

Reviews

An Update on Posterior Tarsal Tunnel Syndrome

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Posterior tarsal tunnel syndrome (PTTS) is an entrapment neuropathy due to compression of the tibial nerve or one of its terminal branches within the tarsal tunnel in the medial ankle. The tarsal tunnel is formed by the flexor retinaculum, while the floor is composed of the distal tibia, talus, and calcaneal bones. The tarsal tunnel contains a number of significant structures, including the tendons of 3 muscles as well as the posterior tibial artery, vein, and nerve. Focal compressive neuropathy of PTTS can originate from anything that physically restricts the volume of the tarsal tunnel. The variety of etiologies includes distinct movements of the foot, trauma, vascular disorders, soft tissue inflammation, diabetes mellitus, compression lesions, bony lesions, masses, lower extremity edema, and postoperative injury. Generally, compression of the posterior tibial nerve results in clinical findings consisting of numbness, burning, and painful paresthesia in the heel, medial ankle, and plantar surface of the foot. Diagnosis of PTTS can be made with the presence of a positive Tinel sign in combination with the physical symptoms of pain and numbness along the plantar and medial surfaces of the foot. Initially, patients are treated conservatively unless there are signs of muscle atrophy or motor nerve involvement. Conservative treatment includes activity modification, heat, cryotherapy, non-steroidal anti-inflammatory drugs, corticosteroid injections, opioids, GABA analog medications, tricyclic antidepressants, vitamin B-complex supplements, physical therapy, and custom orthotics. If PTTS is recalcitrant to conservative treatment, standard open surgical decompression of the flexor retinaculum is indicated. In recent years, a number of alternative minimally invasive treatment options have been investigated, but these studies have small sample sizes or were conducted on cadaveric models.

INTRODUCTION

Posterior tarsal tunnel syndrome (PTTS) is an entrapment neuropathy due to compression of the tibial nerve or one of its terminal branches within the tarsal tunnel in the medial ankle. Compression can extend throughout the tibial nerve to its terminal branches that lie within the fibro-osseous tunnel under the flexor retinaculum.^{1,2} PTTS has many pathophysiology similarities when compared to carpal tunnel syndrome as they both involve anatomical tunnels; however, PTTS is far rarer and, thus, more difficult

to diagnose and treat.³ PTTS can present with a variety of symptoms leading to a pattern of underdiagnosis. Commonly reported symptoms to include but are not limited to pain, cramping, paresthesia, toe weakness, and sensory loss throughout the plantar aspect of the foot and medial side of the ankle.^{2,4} Timely identification of entrapment neuropathy and diagnosis of PTTS can limit the symptoms and long-term effects on the patient.⁴

PTTS is a significant condition that is not only routinely under-diagnosed but also proves to be a challenge to manage and treat.⁵ There still remains ambiguity within the di-

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agnostic and treatment course for PTTS. Misdiagnosing or underdiagnosing PTTS can lead to advanced sensory loss, muscle weakness, pain, and loss of mobility in the affected limb in which patients report difficulty running, walking, and even standing as the condition progresses.^{2,4} Although there is an abundance of intervention and treatment options, there is an inadequate amount of high-level evidence directing the most optimal intervention. Many studies have explored the vascular supply to the tibial nerve in an attempt to discover information about the sources of the symptoms that make PTTS difficult to diagnose and treat.⁶ However, a substantial number of cases of PTTS neuropathy can be successfully treated with conservative interventions, such as wearing a brace, neuropathic pain medication, and activity modification.⁴

ANATOMY

PTTS can cause a variety of neurologic symptoms due to the entrapment and compression of the tibial nerve and its terminal branches within the tarsal tunnel.² The tibial nerve carries fibers from the S1 and S2 nerve root and travels within the sciatic nerve as it passes inferiorly and traverses through the popliteal fossa. The tibial nerve then continues posteriorly to the tibia down the leg, where it provides innervation to the superficial and deep posterior leg compartment muscles. Once it reaches the foot, the tibial nerve passes through a fibrous tunnel known as the tarsal tunnel. The tarsal tunnel is formed by the flexor retinaculum, while the floor is composed of the distal tibia, talus, and calcaneal bones.⁴ After entering the tarsal tunnel within a distinct facial canal, the tibial nerve gives off the medial calcaneal branch (MCN), and then divides into the medial (MPN) and lateral plantar nerves (LPN).^{7,8} These nerves provide sensory innervation to the plantar and medial aspect of the foot as well as motor innervation to the intrinsic muscles of the foot.⁴

The tarsal tunnel contains a number of significant structures, including the tendons of 3 muscles: the flexor hallucis longus, flexor digitorum longus, and the posterior tibialis. The tarsal tunnel also contains the posterior tibial artery, vein, and nerve.⁴ The borders of the tarsal tunnel are defined as the tibia being the anterior border, posterior process of the talus as the posterior border, medial malleolus as the superior border, abductor hallucis as the inferior border, and the calcaneus as the lateral border.¹ The flexor retinaculum, also referred to as the lacinate ligament or internal annular ligament, is made up of a tight fibro-osseous band which attaches to the medial malleolus and extends to the calcaneus.¹ The flexor retinaculum creates an enclosed space by overlaying the tibial nerve.¹

PATHOPHYSIOLOGY

Focal compressive neuropathy of PTTS can originate from anything that physically restricts the volume of the tarsal tunnel. The variety of etiologies include distinct movements of the foot, trauma, vascular disorders (e.g., arteriole and venous malformations, varicose veins), soft tissue in-

flammation (e.g., rheumatoid arthritis, flexor tendonitis), diabetes mellitus, compression lesions, bony lesions (e.g., osteophytes of osteochondroma), masses (e.g., tumors), lower extremity edema (e.g., myxedema in hypothyroidism, pitting edema from fluid overload), and postoperative injury.^{2,4} Various motions and movements of the ankle and foot can cause the space within the tarsal tunnel to become restricted and initiate pain. Specifically, plantarflexion and eversion of the foot at the ankle have been shown to confine the contents within the tarsal tunnel and compress the tibial nerve or its terminal branches.⁴

A number of case reports have identified space-occupying lesions as the identifiable cause. A case study from 2014 describes a 26-year-old male who presented with left ankle and foot pain that worsened with activity. An accessory ossicle was located in the posteromedial portion of his talus resulting in tenosynovitis of the tendons within the tarsal tunnel and compression of the tibial nerve. The patient was successfully treated with surgical release of the tarsal tunnel. This was the first documented case of PTTS secondary to an accessory ossicle.⁹ A similar case in 2019 reported a 46-year-old patient who presented with PTTS secondary to accessory musculature within the posteromedial facet of the ankle.¹⁰ Another PTTS case in 2019 discovered an intraneural ganglion cyst within the tarsal tunnel that progressed to compression of the tibial nerve.¹¹

Varicose veins have been reported to be a fairly common venous etiology of PTTS. A case study in 2017 documented a 39-year-old male presenting with paresthesia and pain on the medial aspect of his right foot and ankle. MRI of the lower extremity showed widespread, advanced venous malformations that resulted in compression of the tibial nerve within the tarsal tunnel.¹²

Peripheral nerve tumors have also been found to initially present as PTTS in patients years before the mass is diagnosed. A 2017 review documented one case of a neurofibroma and another of a schwannoma that had developed on the tibial nerve and led to progressive compression of the tibial nerve.¹³ A similar case report in 2018 reported a 60-year-old male who presented with PTTS due to a schwannoma of the tibial nerve.¹⁴ Lymphomas have also been reported to cause PTTS. In 2018, an 80-year-old otherwise healthy female presented with symptoms of PTTS and was diagnosed with an isolated posterior tibial B-cell lymphoma that engulfed the tibial nerve within the flexor retinaculum.¹⁵ A separate case report in 2018 documented a patient presenting with PTTS secondary to an extranodal natural killer cell lymphoma within the tarsal tunnel. Prior to presentation, this patient had been treated for the malignancy and was presumed to be in remission; however, fluorodeoxyglucose positron emission tomography failed to image the lower extremity until he presented with symptomatic PTTS.¹⁶ This report emphasizes the importance of including a detailed past medical history and a systematic approach to help identify the correct diagnosis for patients presenting with PTTS for unknown reasons.

EPIDEMIOLOGY

The variety of etiologies that are known to cause PTTS leads approximations of prevalence to be largely influenced by the study population characteristics.^{17–21} Unsurprisingly, more recent large-scale epidemiological studies regarding the prevalence of PTTS are lacking. However, in early literature, idiopathic and posttraumatic are the most commonly cited causes of PTTS, with 20–46% of reported cases being idiopathic.²² In one study by Oh and Meyer et al., 43% of the cases were posttraumatic, and 13% were sports-related mechanisms.²³ Two different studies by Takakura et al. and Nagaoka et al. found ganglions or varicosities to be the most common cause.^{18,24} Other less common etiologies include foot deformities, rheumatoid arthritis, synovitis, hypothyroidism, acromegaly, and space-occupying lesions such as a neurilemmoma, lipoma, or anomalous muscle.³ In a more recent publication, Meadows et al. argue PTTS occurs much more frequently in athletes than in the general population secondary to direct contusion, ill-fitting shoes, space-occupying pathology, or lower limb malalignment.⁷ Additionally, sports requiring hyper-dorsiflexion can predispose athletes to develop PTTS due to the increased pressure within the tarsal tunnel during this movement.²⁵

RISK FACTORS

Extra and accessory muscles around the ankle are common variants of ankle anatomy and contribute to an increased risk of developing PTTS.²⁶ The most common accessory muscle associated with PTTS is the flexor digitorum accessorius longus (FDAL) muscle; however, the presence of a tibio calcaneus internus muscle or accessory soleus muscle has also been reported to cause PTTS.²⁶ The accessory muscle mimics a mass lesion in the ankle that can compress the posterior tibial nerve as well as other complications.²⁶ A case report published in 2015 diagnosed a 29-year-old male with bilateral PTTS after MRI revealed the presence of bilateral FDAL within the tarsal tunnel.²⁷ Bilateral tarsal tunnel decompression was performed in combination with resection of the FDAL. At six months postoperatively, the patient had no pain and full sensation bilaterally. A separate case report identified a 17-year-old male presenting with a history of swelling and pain within the posterior medial aspect of the right ankle.²⁸ MRI showed a large accessory soleus muscle resulting in a mild mass effect on the flexor hallucis longus and entrapment of the posterior tibial nerve. Complete resection of the accessory soleus muscle was performed, and the patient was asymptomatic at 12 months postoperatively.

Chronic medial ankle instability is another risk factor associated with the development of PTTS. The deltoid ligament complex is composed of a superficial and deep layer, as well as the calcaneonavicular (spring) ligament.²⁹ These structures are responsible for stabilizing the medial ankle joint. Persistent and repeated ankle sprains can cause pathologic ligament laxity and increase the risk of develop-

ing chronic rotational ankle instability, resulting in associated symptoms, including PTTS.²⁹

Surgical procedures to correct hindfoot varus deformities such as pes cavovarus can also increase the risk of developing a tibial nerve palsy.³⁰ The reduction of the tarsal tunnel during the procedure is believed to lead to the development of posterior tibial nerve compression by the flexor retinaculum, however, the exact mechanism is unclear.^{30–32}

A number of isolated case reports have also documented risk factors that are associated with the development of PTTS. In a report from 2016, a 19-year-old female developed posteromedial ankle pain one year after ankle arthroscopy and arthrotomy to remove two ossicles.³¹ Post-operative MRI showed a lesion consistent with heterotopic ossification (HO) extending from the posteromedial talus and entrapping the posterior tibial nerve within the tarsal tunnel. HO is a reactive phenomenon that results in the formation of trabecular bone within soft tissue. Theoretically, any severe trauma to the ankle can increase the risk of developing HO leading to PTTS. A separate case report from 2019 documented a 55-year-old woman with a history of hyperuricemia and diabetes mellitus presenting with a 6-month history of discomfort in the right foot that had progressively worsened over three weeks.³³ MRI revealed a fusiform mass and fluid surrounding the flexor hallucis longus tendon. The patient was diagnosed with gout tenosynovitis of the flexor hallucis longus tendon with tophus formation, resulting in PTTS. Surgical resection of the tophus mass and tendon sheath resolved the PTTS symptoms. An extremely rare report from 2016 described a 52-year old male who presented with recurrent tarsal tunnel syndrome secondary to three different types of ganglion over a period of 12 years.³⁴ The patient initially presented with a three-month history of paraesthesia, numbness, and pain with no history of trauma. MRI revealed the presence of a single cyst within the tarsal tunnel and was diagnosed with an epineural ganglion cyst after surgical resection. Approximately two years later, the patient presented with recurrent left foot paraesthesia and pain. MRI revealed a lesion invading through the fascicles, indicating the presence of an intra-fascicular ganglion. Three years later, the patient returned with a two-week history of numbness in the left foot. MRI showed a lesion near the subtalar joint that was determined to be an extra-neural ganglion. The most recent follow-up exam showed no indication of a mass or lesion within the tarsal tunnel or surrounding area.

CLINICAL PRESENTATION

The symptoms of tarsal tunnel syndrome may vary depending on the location of nerve compression within the tarsal tunnel.¹ In general, compression of the posterior tibial nerve results in clinical findings consisting of numbness, burning, and painful paresthesia in the heel, medial ankle, and plantar surface of the foot.³⁵ The presence of a positive Tinel sign along the posterior tibial nerve at the ankle is also an indicator of PTTS.³⁵ Pain and paresthesia are typically exacerbated with activity and often worsens throughout the day as a patient is on their feet.¹ Pain while sleeping

may occur if the foot is in an everted and dorsiflexed position throughout the night.⁷ Approximately fifty percent of patients diagnosed with PTTS also report experiencing cold sensation along the medial and lateral plantar nerve distribution.¹⁶ Patients may also present with weakness of toe flexion and extension with severe disease progression due to atrophy of the plantar muscles.¹

DIAGNOSIS

While there is a general consensus that PTTS exists, there is still debate surrounding the diagnosis and etiology of the disease. Nerve conduction studies and electromyography used to diagnose nerve entrapment syndromes have poor diagnostic sensitivity, making the physical exam findings and patient history an important component to diagnosis.³⁶ A positive Tinel sign is the physical exam finding associated with localized nerve compression in nerve entrapment disorders. A positive test is reported when tapping along the nerve elicits the presence of tingling distally in the nerve distribution. Diagnosis of PTTS can be made with the presence of a positive Tinel sign in combination with the physical symptoms of pain and numbness along the plantar and medial surfaces of the foot.²

Additional provocative physical exam maneuvers include the dorsiflexion-eversion test, Trepman test, and triple-compression test.⁴ The dorsiflexion-eversion test involves passive dorsiflexion and eversion of the ankle for 5-10 seconds and evaluating for the presence of pain or numbness at the ankle or sole.³⁷ On the contrary, the Trepman test passively plantarflexes and inverts the ankle to evaluate for the presence of pain or numbness in the same distribution.³⁸ The triple-compression test combines the Trepman and Tinel tests. Studies have demonstrated greater sensitivities and specificities with the dorsiflexion-eversion and triple-compression tests.⁴

Electromyography studies in patients with PTTS can be used to determine what branches of the posterior tibial nerve are affected by compression. A needle electromyography exam is useful in further developing a treatment plan and is often followed by advanced imaging to evaluate the foot for a mass or ganglion cyst in the affected area.²⁰ For example, MRI can be used to visualize areas of bony overgrowth when PTTS is suspected.³⁹ Talocalcaneal coalition results from the fusion of the sustentaculum tali to the talus and can cause PTTS when a bony irregularity protrudes into the tarsal tunnel and compresses the posterior tibial nerve.⁴⁰ In cases of idiopathic PTTS, ultrasound is used to detect enlargement of the posterior tibial nerve within the tarsal tunnel.⁴¹ Ultrasonography can also be used to detect variation in the bifurcation of the posterior tibial nerve in order to better identify the precise area of entrapment and aid in targeted treatment.⁴²

TREATMENT

CONSERVATIVE

Patients with PTTS usually present with poorly localized burning pain and paresthesia along the plantar surface of

the foot. Consequently, management is directed towards relieving pain, inflammation, and tissue stress.⁴³ Initially, patients are treated conservatively unless there are signs of muscle atrophy or motor nerve involvement. Some studies have demonstrated success with rest as well as heat or cryotherapy to the affected region. Additionally, non-steroidal anti-inflammatory drugs, corticosteroid injections, opioids, GABA analog medications, tricyclic antidepressants, and vitamin B-complex supplements have also been shown to effectively manage some of the symptoms.⁴⁴

Furthermore, physical therapy involving muscular stretching and strengthening of the gastrocnemius, soleus, tibialis anterior and posterior, and peroneal and short toe-flexor muscles can be trialed.^{1,44,45} Custom orthotics may also be used to correct overpronation and support the medial and lateral longitudinal arches of the affected foot. These non-operative treatments should be tried for at least 12 to 24 months before surgical management is considered.¹ When PTTS is recalcitrant to conservative treatments, surgical nerve decompression of the tarsal tunnel is recommended.

SURGICAL TREATMENT

If PTTS is recalcitrant to conservative treatment, surgical decompression of the nerve within the tarsal tunnel is recommended. Surgical management begins with identifying the location of nerve entrapment preoperatively.¹ Intraoperatively, a 5-7 centimeter incision is made along the posteromedial aspect of the ankle, starting just proximal to the medial malleolus and extending distally along the course of the posterior tibial nerve.¹ The surgeon then releases the retinacula and any space-occupying lesion, including any vascular leash that may compress the nerve or scar around the nerve. It is also important to explore the posterior tibial nerve distal to its MPN and LPN branches and the abductor hallucis fascia to ensure no additional areas of compression.¹

Surgical treatment outcomes for PTTS vary considerably and are influenced by factors such as age, duration of disease, the timing of surgery, presence of space-occupying mass, and presence of metabolic disease.⁴⁶ In an early study in 2003 by Gondring et al., 60 patients (68 feet) who underwent tarsal tunnel release for PTTS noted significant improvement following return to daily life in quality of work, productivity, and interpersonal relationships. These improvements were noted to be secondary to symptom relief with 51% of patients obtaining subjective symptom relief and 85% obtaining objective complete symptom relief.^{17,47} In another study by Sammarco et al. following 62 patients receiving tarsal tunnel release for at least 12 months following the operation, significant improvements were also noted in quality of life, subjectively and functionally. Improvements in American Orthopaedic Foot and Ankle Society scores from 61/100 to 80/100 were noted postoperatively.⁴⁸ More recent robust evidence of surgical outcomes is, however, limited.

Recurrence of PTTS following surgical release remains a difficult problem for less than 5% of patients.⁴⁹⁻⁵¹ It may be a result of a failure to achieve hemostasis during the pri-

mary surgery, which results in excessive scarring and nerve damage.⁴⁹ Subsequent operations on failed tarsal tunnel decompression result in less predictable outcomes as compared to initial surgical treatment. As a result, recent studies have been published on advancements to improve primary surgery outcomes.

Nerve conduction velocity/electromyography studies have recently been used to diagnose and to help identify the level of entrapment.⁴⁶ Advancements in intraoperative nerve monitoring can also serve as an adjuvant to decompression surgery by ensuring adequate release and improving outcomes.⁴⁶ In a study performed by Still et al., intraoperative neuromonitoring was used to measure the voltage of muscle twitches distal to the tarsal tunnel both before and after the decompression.⁴⁶ Surgeons were able to compare the pre-decompression voltage with periodic voltage measurements throughout the surgery, guiding the surgeon to dissect deeper within the tarsal tunnel if there is inadequate release.⁴⁶ Outcomes were divided into excellent, fair, and poor with 29 out of 38 patients (76%) reporting excellent outcomes, thus suggesting the potential usefulness of this modality in improving outcomes of tarsal tunnel decompression surgery with further randomized control studies.

Lipografting is another technique that has been described in the literature as a potential modality to improve outcomes. Prior literature on nerve wrapping for upper extremity compression neuropathies has shown its ability to serve as a protective barrier to prevent excessive pressure and improve nerve regeneration.^{52,53} Morandi et al. described the first case to date of using lipografting as a therapeutic option for revision tarsal tunnel release in a 65-year-old woman.⁵⁴ Excellent outcomes were reported with a visual analog pain scale improving from 8/10 preoperatively to 1/10 postoperatively, suggesting lipografting after surgical revision neurolysis of the tibial nerve can improve postoperative outcomes.⁵⁴

MINIMALLY INVASIVE TREATMENT

A variety of minimally invasive treatments for PTTS have recently begun to gain traction in the literature for a number of reasons. Generally speaking, the tibial nerve can be compressed at two different regions within the tarsal tunnel; in proximal PTTS the nerve is entrapped within the flexor retinaculum, whereas in distal PTTS it is compressed by the medial intermuscular septum. Marcos et al. argues that ultrasound-guided release of the tibial nerve has the advantage of being an option for patients who are not candidates for conventional surgery, such as those with vascular insufficiency or diabetes.⁵⁵ Additionally, this technique may be associated with faster recovery and fewer postsurgical complications as compared to open surgery.^{4,56}

Marcos et al. previously demonstrated the safety and reproducibility of this approach in 12 fresh cadaveric specimens, performing effective proximal and distal tarsal tunnel releases without injury to surrounding neurovascular structures.⁵⁵ Fernandez-Gibello et al. also conducted an anatomic study on ten cadaveric feet, demonstrating the effectiveness and safety of an ultrasound-guided minimally

invasive surgical approach to decompress the nerve in patients with proximal PTTS.⁵⁶ A similar cadaveric study by Moroni et al. found similar success in select patients with distal PTTS.⁵⁷ Large outcome studies are still lacking, however, ultrasound-guided minimally invasive surgery may be an effective strategy to define the location of decompression and to delineate anatomical structures intraoperatively. Additional prospective trials are needed to evaluate the efficacy and safety of this technique.

One study from Japan utilized a minimally invasive endoscopic approach to release the tarsal tunnel in eight ankles from five patients.⁵⁸ A 1-cm portal incision was created at the proximal medial ankle, and the components of the tarsal tunnel were identified using the Universal Subcutaneous Endoscope (USE) system. Under complete endoscopic guidance, the flexor retinaculum and part of the abductor hallucis muscle were released. The postoperative outcomes demonstrated 100% of the patients reported improved clinical signs, suggesting that this less invasive endoscopic management may be a sufficient alternative to the standard open decompression.

Pulsed radiofrequency (PRF) is another minimally invasive technique being studied for the treatment of PTTS. In a study of two cases by Chon et al., ultrasound-guided PRF was effective in managing intractable PTTS.⁵⁹ PRF has traditionally been used for the treatment of a variety of pain and neuropathic syndromes, such as arthritis, trigeminal neuralgia, and others.⁵⁹ The success of PRF depends on the gap between the targeted nerve and the RF needle tip; therefore, ultrasound guidance in PRF is advantageous in that it allows direct visualization of the nerve and avoids nerve trauma from repeat needling.⁶⁰ Further prospective studies evaluating the efficacy of this modality is also required.

CONCLUSION

PTTS is a rare entrapment neuropathy of the posterior tibial nerve that typically presents with burning, tingling, and shooting pain along the heel and medial aspect of the ankle with symptoms that worsen with standing and walking. Anything that physically restricts the space within the tarsal tunnel increases the risk of developing PTTS, resulting in a broad differential diagnosis. The variety of etiologies inherently make it difficult for studies to accurately describe the prevalence of this syndrome in small sample sizes without concern for bias. Conservative management remains the mainstay of treatment; however, if conservative treatment fails, surgical decompression of the flexor retinaculum is indicated. Although outdated data reported favorable results, more recent data report that outcomes of this surgical procedure continues to be lacking. In recent years, a number of alternative minimally invasive treatment options have been investigated, but these studies have small sample sizes or were conducted on cadaveric models. Additional large-scale prospective studies are required to assess the efficacy and safety of these treatments.

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