Tarsal Tunnel Syndrome

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KEYWORDS

• Tarsal tunnel syndrome • Chronic heel pain
• Space-occupying lesions • Distal tarsal tunnel

Tarsal tunnel syndrome, unlike its similar sounding counterpart in the hand, is a significantly misunderstood clinical entity in every respect. Confusion concerning the anatomy involved, the presenting symptomatology, the appropriateness and significance of various diagnostic tests, conservative and surgical management, and, finally, the variability of reported results of surgical intervention attests to the confusion surrounding this condition. The terminology involved in various diagnoses for chronic heel pain is also a hodgepodge of poorly understood entities, many of which are probably variants of the tarsal tunnel syndrome. Because the diagnosis of carpal tunnel syndrome and its usual causes are well understood, and the name sounds similar to tarsal tunnel syndrome, the diagnostic tools used and the expectations for successful management have not been realized. In fact, the entities have little in common. In the carpal tunnel, the median nerve lies within a fibro-osseous tunnel surrounded by nine tendons, all of which are subject to various inflammatory conditions; also, the underlying carpal bones are subject to synovitis and traumatic dislocations, which create space-occupying lesions. These and hormonal factors result in increased pressure in the tunnel and effects on the nerve, which are easily measurable with delays in nerve conduction and electromyographic changes found in the thenar eminence. These entities do not exist per se in the tarsal tunnel.

CLASSIC TARSAL TUNNEL SYNDROME

Classic or proximal tarsal tunnel syndrome is the entity to which many clinicians refer, when the term, *tarsal tunnel*, is used. It is an entrapment syndrome of the entire tibial nerve behind the medial malleolus and under the flexor retinaculum or laciniate ligament. The deep and superficial aponeuroses of the leg form the ligament, which is closely attached also to the sheaths of the three adjacent flexor tendons—the

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posterior tibial, the flexor digitorum, and the flexor hallucis. The syndrome was described by Kopell and Thompson[1] in 1960 and then named by both Keck[2] and Lam[3] in 1962.

The clinical presentation is typically of posteromedial pain, tenderness posteromedially over the nerve, a positive Tinel sign in some instances, and in some cases bulging of the retinaculum. In some patients, there may be bona fide neurogenic signs, including both the sensation of numbness and actual hypoesthesia and clawing of the toes. Radiographs, in particular a CT scan, may reveal a lesion, but MRI and/or ultrasound imaging may be more helpful. A nerve conduction study may demonstrate slowing of conduction of the nerve, and electromyography of the intrinsic muscles—abductor hallucis, abductor digiti quinti, and interossei—may also be positive.

Space-occupying lesions have been commonly cited in the cause of this problem. These include ganglia from the subtalar joint or tendon sheaths, lipomas, accessory muscles, tenosynovitis of the adjacent flexors (even though they are within independent sheaths), varicose veins, a bone spicule from an adjacent fracture, foreign bodies. Iatrogenic causes, including osteotomies of the os calcis with inadvertent deep penetration medially or with fixation hardware may be implicated as "lesions." Classic presentations include the following cases.

*A middle-aged physician presented with posteromedial ankle pain, no tenderness, and no neurologic deficits. Radiographs and an initial MRI were obtained. Both were interpreted as negative for pathology. The diagnosis was uncertain and there was little evidence. For more than 2 years, the patient treated himself with analgesics and anti-inflammatory drugs, assuming this to be some form of tendon problem. Although mention was made of tarsal tunnel syndrome, the physical findings were too vague to suggest surgical intervention. Two years later, with symptoms interfering with his work and some complaints of rest pain, another MRI, with greater sensitivity, was done. Again, this was interpreted as normal. With increased interest in ultrasound imaging for assessing nerves, a study with this methodology was done and was interpreted as showing a lipoma lying under the nerve (**Fig. 1**A). Surgical decompression and excision of the lipoma confirmed the diagnosis and the patient obtained full relief (see **Fig. 1**B–E). Further review of the MRI reveled that the now obvious lesion had been interpreted as normal fat.*

A woman in her early 20s presented with a similar history. She was found on MRI and ultrasound imaging to have an accessory muscle, the flexor digitorum accessorious, and had a decompression and excision of the muscle; she had slow but complete relief. We have also removed a small number of ganglions from the tunnel, but these have presented typically more distally and are described later.

Surgical management of the classic tarsal tunnel and its various causes requires a release of the laciniate ligament and appropriate management of the underlying lesion. Throughout the literature, there are reports of variable outcomes of the surgical procedure, unlike the results of carpal tunnel release, which, although reliable in classic postmenopausal women with the entity, gives variable results in tarsal tunnel release. My opinion and experience with the classic tarsal tunnel outcomes is that when the space-occupying lesion is discrete, as in the cases described previously, and anatomic damage to the nerve does not appear grossly, the anticipation should be for full relief and recovery. When there is obvious damage to the nerve from a fracture or osteotomy, the nerve recovers in a variable manner. When the diagnosis is made without good objective data and the source of compression is not clear, the outcomes are not favorable.

Fig. 1. (*A*) Ultrasound study showing lipomas lying under the tibial nerve in the tarsal tunnel. (*B*) Incision marking over classic tarsal tunnel. (*C*) Lipoma (at tip of scissors) lying under the tibial nerve. (*D*) Lipoma dissected from under the nerve. (*E*) Lipoma specimen.

DISTAL TARSAL TUNNEL SYNDROME

Distal tarsal tunnel syndrome was first described by Heimkes and colleagues[4] in 1987 and is a fairly common entity, especially combined with chronic plantar fasciitis[5] and on occasion with posterior tibial tendon dysfunction.[6] This condition involves irritation to the terminal branches of the tibial nerve, typically the medial plantar, lateral plantar, and medial calcaneal nerves. It may involve combinations of these or only the first branch of the lateral plantar, the nerve to the abductor digiti quinti. The most commonly involved of the branches is the lateral plantar.

Anatomy

As the tibial nerve travels distally with the posterior tibial artery and vena comitans, it lies plantar to these structures and slightly deeper. The vessels are easily seen just under the laciniate with a darker blue appearance of the veins and a paler blue appearance of the thicker walled artery, lying between the veins. The nerve has a whitish appearance with striations. At variable locations, but typically just under the upper edge of the abductor hallucis muscle, the nerve gives off its various branches (**Fig. 2**).

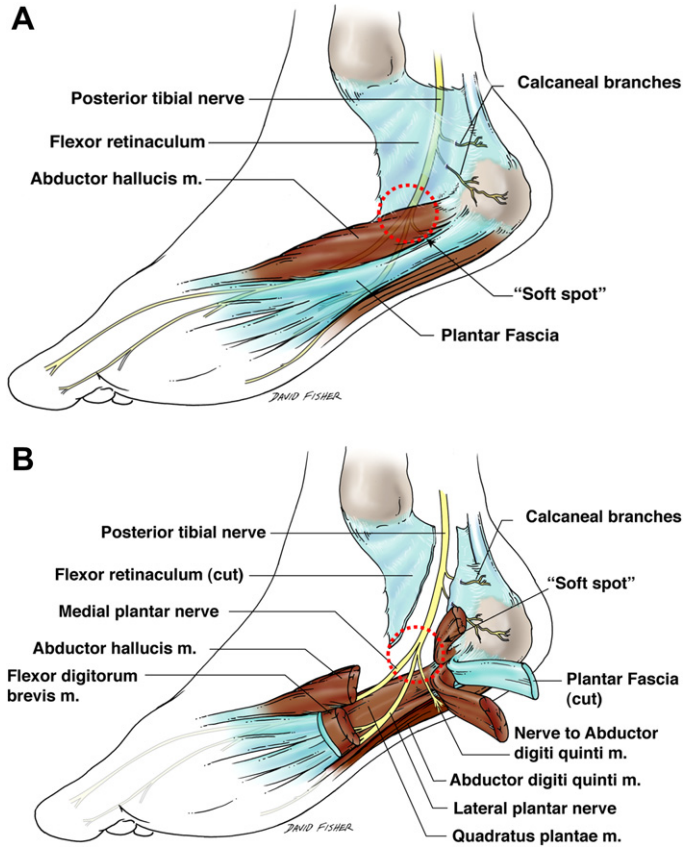

Fig. 2. (*A*) Artist's depiction of the tibial nerve passing under the abductor hallucis. (*B*) Artist's depiction of the tibial nerve and branches in the distal tarsal tunnel. Note the soft spot. (*Courtesy of* John S. Gould, MD.)

Calcaneal branch

Just before passing under the upper edge of the abductor hallucis, the nerve gives off the calcaneal branch or branches, which pass posteriorly, then distally, in the subcutaneous tissue to the skin of the heel. On occasion, the calcaneal branch may emerge under the upper edge of the muscle and then pierce the muscle and its fascia to enter the subcutaneous tissue.

First branch of the lateral plantar

Just under the upper edge of the abductor, the first branch of the lateral plantar is given off posteriorly. On occasion, it may branch from the lateral plantar portion of the main tibial nerve before the upper edge of the abductor but still travels under the muscle with the lateral plantar. The first branch travels under the abductor hallucis and its deep fascia and over the medial fascia of the quadratus plantae. It passes over the edge of the quadratus fascia and under the medial edge of the plantar fascia, then continues transversely across the heel under the flexor digitorum brevis muscle and sends a sensory branch to the central heel skin, and terminates in the muscle of the abductor digiti quinti.

Lateral plantar nerve

The lateral plantar nerve continues distally in the tunnel but is more anterior than its first branch, passing under the deep fascia of the abductor hallucis, under the medial edge of the plantar fascia, and over the quadratus plantae and its overlying fascia and then turning distally under the flexor digitorum brevis muscle, emerging under the plantar fascia to form the intermetatarsal nerve to the 4-5 interspace and a branch to the 3-4 intermetatarsal nerve. It also gives off motor branches the intrinsics.

Medial plantar nerve

The medial plantar nerve emerges typically from the tibial under the abductor hallucis muscle and travels under it with the medial plantar artery and veins, innervating the abductor and then terminating under the plantar fascia into the intermetatarsal nerves to the 1-2, 2-3, and 3-4 interspaces and motor branches to the interossei and lumbricals. In the longitudinal arch subcutaneous tissue, the nerve lies medial to but adjacent to the flexor digitorum and flexor hallucis tendons.

Pathophysiology

Several causes and mechanisms may create compression of the distal tibial nerve and its branches. A space-occupying lesion, such as a ganglion, may involve the lateral and medial plantar nerves with the lesion emerging from the subtalar or talonavicular joints. A fracture of the medial wall of the calcaneus may cause compression. Neurilemmomas and neurofibromas have been noted to affect these nerves. Direct trauma to the heel and surgical misadventures may affect the calcaneal branches. Inflammatory conditions leading to tenosynovitis of the flexor hallucis and/or flexor digitorum may affect the medial plantar nerve as well as harvesting of the flexor hallucis in this area of the longitudinal arch for a tendon transfer. The more typical cause, however, is traction neuritis of the lateral plantar and first branch of the lateral plantar nerve.

Baxter and Thigpen[7] in 1984 attributed "central heel pad syndrome" in runners to involvement of this nerve branch and suggested release of the deep fascia of the abductor hallucis and medial edge of the plantar fascia to relieve this problem. In this country, this nerve branch is commonly known as Baxter nerve. Rondhuis and Huson[8] in 1986 described what they considered compression of the first branch of the lateral plantar nerve and its association with heel pain. Lau and Daniels,[9] in a cadaver study, demonstrated that with selective division of the supporting structures of the longitudinal arch—the plantar fascia, posterior tibial tendon, and interosseous ligaments—increased traction would occur to the nerve and they suggested traction neuritis as a possible cause of distal tarsal tunnel symptoms in conditions that resulted in arch lowering or collapse. Labib and colleagues[6] in 2002 reported the association with posterior tibial tendon dysfunction. DiGiovanni and Gould, in several reports and publications,[5,10] described the association of distal tarsal tunnel with chronic plantar fasciitis, their approach for decompression, and the outcome of the procedures. The concept for the release is based on this literature suggesting the traction origin.

History and Physical Findings

The typical patient complaint is pain in the plantar heel, often in the posteromedial aspect of the heel and ankle and sometimes in the longitudinal arch. The history seems similar to plantar fasciitis and is often wrongly diagnosed and treated.

Plantar fasciitis is a distinct entity involving the origin of the plantar fascia on the calcaneus, is an enthesopathy, and presents as such. Pain is experienced in the plantar heel with the first step in the morning or when arising during the day from

a resting position. The pain remits after a few steps and does not recur with continued walking. The physical finding is discrete tenderness on the medial tubercle of the calcaneus and essentially there alone. The natural course of that disease is that it is self-limited and that it resolves particularly with stretching exercises, with or without simple over-the-counter arch support.

Tarsal tunnel syndrome, as indicated previously, has a broader area of pain involvement and usually begins with prolonged walking, becoming worse the longer one walks. Unlike plantar fasciitis, the pain does not remit spontaneously or immediately with rest. The pain remits more gradually after non–weight bearing. This more gradual resolution of the pain is described by my colleagues and me as "afterburn." The symptoms may also be even more typically neurogenic, with paresthesias and rest or night pain. These more typical neurogenic symptoms, however, are not always present, although the afterburn typically is. More severe neurologic deficits, such as numbness and/or motor deficits, as manifested by clawing of the toes, are not common with tarsal tunnel syndrome and should bring to mind other diagnoses, including generalized neuropathy, radiculopathy, or central lesions, such as multiple sclerosis.

The physical findings in tarsal tunnel syndrome include point tenderness on the posteromedial heel in the soft spot (**Fig. 3**) at the lower edge of the abductor hallucis where the neurovascular structures enter the foot. As the medial border of the heel is palpated from posteriorly and proximal to distal, the probing finger falls into the soft area (described previously). There may be a Tinel sign over this area and pain may radiate proximally and distally. Palpation of the nerve proximally and in the longitudinal arch may also produce tenderness. In addition, there may be tenderness in each of the intermetatarsal spaces distally, suggesting intermetatarsal neuritis or a Morton neuroma. When the history is of heel pain, however, this distal tenderness represents nerve irritability, not an accompanying distal lesion. If the complaint is of metatarsal pain and the tenderness is also distal, then the lesion is distal as well. With intermetatarsal neuritis or Morton neuroma, the complaint and tenderness are distal but the nerve may also be sensitive proximally or over the soft spot, but the lesion is distal and the patient does not complain of heel pain. This emphasizes the mandatory correlation of the history and the physical.

Distal tarsal tunnel with chronic plantar fasciitis[11] is a common entity and often extraordinarily confusing to clinicians. In truth, it is simpler than it seems. The history and the physical findings combine both entities. The typical history is of plantar fasciitis, which has remitted either with standard noninterventional care or, more typically,

Fig. 3. The clinical soft spot where the tibial nerve passes from the ankle to the foot.

with steroid injections to the origin of the plantar fascia. The earlier treatment had been successful, but there is a recurrence. The recurrence may not respond to stretching, injections, or simple inserts.

When the history is carefully obtained, it has changed since the earlier episode. Although there may still be pain with first step in the morning, it may not remit with continued walking or immediately with rest. The afterburn (described previously) may exist. Another presentation is pain with the first step, remission with walking, and then recurrence with prolonged walking and very slow remission of pain afterwards. Finally, there may be rest or night pain, accentuated by pain with first step or walking in general. The physical findings include tenderness over the nerve, over the soft spot, and at the origin of the plantar fascia on the medial tubercle.

Another finding, which validates the impression that this is a traction phenomenon, is that the plantar fascia is attenuated or ruptured. When the ankle is dorsiflexed to neutral or beyond and the great toe is also dorsiflexed, the plantar fascia is placed on stretch. The medial border of the fascia can be palpated with the opposite hand from the metatarsophalangeal joint of the great toe to the heel. When the fascia is attenuated, it becomes less distinct, particularly compared with the normal side, and, in the event of frank rupture, it cannot be felt at all or minimally at best.

Additional Diagnostic Studies

Imaging studies should be obtained to rule out fractures, in particular stress fractures, or other bone lesions. CT can demonstrate such pathology in more detail. Radioactive isotope bone scanning is nonspecific, leading to a variety of diagnoses, such as stress fractures or osteomyelits, which need correlation with the history and physical and more specific objective testing. MRI is extremely sensitive and shows a space-occupying lesion or a subtle stress fracture that needs to be confirmed with CT. It may also show a defect or thickening in the plantar fascia, suggesting a new or chronic rupture, or simply signal changes at the origin of the plantar fascia, often wrongly interpreted as osteomyelitis or stress fracture, but which may only represent the inflammatory reaction of an enthesopathy. There has been a suggestion that signal changes may be found in nerves with traction neuritis as well, but this has not been a consistent finding (Lopez-Ben R, Department of Radiology, Division of Orthopaedic Surgery, University of Alabama at Birmingham, personal communication, 2010).

Electrodiagnostic studies do not confirm conduction delays nearly as frequently in tarsal tunnel syndrome as with the carpal tunnel syndrome, and a positive test is not considered mandatory for surgical intervention as it is for the upper-extremity condition. Electromyography, however, often shows changes in the abductor hallucis muscle and, when tested, in the abductor digiti quinti.[12] Moreover, the testing is essential when there is any suggestion of generalized neuropathy or radiculopathy. With a strong history and physical findings that correlate with distal tarsal tunnel and the failure of a patient to respond to conservative therapy, a negative electrodiagnostic testing does not provide a contraindication for surgery.

Nonoperative Treatment

The term, *nonoperative treatment*, rather than *conservative treatment*, is used to suggest that there are indications where surgery rather than a nonoperative approach may be indicated and be the more conservative therapy.

For nonoperative treatment, my colleagues and I use a custom total contact insert with a posteromedial nerve relief channel (**Fig. 4**).[13] The channel is placed in the medial wall of the heel component and to the midline in the plantar area. The channel corresponds to the anatomy of the lateral plantar nerve and its first branch and is placed

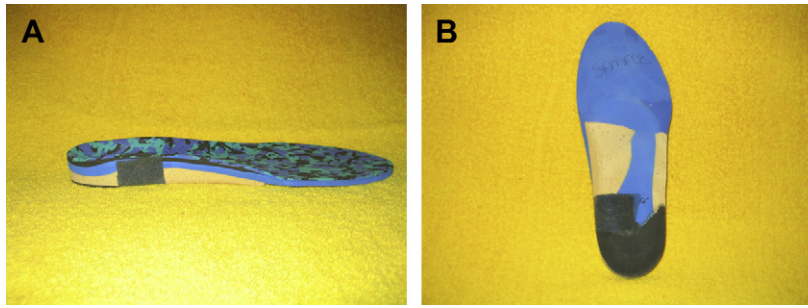

Fig. 4. (*A*) Total contact insert with the posteromedial nerve relief channel (medial view). (*B*) Total contact insert with nerve relief channel extending around plantar medial side.

within the cork, which is used for posting the heel and longitudinal arch. The channel is filled with a viscoelastic polymer. When a patient's diagnosis is central heel pad syndrome or the tenderness is also in the central heel pad in distal tarsal tunnel, the channel is extended more posteriorly both medially and plantarward. When the medial plantar nerve seems to be involved, the distal edge of the channel is feathered and the channel may be continued along the medial longitudinal arch. When a distal tarsal tunnel is treated with a more standard insert, without the channel, the patient often states that the insert makes his problem worse. Adding the channel may be dramatic in providing relief. Although there are no hard data to confirm this impression, my colleagues and I think that approximately two-thirds of never-operated patients respond positively to the use of this insert alone. If patients also have residual plantar fasciitis symptoms, my colleagues and I recommend stretching exercises, as described by DiGiovanni and colleagues.[14]

My colleagues and I have used systemic nonsteroidal anti-inflammatory drugs for patients with the plantar fascia component; oral gabapentin and other drugs to ameliorate neurogenic symptoms, with some success; topical anti-inflammatory drugs and nerve pain relievers; and iontophoresis as administered by physical therapy. Patients are urged to avoid modalities, such as heat, cold, and vibration, which are irritating to nerves, negating the use of hot packs, whirlpool baths, and ultrasound, including phonophoresis.

Operative Intervention

Operative intervention is based on two concepts: (1) treating the residuals of chronic plantar fasciitis and (2) the effort to decompress the tibial nerve and its branches, with emphasis on the lateral plantar and its first branch and the medial calcaneal and the medial plantar when indicated. Medial calcaneal decompression is usually done in recurrent surgery and is discussed in that article by John S. Gould on The Failed Tarsal Tunnel Release. Medial plantar decompression is not routinely done but is discussed. Treatment of the chronic plantar fasciitis component is based on the observation that chronic ruptures of this tissue tend to remain painful with chronic tenderness of the fascia at its origin or with the development of neurogenic symptoms. Complete release of this fascia, as with partial ruptures of other collagen structures, such as tendons (eg, long head of the biceps or flexor carpi radialis), provides relief. The decompression of the nerve is based on the concept of traction of the nerve with the fulcrum of pressure as it passes under the distal edge of the deep fascia of the abductor hallucis and medial edge of the plantar fascia and over the fibers of the quadratus plantae fascia. The nerve can be observed as tented

under and over these structures at the soft spot as it makes a significant turn from the ankle into the foot. The releases of the structures allow the nerve to lie freely at this angle.

Because partial rupture of the plantar fascia seems to remain symptomatic, if it is necessary to release some of the plantar fascia to relieve the nerve, my colleagues and I have been hesitant to not release it all. Concern over dorsal arch and lateral border pain after release of the entire plantar fascia has led to a postoperative regimen of non–weight bearing for 4 weeks and the use of the insert postoperatively during the first year. My colleagues' and my experience has shown that this nonweight bearing and use of the insert will obviate the dorsal and lateral foot pain after this procedure.

Operative Indications

Most patients with a diagnosis of distal tarsal tunnel syndrome with or without chronic plantar fasciitis deserve a trial of nonoperative treatment. If the history and physical are compatible with the diagnosis and there is no indication of radiculopathy or generalized neuropathy or that and other possible diagnoses have been appropriately ruled out, a 6-week trial of treatment with the total contact insert, as described previously, is indicated.

If, at 6 weeks, patients are somewhat improved, this approach is continued for another 6 weeks. As long as improvement is seen, my colleagues and I continue nonoperatively.

If patients are not improved at 6 weeks, initially, the orthotic device is examined to be certain it has been properly made and fits well. Patients are carefully asked if they are using it constantly when up and about. If patients have been compliant and the device is appropriate, diagnostic points are reviewed, and, if still consistent with the initial impression, the surgery is recommended.

If patients were improving with the insert and have reached a plateau, my colleagues and I go through the same procedure and also ask if the patients are satisfied with continuing to use the insert rather than having surgery.

Some patients present with a history and physical totally compatible with the distal tarsal tunnel diagnosis and a significant period of nonoperative treatment, which may be similar to that of my colleagues' and mine but perhaps without the exact regimen. In these cases, I discuss both approaches with the patients. I insist they obtain the type of insert my colleagues and I recommend or modify the one they have to include the nerve relief channel. Patients are usually scheduled for surgery and the insert obtained for them to start to wear. I tell the patients my colleagues and I will proceed as scheduled unless they are so improved with the new inserts that they wish to delay the surgery.

Operative Technique

The surgery is performed under general or regional anesthesia, but not local, which obscures the anatomy. It is usually an outpatient procedure.[16] A thigh or high calf tourniquet is used along with good lighting and magnifying loupes (3.5× or 4.5×). With the patient in the supine position and the leg externally rotated, a curvilinear incision is begun midway between the posterior edge of the medial malleolus and the medial border of the Achilles tendon. The incision continues distally, curving gently forward to cross the soft spot and then continues across the plantar skin just distal to the heel pad approximately three-fourths of the distance across the plantar surface (**Fig. 5**A).

The vascular bundle is noted under the laciniate ligament. The ligament is divided over the bundle distally to the abductor hallucis superficial fascia, which is also

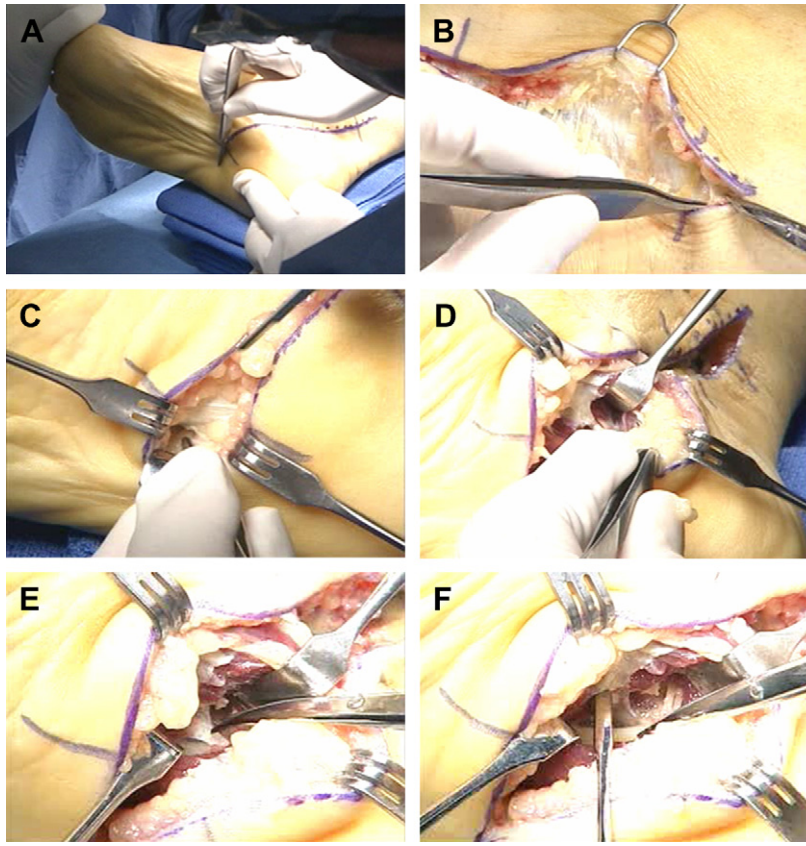

Fig. 5. (*A*) The posteromedial incision used for the tarsal tunnel release. (*B*) The laciniate ligament. (*C*) The convex plantar fascia. (*D*) Release of the deep fascia of the abductor hallucis. (*E*) The interval between the abductor hallucis and the flexor digitorum brevis. (*F*) The lateral plantar nerve.

divided, watching for the occasional calcaneal branch, which may pierce it in its proximal substance (see **Fig. 5**B). The subcutaneous fat over the plantar fascia is divided and deeper retractors, such as Senn, are inserted. The convex shape of the plantar fascia is noted and a small angled retractor is placed at the medial edge of the abductor digiti quinti. The entire plantar fascia is divided from the abductor hallucis to the abductor digiti quinti (see **Fig. 5**C).

Dissecting under the abductor hallucis, the muscle is teased away from its deep fascia, approaching the muscle either proximally or distally. A small right angle is inserted into the space between the muscle and fascia, and the deep fascia is divided with tenotomy scissors (see **Fig. 5**D). The division is begun either proximally or distally and then completed from the other direction.

Across from the plantar edge of the abductor hallucis, the muscle of the flexor digitorum brevis is lifted and a deep fascia of variable thickness is also divided. Care is taken throughout this process to avoid lacerating any of the small branches of the posterior tibial artery or veins. If this occurs, I divide the entire abductor hallucis muscle or even the medial edge of the flexor brevis with a cutting cautery to gain

good access to the bleeder. To stop this bleeding, I use either bipolar cautery or a small Ligaclip.

Assuming that the incision is properly placed and the abductor is intact, I open the interval between the abductor hallucis and the flexor digitorum brevis with a small self-retaining retractor (Weitlaner) (see **Fig. 5**E). Within this interval are the vascular structures, the first branch of the lateral plantar nerve and the lateral plantar nerve. The first branch lies closer to the posterior heel, the veins with the artery in the middle lie more anteriorly, followed by the lateral plantar nerve, which lies more anteriorly and a little deeper than the vascular structures. Filmy fascia may be found over the nerve, which, with dissection, becomes distinct as a white serrated tubular structure (see **Fig. 5**F).

At this point, the medial plantar nerve has divided off more proximally and is traveling anteriorly under the abductor muscle. Moving the lateral plantar nerve aside with the tip of the tenotomy scissors and a fine forceps, the underlying fibers of the quadratus plantae are visualized and divided.

If the entire abductor hallucis muscle is divided, all of the anatomy is easily visible, but this is not necessary unless vascular access for bleeding is needed; the surgeon is doing a recurrent case that requires full visualization of the nerve; decompression of the medial plantar nerve is required; or the surgeon is disoriented and better visualization of the neurovascular anatomy is needed. I usually do not release the muscle and believe this adds to the morbidity of potentially more scarring.

Additional decompression is done when there is a septum between the vessels and the lateral plantar nerve. This may be seen looking under the abductor muscle and is suspected when seen distally in the interval between the abductor and flexor. The muscle may have to be divided when this is present.

When the medial plantar nerve is involved, I use the same initial approach, divide the muscle, and then, noting exactly where the medial neurovascular bundle is running, make a skin incision over this pathway and decompress the vessels and nerve along this route through the muscle.

The closure consists of dissolving suture (4-0 Monocryl) for the subcutaneous skin of the ankle, with 4-0 nylon on the skin, and skin-only closure with 3-0 nylon for the glabrous skin area plantarly and distally medially.

Postoperatively, the patient is placed in a soft bulky dressing and kept non–weight bearing for the first 2 weeks. Sutures are removed at this time and non–weight bearing is continued for 2 more weeks. No dressing is needed and the patient can bathe the foot. Motion of the foot and toes is encouraged to maintain the gliding of the nerve. At 4 weeks, the patient is allowed to walk with the total contact insert in a shoe or sandal, which is continued for 9 months.

Outcomes

DiGiovanni and Gould[15] reported an 82% rate of total recovery in a group of primary releases done and followed for at least 2 years, with no recurrences and no patients made worse. In a group of 104 feet in 92 patients operated at the American Sports Medicine Institute, between 1996 and 2000, Hollis and colleagues (Hollis M, Ferguson A, Gould JS, et al. American sports medicine institute review of 104 feet [92 patients] following the complete plantar fascia and tarsal tunnel release between 1996–2000, unpublished data, 2000) found that the average time for patients to reach a plateau of recovery was 19.6 months, with a range of 6 months to 2.5 years. A further report from Gould and DiGiovanni[16] has confirmed continued success with this operative approach with excellent long-term outcomes. The approaches and outcomes related to relapsed, recurrent, or failed procedures are reported by John S. Gould elsewhere in this issue in a subsequent article.

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