

Neurolysis for Failed Tarsal Tunnel Surgery

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ABSTRACT

The purpose of the present study was to investigate the causes of failure after tarsal tunnel release and the operative findings in the secondary interventions and the outcomes. The data from 8 patients who had undergone revision surgery for failed tarsal tunnel release at least 12 months earlier were evaluated retrospectively. Only the patients with idiopathic tarsal tunnel syndrome were included, and all had unilateral symptoms. Neurophysiologic tests confirmed the clinical diagnosis of failed tarsal tunnel release in all patients. Magnetic resonance imaging revealed varicose veins within the tarsal tunnel in 1 patient (12.5%) and tenosynovitis in another (12.5%). Open tarsal tunnel release was performed in all patients, and the tibialis posterior nerve, medial and lateral plantar nerves (including the first branch of the lateral plantar nerve), and medial calcaneal nerve were released in their respective tunnels, and the septum between the tunnels was resected. The outcomes were assessed according to subjective patient satisfaction as excellent, good, fair, or poor. During revision surgery, insufficient release of the tarsal tunnel, especially distally, was observed in all the patients, and fibrosis of the tibialis posterior nerve was present in 1 (12.5%). The outcomes according to subjective patient satisfaction were excellent in 5 (62.5%), good in 2 (25%), and fair in 1 (12.5%). The fair outcome was obtained in the patient with fibrosis of the nerve. Insufficient release of the tarsal tunnel was the main cause of failed tarsal tunnel release. Releasing the 4 distinct tunnels and permitting immediate mobilization provided satisfactory results in patients with failed tarsal tunnel release.

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Tarsal tunnel syndrome (TTS) is the clinical description of entrapment of the tibialis posterior nerve and/or its branches within the fibro-osseous tarsal tunnel under the flexor retinaculum on the medial side of the ankle. It is characterized by pain, altered sensation, paresthesia affecting the plantar aspect of the foot, and motor weakness of the intrinsic foot muscles. It is not as prevalent as the other entrapment neuropathies, but it has also been regularly underdiagnosed (1). Accurate diagnosis of TTS is challenging, because false-negative results from neurophysiologic studies have been common, contributing to the underdiagnosis of the condition (1,2).

Many causes of TTS are known, including trauma, foot deformities, and space-occupying lesions (i.e., ganglia, lipomas, accessory muscles, varicosities, and tenosynovitis), but still, many cases will be idiopathic (3). The choice of treatment is operative decompression when

conservative methods have failed to improve the symptoms. Most patients' symptoms will disappear with operative decompression; however, the symptoms can persist, recur, or become worse in some. Throughout the published data, reports have been made of the variable outcomes of tarsal tunnel release (TTR) (2,4,5). The failure rate of TTR has ranged from 4% to 56% (1,2,6,7).

Failure of operative decompression of the tarsal tunnel can result from many causes. The most common causes have been associated space-occupying lesions in the tarsal tunnel, in which case TTR alone will not suffice. However, many of the failures have resulted from a lack of appreciation of the involved anatomy or from an inadequate technique (1,2,4,6), with insufficient TTR a frequent error. The wide variation in the success rate has also resulted from patient selection and the timing of the surgical intervention. Failed TTR is a frustrating condition, and the reoperation outcomes for failed TTR will be less predictable than those for the initial surgical treatment (4).

The primary aim of the present study was to evaluate the outcome of neurolysis for failed tarsal tunnel surgery. The secondary aim was to investigate the causes of failure. We present the clinical presentation, neurophysiologic findings, operative findings, and the outcomes of revision surgery in 8 patients with failed TTR.

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Patients and Methods

The primary aim of the present study was to evaluate the outcome of neurolysis for failed tarsal tunnel surgery. Our secondary aim was to investigate the causes of failure. The data from 8 patients (5 males [62.5%] and 3 females [37.5%]; mean age 52 years, range 40 to 63) who had undergone revision TTR for the diagnosis of failed TTS surgery were evaluated retrospectively. All the patients provided informed consent.

The patients were treated from September 2010 to May 2012 and were recruited into the study from June 2013 to August 2013. The patients had undergone primary surgery at least 12 (range 12 to 19; mean 15) months before revision TTR and reported unrelieved clinical symptoms. The mean duration of follow-up after revision surgery was 16 (range 12 to 31) months. All the patients had unilateral symptoms. Only patients with idiopathic, failed TTR were included. Those patients with previous ankle injury, metabolic disorders such as diabetes, or any type of neuropathy were excluded. None of the patients had any known associated problem that might have reasonably been responsible for the TTS.

All the primary surgeries used to treat the patients in the present investigation were performed by surgeons other than us, including 2 neurosurgeons, 1 plastic surgeon, and 5 orthopedic surgeons. Surgical incisions of different lengths and shapes were present on the medial side of the ankles or feet. In 1 patient (12.5%), only 1 transverse incision was present on the medial aspect of the foot, without proximal extension (Fig. 1). None of the patients had a history of operative site infection or delayed wound healing. Postoperatively, 3 patients (37.5%) had undergone immobilization with a splint for 3 weeks, and the remaining 5 patients (62.5%) had not borne weight on their operated foot for the first 3 to 4 weeks postoperatively.

All the patients underwent repeat evaluation clinically, radiologically, and neurophysiologically. Every patient underwent a physical examination and neurophysiologic tests of both feet. The clinical evaluation also included physical examination of the lumbar spine. The clinical diagnosis of TTS was determined by a history of pain, paresthesia, activity-related pain, and a sensory deficit on the plantar aspect of the foot, a positive Tinel's sign over the tarsal tunnel, positive dorsiflexion-eversion test results (8), and weakness of the intrinsic foot muscles (loss of toe flexor and abduction strength).

All patients reported pain over the tarsal tunnel with radiation to the plantar aspect of the foot, including the heel, that was exacerbated by activity, paresthesia, and a sensory deficit on the plantar aspect of the foot. Five patients (62.5%) reported that their symptom severity was the same as during the preoperative period, without improvement (persistent), and 3 patients (37.5%) reported increased severity



Fig. 2. Weakness of the intrinsic foot muscles as demonstrated by the lack of toe abduction in the right foot.

(worsened). Well-healed surgical incisions of different lengths and shapes were present on the medial side of the ankles or feet.

None of the patients had heel valgus or hypermobility of the ankle, and the lumbar examination findings were normal in all. All the patients experienced pain with deep palpation over the tarsal tunnel (the Valleix sign) and dysesthesia and paresthesia on the plantar aspect of the foot. Tinel's sign over the tarsal tunnel just posterior to the medial malleolus was positive in all patients, and a sensory deficit on the plantar aspect of the foot was present in 5 (62.5%). The dorsiflexion-eversion test result was positive in 3 (37.5%), and weakness of the intrinsic foot muscles was present in 1 patient (12.5%; Fig. 2). The symptoms were aggravated by standing and walking. All patients had a clinical diagnosis of failed TTR and ongoing TTS from the clinical evaluation.

All patients had anteroposterior and lateral radiographs of the ankle available, and 4 patients (50%) had magnetic resonance imaging (MRI) studies available. The radiographs revealed normal findings in all the patients. The MRI findings showed varicose veins within the tarsal tunnel (Fig. 3) in 1 patient (12.5%) and tenosynovitis (Fig. 4A and B) in 1 patient (12.5%). The former patient had been referred to a vascular surgeon for varicose vein treatment, and endovenous laser ablation was performed 6 months before secondary TTR. The patient underwent surgery because of persistent TTS symptoms. The latter patient had been treated with a splint, nonsteroidal anti-inflammatory medication, and cold pack application; revision TTR was performed because of persisting and deteriorating neurologic symptoms.

The neurophysiologic tests involved nerve conduction studies and needle electromyography of both feet. The nerve conduction studies involved measurement of the amplitude of sensory nerve action potentials, sensory nerve conduction velocity, distal motor latency (DML) of the medial and lateral plantar nerves, and motor nerve action potentials. The motor nerve action potentials of both feet were also tested and compared. The difference in the DML value of the medial and lateral plantar nerves was also considered. An amplitude of sensory nerve action potentials of more than 5 μ V, sensory nerve conduction velocity of more than 40 m/s, an DML of less than 6.2 ms of the medial plantar nerve to abductor hallucis muscle and less than 7 ms of the lateral plantar nerve to the abductor digiti quinti muscle, and an amplitude of motor nerve action potentials of more than 5 mV were considered normal. A difference in the DML value of 1 ms for the medial and lateral plantar nerves was accepted to indicate TTS. Mixed nerve conduction studies were also tested when necessary.

The pathologic findings on electromyography from the abductor hallucis and abductor digiti quinti muscles included fibrillation and positive wave activity, reduced recruitment, and abnormalities in the



Fig. 1. Transverse incision on the medial aspect of the foot (arrowheads) without proximal extension.



Fig. 3. T₂-weighted, fat-saturated, sagittal magnetic resonance image showing varicose veins within the tarsal tunnel.

configuration of the motor unit action potentials. All tests were conducted in normal clinical settings at room temperature by a single neurologist. The new neurophysiologic test findings confirmed the clinical diagnosis of failed TTR. The contralateral foot was neurophysiologically intact in all 8 patients. The patient data and neurophysiologic test results are listed in [Table](#).

Surgical Technique and Postoperative Care

All patients received combined spinal and epidural anesthesia and underwent open TTR and exploration of the tibialis posterior nerve and its branches under pneumatic tourniquet hemostasis. The procedure started with a curved medial incision centered between the medial malleolus and Achilles tendon 2 cm proximal to the medial malleolus and extending to the origin of the abductor hallucis muscle on the medial aspect of the foot. The previous incisions were used when possible; however, usually, only the distal portion required lengthening. The flexor retinaculum was divided completely from its proximal border to the level of the dorsal abductor muscle belly, which is the end of the tarsal tunnel, and the tibialis posterior nerve,

artery, and vein were identified. The tibialis posterior nerve was freed from fibrous bands, and decompression was performed distally where the tibialis posterior nerve passes under the abductor hallucis muscle ([Fig. 5](#)). The superficial abductor hallucis fascia was divided, the muscle was retracted distally, and the deep fascia of the abductor hallucis muscle was identified and released. The tunnels of the medial and lateral plantar nerves were identified and released, and the septum between the 2 tunnels was divided and resected. The proximal medial edge of the plantar fascia was released, when necessary. The first branch of the lateral plantar nerve (Baxter's nerve) was also identified and released. The calcaneal nerve branches were identified, and their tunnels were released by dividing the fascia of the abductor hallucis muscle. The tibialis posterior and medial and lateral plantar nerves were checked for internal fibrosis. If the distinct nerve fascicles could not be seen, that was interpreted as intraneural fibrosis, and a linear incision was made into the perineurium to release the fascicles. The tunnel was explored for space-occupying lesions, the pneumatic tourniquet was deflated, and the wound was closed after meticulous hemostasis.

Postoperatively, the foot was placed in a bulky dressing for 1 week, and full weightbearing was allowed immediately with crutches. Full weightbearing without crutches was permitted as tolerated in the second week. Ankle range of motion exercises were started on the first postoperative day.

The outcomes were assessed according to subjective patient satisfaction as excellent, good, fair, or poor. "Excellent" was considered to indicate complete symptom resolution, no functional limitations, and no analgesic medication requirements; "good" to indicate marked improvement with minor symptoms that did not interfere with function, and pain medication not taken regularly; "fair" to indicate some decrease in symptom intensity, with pain and functional impairment occurring regularly and pain medications regularly required; and "poor" to indicate no improvement or a worsening of symptoms ([9](#)).

Results

During surgery, insufficient distal release was observed in all patients. The superficial fascia of the abductor hallucis muscle was divided in only 2 patients (25%), and the deep abductor hallucis fascia was released in none. In the patient with the transverse incision on the medial aspect of the foot, only a 1-cm distal portion of the flexor retinaculum and the superficial fascia of the abductor hallucis muscle had been released, without releasing even the tibialis posterior nerve. In 4 patients (50%), only one half of the flexor retinaculum had been released proximally, including the patient with varicose veins and the patient with tenosynovitis. This partial division of the flexor

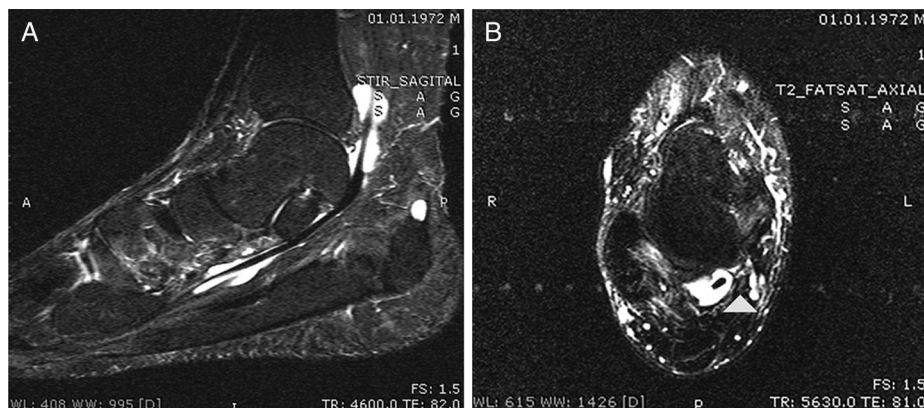


Fig. 4. (A) T₂-weighted, fat-saturated, sagittal magnetic resonance image showing tenosynovitis of the flexor digitorum longus tendon within the tarsal tunnel. (B) T₂-weighted, fat-saturated, axial magnetic resonance image showing tenosynovitis of the flexor digitorum longus tendon within the tarsal tunnel compressing the tibialis posterior nerve (*arrowhead*).

Table
Patient data

Patient No.	Age (yr)/Sex	Side	Primary Surgery (mo)	Follow-up (mo)	Amplitude of SNAP (mV) (normal 5 to 15 mV)	SNCV (m/s) (normal 40 m/s)	MNAP (normal 5 to 15 mV)	DML (ms)		EMG	MRI
								MPN to Abductor Hallucis (normal <6.2 ms)	LPN to Abductor Digiti Quinti (normal <7 ms)		
1	63/M	Left	19	14	2	31	2	4.9	5	Pathologic	No
2	47/F	Right	18	18	4	36	5	5.3	5.8	Pathologic	No
3	60/M	Left	17	16	4	35	5	5.1	5	Pathologic	Yes, no pathologic findings
4	50/F	Right	14	12	4	34	4	4.8	5	Pathologic	Yes, varicose veins
5	55/F	Left	14	12	3	32	4	4.8	6	Pathologic	No
6	40/M	Right	13	13	3	35	3	5	5.3	Pathologic	Yes, tenosynovitis
7	52/M	Left	13	12	4	38	4	5	5.2	Pathologic	No
8	49/M	Right	12	31	Absent	Absent	2	4.1	5	Pathologic	Yes, no pathologic findings

Abbreviations: DML, distal motor latency; EMG, electromyography; F, female; LPN, lateral plantar nerve; M, male; MNAP, motor nerve action potentials; MPN, medial plantar nerve; MRI, magnetic resonance imaging; SNAP, sensory nerve action potentials; SNCV, sensory nerve conduction velocity.

retinaculum had produced increased compression on the nerve by the narrowed retinaculum, and the 3 patients (37.5%) who had reported increased symptom severity after the index operation were among these 4 patients. The medial and lateral plantar nerves had not been released in their respective tunnels in any of the patients.

Varicose veins were apparent in the patient in whom MRI had revealed varicosities within the tarsal tunnel preoperatively. The varicosities were ligated and distal release was performed in this patient.

In 1 patient (12.5%), some scarring and visible fibrosis of the main trunk of the tibialis posterior nerve was present, and a linear incision was made into the perineurium, releasing the circumferential fibrosis. This patient was the third patient in whom partial division of the flexor retinaculum had produced increased compression on the nerve.

Symptomatic improvement was obtained in all 8 patients. The outcomes according to subjective patient satisfaction were excellent in 5 (62.5%), good in 2 (25%), and fair in 1 (12.5%). The fair outcome was obtained in the patient with fibrosis of the nerve. The fair and good outcomes were also obtained in those patients in whom partial division of the flexor retinaculum had produced increased compression on the nerve. No early or late complications developed.

Discussion

TTS is a relatively uncommon entrapment neuropathy, although the published data contain no precise estimate of its prevalence. We believe that the condition has been underdiagnosed and frequently misdiagnosed. The choice of treatment, just as for other entrapment neuropathies, is surgical decompression when conservative methods have failed to improve the symptoms. Most patients' symptoms will disappear or improve with surgical decompression. The outcomes of TTR

have varied, with failure rates of 4% to 56% reported (1,2,4,7), particularly in patients in whom a specific etiology could not be identified (10). We believe that these rates do not always reflect the “true” failure rate, because many studies have been of varieties of surgical release, including extensile release (11), the 4-medial ankle tunnel release (tibialis posterior, medial and lateral plantar nerves, and calcaneal nerve) (1,3,7,12,13), proximal-only TTR (2,3), distal-only TTR (6,14), selective nerve release (2,4,6,15), and shorter incisions (16). Again, only a few studies have been published and have included small numbers of patients with failed TTR, variable results, and a wide range of surgical procedures (4). We identified no reports on the rate of second surgery for failed TTR.

Failure of surgical decompression of the tarsal tunnel can result from many causes. Failed TTR has 3 distinct presentations (4,17):

1. Persistent—the patient shows no improvement
2. Recurrent—the patient experiences temporary relief for a period, with subsequent recurrence of the symptoms
3. Worsening after the initial procedure

Barker et al (4) hypothesized that patients with recurrent and persistent TTS will have had incomplete release of the medial plantar, lateral plantar, and/or calcaneal tunnels and that patients with worsening complaints will have a painful neuroma of the calcaneal and/or saphenous nerves, in addition to the incomplete release of these nerves. We were unable to detect a neuroma in our 3 patients in whom the symptoms had worsened after the first surgery; however, they all had undergone partial proximal division of the flexor retinaculum, which had consequently produced increased compression on the nerve. To make a distinction, the initial complaints before the index surgery must be compared with the current complaints, and metabolic disorders, such as diabetes and hypothyroidism, should be considered. The potential etiologies of failed TTR include the following:

1. An incorrect initial diagnosis of TTS
2. Missed space-occupying lesions in the tarsal tunnel
3. Inadequate release owing to a lack of understanding of the anatomy involved
4. Bleeding with subsequent scarring, leading to adhesive neuritis
5. Intraneural damage initially or postoperatively
6. Double crush syndrome
7. Idiopathic

Differentiating among these different causes of failure can guide the surgeon in the treatment strategy and increase the treatment success for this recalcitrant condition (4,17). We believe that the initial diagnosis of TTS was correct in all our patients and that inadequate release was the main reason for failure. Inadequate release was also present in the 2 patients with varicose veins and tenosynovitis, whose

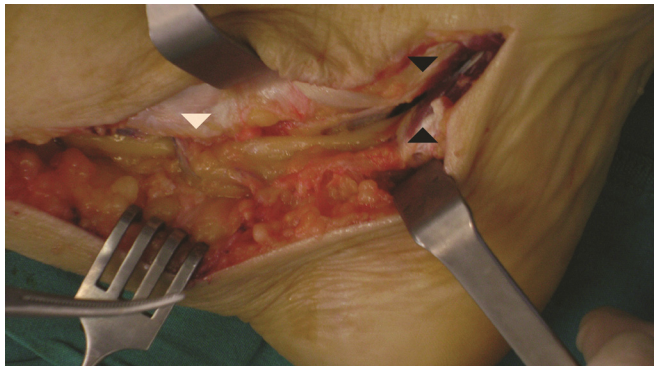


Fig. 5. The flexor retinaculum was not released. The tibialis posterior nerve was freed from fibrous bands (white arrowhead), and decompression was performed distally where the tibialis posterior nerve passes under the abductor hallucis muscle (black arrowheads).

symptoms would have been relieved if the TTR had been performed sufficiently, even in the presence of space-occupying lesions.

Although the published data on TTS presents a confusing picture of the entity itself, the etiology, and the anatomy involved, failed TTS surgery presents an even more complicated picture. The published data on failed TTR are scarce. Previously, some case reports helped us to understand the mechanism of failure after tarsal tunnel decompression (9,18–20). All these reports had focused on previously inadequate distal release or entrapment of the tibial nerve in the scar tissue with perineural fibrosis. Larger studies were reported later, again with emphasis on inadequate distal release and entrapment of the nerve in the scar tissue (4,21,22). In these studies, vein wrapping in the case of entrapment of the nerve in the scar tissue and, when present, neuroma resection were reported. However, the recommendation for either primary or secondary TTR was release of the tibial, medial plantar, lateral plantar, and calcaneal nerves in their respective tunnels and excision of the septum between the medial and lateral plantar tunnels (4,10).

We observed that in most revision TTR surgeries, the flexor retinaculum was lax and likely not the source of entrapment. This operative finding was consistent with reports that increased pressure within the tarsal tunnel was not the sole cause of recurrent TTS (3). The medial septum, which can be viewed after the abductor hallucis has been retracted is recognized as a site of compression of the medial and lateral plantar nerves (4,10). Pressure within the medial and lateral plantar nerve tunnels has been shown to be significantly greater than that in the tarsal tunnel (4,23). Therefore, decompression of the tarsal tunnel nerves passed the abductor hallucis muscle is recommended.

Another reason for the earlier failure rates was probably the use of postoperative ankle immobilization and crutches (2). Full weightbearing and ankle range of motion exercises were started immediately in our patients to prevent adherence of the nerve to the surgical site. Early mobilization will permit the nerve to glide through the operative bed (2).

Imaging studies can be critical to establishing the correct diagnosis after failed TTR, especially in cases with an initial misdiagnosis (24). Radiographs and, when necessary, computed tomography scans should be obtained to rule out fractures or other bone lesions. MRI and ultrasonography are extremely sensitive and will show space-occupying lesions, accessory muscles, varicose veins, tenosynovitis, plantar fascia pathologic features, and signal changes in the nerves. This is especially important if MRI had not been performed before the initial TTR (24). MRI has also been shown to be effective in demonstrating incomplete surgical release of the flexor retinaculum (20).

Chhabra et al (5) reported that the preoperative evaluation in 90% of patients before repeat TTR revealed nerve repeat entrapment related to focal fibrosis using high-resolution magnetic resonance neurography (MRN). The injured nerves could be visualized using MRN, with a sensitivity of 77% for the tibialis posterior nerve, 100% for the medial plantar nerve, and 100% for the lateral plantar nerve. MRN had an overall sensitivity of 91% for the presence of focal fibrosis and 67% for neuroma detection. Regional muscle denervation could also be evaluated on MRN studies. We strongly recommend MRI in the evaluation of patients with failed TTR.

The use of a neurophysiologic examination of suspected TTS and failed TTS surgery has been controversial. Some investigators have claimed that the diagnosis of TTS should be clinical, and others have considered the neurophysiologic examination to be the definitive objective tool for the diagnosis of TTS (25,26). We believe that the neurophysiologic examination is crucial in the evaluation of failed TTS surgery, especially in the differential diagnosis of other peripheral nerve pathologic features that can produce pain and paresthesia in the sole of the foot, such as lumbar radiculopathy and polyneuropathy. However, neurophysiologic examination of TTS can be difficult, and a minor deviation from the standard method can lead to false-negative results. This

technical difficulty and test results inconsistent with the clinical findings have led surgeons to conclude that the neurophysiologic examination is not sensitive in detecting TTS. They have usually diagnosed TTS without objective neurophysiologic findings. False-negative test results have been common (1). Normal motor responses, which cannot exclude TTS as a pathologic entity, could be restricted to only the sensory fibers, especially early in the disease course (25,26).

Consequently, releasing the 4 distinct tunnels and permitting immediate mobilization can provide satisfactory results after failed TTR. The preoperative evaluation should include a thorough neurophysiologic examination and imaging studies. The shortcomings of the present study were the retrospective design and the unvalidated patient subjective scoring method we used. The results of our investigation, however, can be used by future investigators interested in the surgical results of tarsal tunnel surgery.

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