



Case Report

Tarsal Tunnel Syndrome Secondary to Accessory Musculature

A Case Report

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Abstract: Tarsal tunnel syndrome (TTS) is a relatively uncommon compression neuropathy caused by impingement of the tibial nerve or one of the terminal branches. The presence of accessory musculature at the posteromedial aspect of the ankle has been identified as a rare cause of this condition. Despite the rarity of this condition, it must be considered in patients with refractory symptoms consistent with tibial nerve dysfunction. The accurate diagnosis of this condition relies heavily on a detailed history and physical examination, adequate imaging read by both surgeon and trained musculoskeletal radiologist, as well as a high level of suspicion for such pathology. In this case report, we describe a 46-year-old male with history, examination, and imaging all consistent with TTS secondary to accessory musculature. Following excision of an accessory soleus and flexor digitorum accessorius longus, as well as simultaneous tarsal tunnel release, the patient experienced full resolution of his symptoms. This highlights the importance of

considering accessory musculature as a potential cause of TTS in patients presenting with tibial compression neuropathy.

Levels of Evidence: Level V: Case Report

Keywords: tarsal tunnel syndrome; forefoot; toe; midfoot; nerve compression syndromes; neurological problems; foot surgery techniques; diagnostic and therapeutic techniques; MRI diagnoses

Tarsal tunnel syndrome (TTS) is a relatively uncommon compression neuropathy caused by impingement of the tibial nerve or one of the terminal branches. There are multiple etiologies for TTS, including both intrinsic and extrinsic causes of compression of the tibial nerve.¹ Intrinsic causes include space occupying lesions leading to compression of the contents

within the tarsal tunnel. This may include ganglion cysts, lipoma or other tumor, perineural fibrosis, varicosities or other vascular malformations, and prominent medial osteophytes. Extrinsic causes leading to external compression on the tarsal tunnel include prominent shoe wear, previous trauma, tarsal coalition or flatfoot deformity leading to hindfoot

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valgus, postoperative scarring, edema of the lower extremity, systemic inflammatory disease, and rarely, accessory musculature at the posteromedial aspect of the leg.

The tarsal tunnel is an anatomic structure bordered by the talus and calcaneus medially, the abductor hallucis inferiorly, and the roof formed by the flexor retinaculum.² The contents of the tarsal tunnel include the tendons of the

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tibialis posterior, flexor digitorum longus, and flexor hallucis longus, in addition to the posterior tibial nerve, artery, and vein. The tibial nerve has 3 terminal branches, including the medial plantar, lateral plantar, and medial calcaneal nerves. Compression of the tibial nerve or any one of these terminal branches may lead to a constellation of symptoms consistent with compression neuropathy.

Patients presenting with pain secondary to compression of the tibial nerve will often report pain with prolonged standing or walking, medial or plantar foot pain at rest, and burning, numbness, or tingling along the plantar medial aspect of the foot.^{3,4} Patients may also complain of swelling at the posteromedial ankle. Physical examination may reveal tenderness to palpation at the posteromedial ankle along the tarsal tunnel, as well as a positive Tinel's sign over the tibial nerve. One may also experience pain or numbness during compression of the tarsal tunnel. Other less common physical examination findings have been described, including hypoesthesia and clawing of the toes.³

Workup for TTS should first include weightbearing radiographs to evaluate the surrounding osseous structures and overall foot alignment. Advanced imaging may include ultrasound to evaluate for space occupying lesions within the tarsal tunnel, or more commonly, magnetic resonance imaging (MRI) of the hindfoot to further evaluate both extrinsic and intrinsic causes of compression. Electromyographic (EMG) studies may also be obtained to evaluate for neurologic compression and involvement, although it should be noted a negative EMG does not rule out the presence of TTS. Positive EMG findings may include delayed nerve conduction and decreased amplitude of motor action potentials of the intrinsic muscles of the foot.

Following accurate diagnosis of TTS, there are various nonoperative modalities that have proven to be effective for symptom relief. These include activity modification, anti-inflammatories, inserts with a mild arch support, and physical

therapy, including targeted stretching exercises. Medications proven effective in the setting of neuritis or neuropathy such as gabapentin may also be of benefit.³ If conservative management fails and the patient elects to proceed with surgery, the surgical plan largely depends on the etiology of the compression neuropathy.

In this case report, we present a case of TTS secondary to extrinsic compression of the tarsal tunnel due to the presence of accessory musculature. Historically, accessory musculature has been felt to be a rare cause of compression tibial neuropathy. In 1975, Nathan et al⁵ discussed the likelihood of accessory musculature as a cause of TTS being overshadowed by more apparent causes of TTS due to the relative rarity and lack of awareness of accessory anatomy. However, a growing number of recent case reports implicating accessory anatomy as the etiology for compression at the tarsal tunnel suggests this may not be as rare as previously thought. Reasons for growing awareness and reporting may include increased implementation of MRI in routine diagnosis and preoperative planning, increased surveillance by both radiologists and orthopedists for accessory muscles as a cause of TTS, or a combination of the above.

Case Report

History of Present Illness

The patient is an active 46-year-old male who presented to clinic with worsening exertional right ankle pain, which had progressed over the past 4 years. He complained of pain at the posteromedial aspect of the hindfoot that progressed during activity and was relieved with rest. He also described associated numbness and tingling along the medial ankle and plantar aspect of his foot that became more severe with extended periods of activity.

Physical Examination

On examination, he had neutral standing alignment through his right hindfoot and midfoot. His symptoms

were mildly aggravated with dorsiflexion and eversion of the ankle. There was also a positive Tinel's sign with tapping along the tarsal tunnel over the tibial nerve. This recreated his posteromedial ankle pain and also elicited numbness and tingling along the plantar aspect of his foot. With his foot held in plantarflexion and inversion, a compression test at the tarsal tunnel was positive, and aggravated his posteromedial ankle pain, as well as tingling, which radiated to the plantar aspect of his foot.

Advanced Imaging/Other Studies

An MRI of his right hindfoot was obtained to further evaluate the etiology of his pain. The MRI was reviewed by both the senior author as well as a trained musculoskeletal radiologist and revealed the presence of both an accessory soleus as well as a flexor digitorum accessorius longus muscle (Figure 1). An EMG of his right lower extremity was positive for tibial nerve compression at the level of the tarsal tunnel with prolonged distal motor and sensory latencies.

Treatment Decision Making

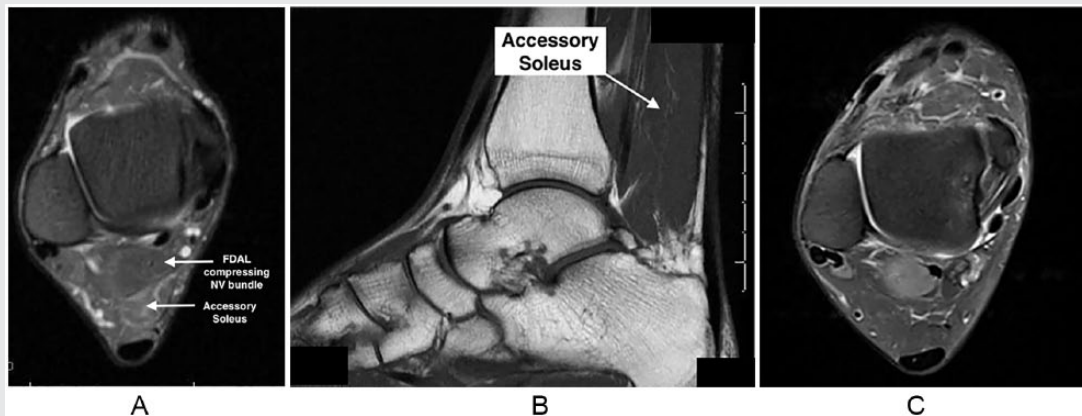
The patient first proceeded with conservative management, which included anti-inflammatories, brace wear, activity modification, and an extended course of physical therapy without any relief of his symptoms. Because of the prolonged presence and disabling nature of his pain, and lack of improvement with conservative modalities, the patient elected to proceed with surgical management.

Operative Technique

The patient was placed in the prone position with his operative extremity elevated to allow full access to the posteromedial aspect of the right ankle. A posteromedial approach to the right ankle was undertaken. The incision extended from just medial to the Achilles tendon proximally, down to the most distal aspect of the tarsal tunnel (Figure 2).

Figure 1.

Axial (A) and sagittal (B) magnetic resonance images revealing the presence of both an accessory soleus and a flexor digitorum accessorius longus (FDAL). (C) Axial MRI image of normal ankle without evidence of accessory musculature.

**Figure 2.**

The incision was carried just medial to the Achilles tendon distally along the tarsal tunnel.

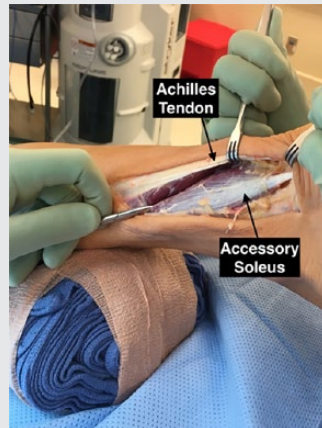


The flexor retinaculum, forming the roof of the tarsal tunnel, was identified and incised in line with the incision. This allowed full access to the contents of the tarsal tunnel.

At this point, the accessory soleus (AS) was easily identifiable deep to the Achilles tendon within the retrocalcaneal space (Figure 3). The AS was bluntly dissected and separated from surrounding soft tissue structures along its fascial plane. The AS was then followed distally to its insertion on the medial aspect of the calcaneus just

Figure 3.

The accessory soleus was easily identified within the retrocalcaneal space, deep to the Achilles tendon.

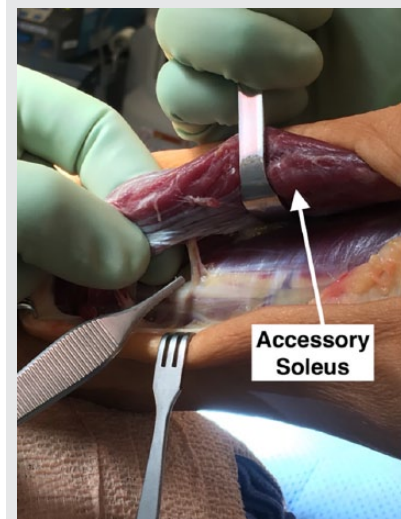


anterior to the Achilles insertion (Figure 4). The AS insertion was released sharply off the calcaneal tuberosity. Dissection was then carried proximally to the AS origin at the soleus muscle belly. The AS was dissected free from the soleus muscle belly and was removed from the field (Figure 5).

Following excision of the AS, the posterior tibial tendon and flexor digitorum longus were identified at the

Figure 4.

The accessory soleus was followed distally to its insertion on the medial aspect of the calcaneus just anterior to the Achilles insertion.



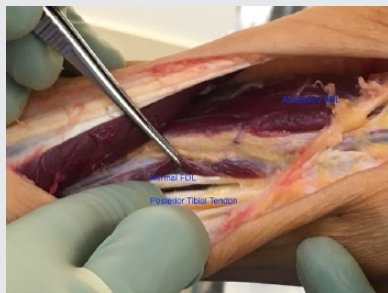
level of the Steida process of the talus (Figure 6). The neurovascular bundle including the posterior tibial artery and vein, and tibial nerve were identified within the tarsal tunnel, dissected free, and protected for the remainder of the case. With the neurovascular bundle

Figure 5.

Accessory soleus following excision.

**Figure 6.**

The flexor digitorum accessorius longus (FDAL) was located posterior to the normal flexor digitorum longus, creating pressure on the neurovascular bundle.



retracted anteriorly, the flexor hallucis longus (FHL) tendon and muscle belly were identified. An additional muscle belly was located posterior to the FHL. This was followed distally to the musculotendinous junction, and the additional muscle was identified as the flexor digitorum accessorius longus (FDAL). Distally, the medial calcaneal branch of the tibial nerve was identified and dissected free. There was obvious compression of the medial calcaneal nerve secondary to the space occupying FDAL. In similar fashion to the AS, the FDAL was bluntly dissected and released from surrounding soft tissue structures.

Figure 7.

Flexor digitorum accessorius Longus after being released from the medial aspect of the calcaneus.



After careful retraction and protection of the medial calcaneal nerve, the FDAL tendon was followed along its course to the insertion at the medial aspect of the calcaneus and was released sharply from its insertion (Figure 7). The FDAL was then followed proximally and released from its proximal origin at the flexor digitorum muscle belly. Following excision of the FDAL (Figure 8), the function of the FDL and FHL were confirmed to remain intact.

Postoperative Course

The patient was kept nonweightbearing for the first 3 weeks following surgery to allow time for his incision to heal. Once

Figure 8.

Flexor digitorum accessorius longus following excision.



his incision had healed and sutures were removed, he was allowed to advance his weightbearing to tolerance in a CAM boot, with light range of motion daily. Six weeks postoperatively he was started in physical therapy, slowly weaned out of the boot, and advanced his activities as pain and strength allowed. By 3 months postoperatively, he was doing well and back to full activity with complete relief of his symptoms.

Discussion

There are multiple studies which have evaluated the etiology of compression at the tarsal tunnel. Cimino conducted a literature review of 25 studies and 186

cases of TTS. Trauma was found to be the most common cause of TTS, involving 17% of the reported cases. Varicosities and heel varus followed closely thereafter, at 13% and 11%, respectively.¹ Similarly, Lau et al⁶ categorized TTS as having three primary etiologies: trauma, deformities of the foot, and space occupying lesions. Previously reported traumatic causes of TTS include missed fractures of the talus and calcaneus, which share the commonality of reducing the cross-sectional area within the tarsal tunnel.^{7,8} Foot deformities as described by Lau et al⁶ implicate abnormalities like hindfoot varus with a pronated forefoot causing TTS as this combination results in stretching and neuropraxia of the plantar nerve. Finally, space occupying lesions, both intrinsic and extrinsic to the tunnel, can contribute to TTS. Common lesions include the presence of tendon sheath ganglia or varicosities causing compression on the nerve, as well as accessory musculature.⁹

Accessory muscle entrapment neuropathy is a relatively unusual etiology of tarsal tunnel syndrome despite some studies citing as high as 16.3% of cases with this cause.¹⁰ This may, in part, be a result of poor surveillance on imaging or lack of awareness of accessory muscles as a cause of TTS. However, a growing body of case reports in recent literature suggests a change in TTS diagnosis and care.

Four accessory muscles have been described within the region of the ankle: AS, FDAL, peroneocalcanus internus, and tibio-calcaneus internus. In a 13-year study at a single institution, 41 patients (49 feet) were surgically treated for TTS and etiologic assessment yielded 12.2% secondary to FDAL and 4.1% secondary to AS. The authors concluded the prevalence of TTS as a result of accessory muscles to be 16.3%.¹⁰

The accessory soleus muscle has been described as one of the most common accessory muscles in the ankle.¹¹ However, true incidence of AS in the population is unclear. A 10-year study that reviewed 4771 MRIs of the ankle found 35 cases of AS, giving a

prevalence of 0.73%. The authors do note, however, that these conclusions were drawn from a symptomatic patient population.¹² Cadaveric studies have placed the prevalence as high as 5.5% in the general population. Other demographics include a higher prevalence in athletes as well as bilaterality in 15% of cases.¹³ There is an associated 2:1 male predominance appearing usually in the second or third decade of life and is most commonly associated with exertional pain.¹⁴ The incidence of FDAL was reported as 6% in one study that examined 100 asymptomatic individuals.¹⁵ A cadaveric study (n = 68) found FDAL present in 8% of the limbs.¹⁶ FDAL is also reported to be more common in men.¹³

To the authors' knowledge, there have yet to be any published literature on a patient with both FDAL and AS accessory muscles when presenting with TTS. If taken as independent variables, the likelihood of having both of these accessory muscles would be approximately 0.04%. There currently is no research to suggest that the presence of one accessory muscle predisposes one to a second accessory muscle.

In this case, adequate imaging and a high index of suspicion were vital in the identification of both accessory muscles. The AS and FDAL were identified on MRI by the primary surgeon in conjunction with a trained musculoskeletal radiologist. This highlights importance having a trained musculoskeletal radiologist available to aid in the identification and diagnosis of TTS secondary to the presence of accessory musculature.

Conclusions

We present a case of a 46-year-old male with 2 accessory muscles causing his TTS, which is, to date, unreported in the literature. This case emphasizes the importance of a high index of suspicion and careful evaluation of advanced imaging for proper diagnosis and treatment. In the treatment of TTS, we therefore recommend consideration of accessory muscles as a potential etiology.

Declaration of Conflicting Interests

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Ethical Approval

Not applicable.

Informed Consent

Not applicable.

Trial Registration

Not applicable.

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