased the local irritation to the nerve. After bony debridement, the PT nerve was no longer in contact with the posterior surface of the HST. The patient's complete relief of symptoms following surgery supports HST as an etiologic factor for TTS.

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