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Tarsal Tunnel Syndrome

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AUTHOR AND EDITOR INFORMATION

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Author and Editor Disclosure

Synonyms and related keywords: tarsal tunnel neuropathy, entrapment neuropathy of the tibial nerve, posterior tibial neuropathy, compression of the tibial nerve

Patient Education

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INTRODUCTION

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Tarsal tunnel syndrome is a condition that is caused by compression of the tibial nerve or its associated branches as the nerve passes underneath the flexor retinaculum at the level of the ankle or distally.

History of the Procedure

Tarsal tunnel syndrome is analogous to carpal tunnel syndrome of the wrist. In 1962, Keck and Lam first described the syndrome and its treatment.

Problem

Tarsal tunnel syndrome is a multifaceted compression neuropathy that typically manifests with pain and paresthesias that radiate from the medial ankle distally and, occasionally, proximally. These findings may have a variety of causes, which can be categorized as extrinsic, intrinsic, or tensioning factors in the development of signs and symptoms of tarsal tunnel syndrome.

Extrinsic causes may contribute to the development of tarsal tunnel syndrome. Examples include external trauma due to crush injury, stretch injury, fractures, dislocations of the ankle and hindfoot, and severe ankle sprains.

Local causes may be intrinsic causes of the neuropathy. Examples include space-occupying masses, localized tumors, bony prominences, and a venous plexus within the tarsal canal.

Nerve tension caused by a valgus foot can cause symptoms that are identical to those of a circumferential nerve compression.

Symptoms of tarsal tunnel syndrome vary from individual to individual, but clinical findings generally include the following: sensory disturbance that varies from sharp pain to loss of sensation, motor disturbance with resultant atrophy of intrinsic musculature, and gait abnormality (eg, overpronation and a limp due to pain with weight bearing).

A hindfoot valgus deformity may potentiate the symptoms of tarsal tunnel syndrome because the deformity may increase tension due to an increase in eversion and dorsiflexion.

Frequency

To the authors' knowledge, no studies have demonstrated a statistical association for tarsal tunnel syndrome with work conditions or activities of daily living. The prevalence and incidence of tarsal tunnel syndrome have not been reported.

Etiology

Several factors may contribute to the development of tarsal tunnel neuropathy. Soft-tissue masses may all contribute to compression neuropathy of the posterior tibial nerve. Examples of such masses include lipomas, tendon sheath ganglia, neoplasms within the tarsal canal, nerve sheath and nerve tumors, and varicose veins. Bony prominences and exostoses may also contribute to the disorder. A study by Daniels et al demonstrated that a valgus deformity of the rearfoot may contribute to the neuropathy by increasing the tensile load on the tibial nerve.[1]

Pathophysiology

Tarsal tunnel syndrome is a compression neuropathy of the tibial nerve that is situated in the tarsal canal. The tarsal canal is formed by the flexor retinaculum, which extends posteriorly and distally to the medial malleolus.

The symptoms of compression and tension neuropathies are similar; therefore, differences in these conditions cannot be simply identified by the symptoms alone. In certain instances, compression and tension neuropathies may coexist.

The double-crush phenomenon originates from work published by Upton and McComas in 1973. The hypothesis behind this phenomenon may be stated as follows: Local damage to a nerve at one site along its course may sufficiently impair the overall functioning of the nerve cells (axonal flow), such that the nerve cells become more susceptible to compression trauma at distal sites than would normally be the case.

The nerves are responsible for transmitting afferent and efferent signals along their length, and they are also responsible for moving their own nutrients, which are essential for optimal functioning. The movement of these intracellular nutrients is accomplished through a type of cytoplasm within the nerve cell called axoplasm (referring to cytoplasm of the axon). The axoplasm moves freely along the entire length of the nerve. If the flow of the axoplasm (ie, axoplasmic flow) is blocked, the nerve tissue that is distal to that site of compression is nutritionally deprived and more susceptible to injury.

Upton and McComas further suggested that a high proportion (75%) of patients with one peripheral nerve lesion did, in fact, have a second lesion elsewhere. The authors implied that both lesions contribute to the patients' symptoms. These lesions were originally studied in cases of brachial plexus injury with an increased incidence of carpal tunnel neuropathy. An analogous example of the double-crush phenomenon in the feet would be a compression of the S1 nerve root, resulting in an increased likelihood of compression neuropathy in the tarsal canal.

Clinical

History

Clinical assessment of the patient with suspected peripheral neuropathy should include careful review of the past medical history, with attention to systemic diseases that can be associated with peripheral neuropathy, such as diabetes and hypothyroidism.

Many medications can also cause a peripheral neuropathy. These include nitrous oxide, colchicine, metronidazole, lithium, phenytoin, cimetidine, disulfiram, chloroquine, amitriptyline, thalidomide, cisplatin, pyridoxine, and paclitaxel. Conditions that are related to these drugs typically involve distal symmetric sensorimotor neuropathy. Any patient drug or alcohol use or exposure to solvents and heavy metals should be investigated.

Patients should also be questioned about their exposure to the human immunodeficiency virus (HIV), vitamin use, Lyme disease, and foreign travel (ie, exposure to leprosy). A family history that demonstrates the familial presence of hammer toes, cavus foot, gait abnormalities, and muscle weakness may indicate a long-standing or familial neuropathy.

Physical examination

Patients typically present with vague symptoms of foot pain, which can sometimes be confused with plantar fasciitis. Findings of pain, paresthesias, and numbness are not uncommon. In some cases, atrophy of the intrinsic foot muscles may be noted, although this may be clinically difficult to ascertain. Eversion and dorsiflexion may cause symptoms to increase at the endpoint range of motion.

The Tinel sign (radiation of pain and paresthesias along the course of the nerve) may often be induced posterior to the medial malleolus. Symptoms generally subside with rest, although they typically do not disappear altogether. (Percussion of a nerve with a resultant distal manifestation of paresthesias is known as the Tinel sign. This should not be confused with the Phalen sign, which is compression of the suspected nerve for 30 seconds, with subsequent reproduction of the patient's symptoms.)

Physical examination may demonstrate reduced sensitivity to light touch, pinprick, and temperature in patients with distal symmetric sensorimotor neuropathy.

Radiographic examination of the patient's limbs may demonstrate loss of bone density, thinning of the phalanges, or evidence of neuroarthropathy (eg, Charcot disease) in long-standing neuropathies. Additionally, trophic changes may include pes cavus, loss of hair, and ulceration. These findings are most prominent in those with diabetes, amyloid neuropathy, leprosy, or hereditary motor sensory neuropathy (HMSN) with prominent sensory involvement. Perineural thickening may be noted in cases of leprosy and amyloid neuropathy.

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A positive history combined with supportive physical findings (see Clinical, Physical examination) and positive electrodiagnostic results makes the diagnosis of tarsal tunnel neuropathy highly likely. Patients with a high likelihood of nerve compression generally have a good clinical result after surgical decompression of the tibial nerve. It is important to note, however, that the absence of positive electrodiagnostic results does not rule out the possibility of decompression for treating the symptoms of tarsal tunnel syndrome.

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RELEVANT ANATOMY

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The tarsal tunnel is a structure in the foot that is formed between the underlying bones of the foot and the overlying fibrous tissue. The flexor retinaculum (laciniate ligament) constitutes the roof of the tarsal tunnel and is formed by the deep fascia of the leg and the deep transverse fascia of the ankle. The proximal and inferior borders of the tunnel are formed by the inferior and superior margins of the flexor retinaculum. The floor of the tunnel is formed by the superior aspect of the calcaneus, the medial wall of the talus, and the distal-medial aspect of the tibia. The remaining fibroosseous canal forms the tibiocalcaneal tunnel. The tendons of the flexor hallucis longus muscle, flexor digitorum longus muscle, tibialis posterior muscle, posterior tibial nerve, and posterior tibial artery pass through the tarsal tunnel.

The posterior tibial nerve lies between the posterior tibial muscle and the flexor digitorum longus muscle in the proximal region of the leg and then passes between the flexor digitorum longus and flexor hallucis longus muscle in the distal region of the leg. The tibial nerve passes behind the medial malleolus and through the tarsal tunnel and then bifurcates into cutaneous, articular, and vascular branches. The main divisions of the posterior tibial nerve include the calcaneal, medial plantar, and lateral plantar nerve branches. The medial plantar nerve passes superior to the abductor hallucis and flexor hallucis longus muscles and later divides into the 3 medial common digital nerves of the foot and the medial plantar cutaneous nerve of the hallux. The lateral plantar nerve travels directly through the belly of the abductor hallucis muscle, where it later subdivides into branches.

The innervation of the branches of the posterior tibial nerve is as follows:

- Calcaneal branch – Medial and posterior aspects of the heel
- Medial plantar branch – Cutaneous branches to the plantar medial aspect of the foot; motor branches to the abductor hallucis and flexor digitorum brevis muscles; and branches to the talonavicular and calcaneonavicular joints
- Lateral plantar branch – Motor branches to the abductor digiti quinti and quadratus plantae muscles; cutaneous nerve to the fifth digit; communicating branch to the fourth common digital nerve; motor branches to the lumbricals; second, third, and fourth interossei branches to the transverse head of the adductor hallucis and the muscles of the first interosseous space

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CONTRAINDICATIONS

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Surgery is contraindicated in patients who are not medically stable enough to undergo this elective procedure. In addition,

appropriate medical workup should be initiated in patients who may have medical comorbidities that may preclude them from undergoing such procedures.

Several conditions may mimic or coexist with tarsal tunnel neuropathy. Surgical treatment may depend on an accurate determination of the conditions that are similar to tarsal tunnel syndrome but do not improve after surgical decompression.

The differential diagnosis for tarsal tunnel syndrome may include plantar fasciitis; stress fractures of the hindfoot, particularly the calcaneus; herniated spinal disk; peripheral neuropathies, such as those caused by diabetes or alcoholism; and inflammatory arthritides, such as Reiter syndrome or rheumatoid arthritis.

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WORKUP

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Laboratory Studies

- Electromyography (EMG) and nerve conduction velocity (NCV) studies may be a useful initial tool in evaluating suspected cases of tarsal tunnel syndrome and in confirming the presence of neuropathy. Additionally, the type of nerve fiber (sensory, motor, or both) and the pathophysiology (axonal vs demyelinating and symmetric vs asymmetric) can be differentiated with the information obtained from EMG and/or NCV. A physiatrist or neurologist who is experienced in extremity EMG and NCV studies can best perform these tests.

  EMG studies may demonstrate prolonged posterior tibial distal nerve latency to the abductor hallucis or abductor digiti quinti muscles. This finding may also be accompanied by low motor amplitude or absent responses in either of these muscles. Early on, medial and/or lateral plantar sensory action potentials may be affected with prolonged latency, slowed velocity, and decreased amplitude. Sensory action potentials may be unobtainable in advanced cases of tarsal tunnel syndrome.

  Needle examination of the abductor hallucis and/or abductor digiti quinti muscles may show denervation and active and/or chronic changes. To ensure that this finding is not an S1 root lesion, the posterior tibial muscles above the tarsal tunnel (posterior tibialis) or muscles other than the posterior tibial muscles (extensor digitorum brevis) should demonstrate sparing. The lumbosacral paraspinal muscles should be intact.

  EMG and NCV testing values include the following:

  - Prolonged distal motor latency: Terminal latencies of the abductor digiti quinti muscle (lateral plantar nerve) longer than 7.0 ms are abnormal.
  - Terminal latencies of the abductor hallucis muscle (medial plantar nerve) longer than 6.2 ms are abnormal.
  - Fibrillations in the abductor hallucis muscle may be present.

  Repeat EMG studies that are performed 6 months after surgery may aid in assessing the physiologic success of the decompression procedure in patients who had positive results. Decreases may be noted in the distal latencies.

  Results of NCV studies may be normal in patients with small fiber neuropathies. Additionally, lower-extremity sensory responses may be absent in normal elderly patients. Therefore, electrodiagnostic testing should not be a substitute for a good clinical examination.

- Diabetes mellitus produces a distal, symmetric sensory and motor polyneuropathy. This is an axonal neuropathy due to degeneration of distal axons. Diabetes also produces a neuropathy due to microangiopathy, which results in a proximal, asymmetric mononeuropathy (primarily motor nerves). The initial evaluation should include urinalysis and tests of the fasting serum glucose level, hemoglobin A1C (HbA1C/HgA1C), blood urea nitrogen

(BUN), creatinine, complete blood cell count (CBC), erythrocyte sedimentation rate (ESR), and vitamin B12 levels.

- The arthritis that is associated with Reiter syndrome typically affects the knees, ankles, and feet, causing pain and swelling; wrists, fingers, and other joints are less often affected. Patients with Reiter syndrome commonly develop inflammation where the tendon attaches to the bone, a condition called enthesopathy. Enthesopathy may result in heel pain and the shortening and thickening of fingers and toes. Some patients who are affected with Reiter syndrome also develop heel spurs that are associated with chronic or long-lasting foot pain.

  Men between the ages of 20 and 40 years are most likely to develop Reiter syndrome. It is the most common type of arthritis that affects young men; among men younger than 50 years, about 3.5 per 100,000 develop Reiter syndrome each year. Approximately 3% of all men with a sexually transmitted disease develop Reiter syndrome. Women can also develop this disorder, though less often than men, with features that are often milder and more subtle.

  About 80% of affected patients are positive for human leukocyte antigen (HLA)-B27. Only 6% of people who do not have Reiter syndrome have the HLA-B27 gene.

  In performing a rule-out workup of underlying systemic arthritic conditions, ESR, rheumatoid factor (RF), and antinuclear antibody (ANA) serology should be performed.

  Typically, patients with rheumatic diseases, including Reiter syndrome, have an elevated ESR. However, in Reiter syndrome, results of RF and ANA testing are negative; therefore, HLA-B27 typing may be useful in differentiating this seronegative arthropathy from other arthritides.

- Generalized amyloidosis can cause a peripheral neuropathy due to pressure atrophy of nerve fibers. The central nervous system is not affected except in areas that lack a blood-brain barrier, such as the choroid plexus and pineal gland. Nerve biopsy is helpful in specific cases to diagnose leprosy, amyloid neuropathy, sarcoidosis, and leukodystrophies.

Imaging Studies

- Magnetic resonance imaging (MRI) and ultrasonography may be useful modalities in suspected cases of soft-tissue masses and other space-occupying lesions in the tarsal tunnel.

  Additionally, MRI is useful in assessing for flexor tenosynovitis and unossified subtalar joint coalitions.

- Plain radiography is useful in evaluating the patient's underlying foot structure, fractures, bony masses, osteophytes, and subtalar joint coalition.

Diagnostic Procedures

- See Laboratory Studies and Imaging Studies

Histologic Findings

Regarding neuroma in continuity, in most cases, the nerve has an intact perineural sheath. This entity may result from chronic nerve compression and irritation, which causes nerve swelling. The proliferation of fibrous tissue causes nerve compression; therefore, this type of entity must be decompressed, and the fibrous tissue may need to be removed.

Ganglion cysts that cause compressive peripheral neuropathies are unusual, but when combined, they are not an uncommon etiology. The source and cause of ganglion cysts remain unsettled issues; one theory was fibrillar degeneration of collagen, with accumulation of intracellular and extracellular mucin. If encountered during surgery, these lesions need to be removed en toto as part of thorough nerve decompression.

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TREATMENT

Medical therapy

Medical therapy for tarsal tunnel syndrome may consist of local injection of steroids into the tarsal canal. An acceptable conservative approach in the early treatment of tarsal tunnel neuropathy includes the use of local anesthetics and soluble steroids, which may aid in the reduction of the patient's pain. These therapies may occasionally produce complete relief of symptoms, but they need to be performed judiciously, as additional nerve injury may occur from improperly placed syringe needles. Physical therapy may be of some value in reducing local soft-tissue edema, thereby easing pressure on the compartment.

Also, in symptomatic patients who exhibit a contracture of the gastrocnemius muscle of the triceps surae, stretching exercises that are designed to improve the flexibility of the gastrocnemius should be initiated. In cases in which the patient has a pes planovalgus foot type, a well-designed foot orthosis may reduce tension on the tibial nerve by decreasing the load on the medial column. This is accomplished by providing a medial longitudinal posting on the orthotic for both the hindfoot and forefoot.

The use of night splints with the foot in plantar flexion and varus may be considered in patients with a valgus foot. The long-term efficacy of this modality lacks well-controlled comparison studies with outcome measures, but it is commonly used in clinical practice.

Surgical therapy

When conservative therapy fails to alleviate the patient's symptoms, surgical intervention may be warranted. Space-occupying masses require removal. Numerous reports exist of neurilemoma of the tibial nerve, which may need to be removed. A thorough knowledge of the local anatomy is a prerequisite before attempting release of the affected nerve.

External neurolysis of the nerve may be necessary if surgical exploration demonstrates adhesions or scar tissue as the cause of the nerve impingement. Moreover, if scarring or entrapment encapsulates the nerve tissue, in addition to external neurolysis, a release of the epineurium is warranted.

Preoperative details

The patient may be placed in either the supine or the lateral recumbent position to facilitate exposure of the medial aspect of the operative foot. Use of a pneumatic tourniquet is recommended.

Intraoperative details

A curved incision should be made approximately 1 cm posterior to the distal tibia and carried in the plantar direction, paralleling the shaft and malleolus and curving gradually toward the sustentaculum tali. The retinaculum should be identified and carefully released in its entirety. The posterior tibial nerve should be identified, visualized, and left undisturbed along the course until its bifurcation at the porta pedis. Care should be exercised to avoid cutting the small calcaneal branches that arise from the posterior tibial nerve; these branches are often surrounded by fatty tissue and may be difficult to easily visualize.

The medial plantar branch of the posterior tibial nerve should be identified and traced along the margin of the flexor sheath of the hallucis longus. The lateral branch should be followed into the abductor hallucis. Any fibrous bands that are noted to be constricting the nerves should be carefully released.

After release, all the branches of the tibial nerve should be lying free of any fascial covering. The tourniquet should be deflated to observe for and control bleeding. A layered closure should be performed, including the subdermal layer but not the flexor retinaculum. The skin may be closed with sutures or staples; a drain is not necessary. In a tarsal tunnel release, a layer closure of the wound should be performed by taking care not to reapproximate the extensor retinaculum, because this is the most common cause of the entrapment neuropathy.

Postoperative details

A mild compression dressing and the initial immobilization should be applied with slight inversion to the affected area by using a splint for 3 weeks of non—weight bearing. After the splint is discontinued, the patient may begin joint mobilization and a graduated return to weight bearing.

Follow-up

The patient should be non—weight bearing for a period of 3 weeks to allow for proper healing. Early mobilization should be initiated to decrease the formation of scar tissue, which may itself contribute to compression neuropathy. The use of surgical shoe aids in reducing pressure on the surgical site is recommended. Formal physiotherapy may be helpful for the patient to regain strength and motion and for the relief of residual pain.

After suture removal, the patient should be able to resume the use of soft shoes, taking care to avoid shoes that may cause pressure or irritation of the surgical site. In patients who have a pes planus foot type, insole orthoses should be considered to stabilize the medial column.

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COMPLICATIONS

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Because of the anatomy of the affected region, several complications of compression release surgery may arise, most of which can be minimized with meticulous dissection and careful identification of the local anatomy.

Laceration of the nerve or posterior artery could have significant deleterious effects on foot function. A failure to adequately release the retinaculum along its entire course may lead to poor postoperative results. This is the most likely etiology of surgical failure.

Additionally, associated plantar fasciitis may be a cause of persistent pain in the medial heel region after decompression, which may need to be addressed separately. A case study by Kim and Dellon demonstrated that a neuroma of the distal saphenous nerve may need to be considered as a causative factor if pain continues after surgical release.[2]

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OUTCOME AND PROGNOSIS

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Properly performed decompression may yield satisfactory results. An initial marked decrease in pain and paresthesias may occur, followed by a reduction of symptoms to the extent that the patient may be able to tolerate the symptoms. Complete resolution of symptoms may not be possible because the disorder has numerous etiologies and because the likelihood of irreversible nerve damage exists. An increase in pain after decompression, however, is extremely rare.

Studies by Mann demonstrated that approximately 75% of patients who undergo surgical decompression have appreciable pain relief, and 25% obtain little or no relief.[3] Mann also stated that a repeat surgical exploration of a previous tarsal canal release rarely causes appreciable benefit to the patient.

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FUTURE AND CONTROVERSIES

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Some concern exists regarding whether decompression of the tibial nerve in patients with marked pes planovalgus deformity may cause a deleterious effect because decompression of the medial retinacular compartment may be associated with an increase in nerve tension. Questions arise regarding whether a joint stabilization procedure may be a necessary adjunct in determining long-term postoperative success. To the author's knowledge, no studies have been performed to assess the long-term efficacy of decompression and stabilization, decompression and orthoses management, and decompression alone.

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MULTIMEDIA

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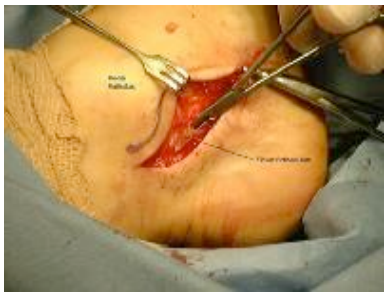
Media file 1: **Surgical approach for release of the flexor retinaculum in a patient with tarsal tunnel syndrome.**



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