

Neurovascular decompression for idiopathic tarsal tunnel syndrome: technical note

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Abstract

Objective—The surgical outcome of idiopathic tarsal tunnel syndrome (TTS) is reported to be worse than that attributable to ganglion, tarsal coalition, or tumour, and therefore further development in the surgical treatment for idiopathic TTS is considered to be necessary. Here the efficacy of neurovascular decompression for patients with idiopathic TTS is evaluated.

Methods—Twelve feet from nine patients with idiopathic TTS were treated. The patients were aged 52–78 years (mean 64.6 years), and all of them complained of pain or dysaesthesia of the sole of the foot. The posterior tibial nerve was freed from the attached arteriovenous complex (posterior tibial artery and veins). The dissected nerve had a flattened appearance in all of the patients, suggesting nerve compression by the adjacent arteriovenous complex and superficially by the flexor retinaculum. A graft of fat was inserted as both a cushion and an antiadhesive between the vessels and the nerve to achieve neurovascular decompression.

Results—Patients on whom neurovascular decompression was performed had resolution or lessening of symptoms in their feet. Neither wound infection nor recurrence of symptoms was found during the follow up period (mean 26.8 months).

Conclusion—Neurovascular compression syndrome plays a part in idiopathic TTS, and adding neurovascular decompression to resection of the flexor retinaculum is effective.

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Keywords: idiopathic tarsal tunnel syndrome; neurovascular decompression; peripheral entrapment neuropathy; surgery

Tarsal tunnel syndrome results in peripheral nerve entrapment much less often than carpal tunnel syndrome or cubital tunnel syndrome.¹ Previous investigations contain reports of cases involving no more than about 50 feet.^{1–8} The aetiological causes of the tarsal tunnel syndromes are mainly the presence of a ganglion, osseous prominence with tarsal bone coalition, trauma, varicose veins, neurinoma, hypertrophy of the flex or retinaculum, and some are idiopathic.^{1 2 6 8–11} The outcome of surgical treatment of idiopathic tarsal tunnel syndrome is generally good^{2 3 5–7}; however, patients in whom surgical treatment has resulted in only a partial improvement or no improvement have also been reported.^{2 4 6–8 10 12}

Our surgical method of adding neurovascular decompression to tarsal tunnel opening was found to be effective for idiopathic tarsal tunnel syndrome although the number of patients in which this treatment has been used is small. Here, we present our method in detail.

Patients and methods

We surgically treated 12 feet in nine patients (three men and six women) with idiopathic tarsal tunnel syndrome (table). The patients were aged 52–78 years (mean 64.6 years) at the time of treatment, and the affected foot was the right in six, left in six, and bilateral in three. All of the patients complained subjectively of pain or numbness in the sole of the foot and showed objective hypaesthesia. Sensory disturbance of the sole excluding the heel (heel sparing), which suggested a lack of involvement of the calcaneal branch of the posterior tibial nerve, was found in nine feet. Tinell’s sign at the posteroinferior point of the medial malleolus, running to the first toe, was positive in all of the feet. Posterior tibial nerve blocking in the tarsal tunnel using 3 ml 1% lidocaine⁴ resolved the sole pain or dysaesthesia in all of the patients. Sensory nerve conduction velocity (SCV) measurement was carried out in five feet.

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Summary of clinical findings in the nine patients (12 feet)

Case No	Age/Sex	Foot No	Side	Complaints/Symptoms	Tinell’s sign	Heel sparing	Duration (onset–treatment)	Surgery	Follow up	Outcome
1	56/F	1	L	Foot sole burning pain and hypaesthesia	+	+	17M	TTO only	61M	Unchanged
2	52/M	2	R	Foot sole burning pain and hypaesthesia	+	+	3M	TTO + NVD	38M	Excellent
		3	L	Foot sole burning pain and hypaesthesia	+	+	3M	TTO + NVD	37M	Excellent
3	53/F	4	R	Foot sole numbness and hypaesthesia	+	+	7M	TTO + NVD	35M	Excellent
4	52/F	5	R	Foot sole pain and hypaesthesia	+	–	16M	TTO + NVD	35M	Good
		6	L	Foot sole pain and hypaesthesia, abductor hallucis paresis	+	–	16M	TTO + NVD	34M	Excellent
5	78/F	7	R	Foot sole burning pain and hypaesthesia	+	+	5M	TTO + NVD	30M	Good
6	77/F	8	L	Foot sole burning pain and hypaesthesia	+	+	6M	TTO + NVD	12M	Fair
7	63/F	9	L	Foot sole burning pain and hypaesthesia	+	+	24M	TTO + NVD	11M	Excellent
		10	R	Foot sole burning pain and hypaesthesia	+	+	24M	TTO + NVD	10M	Excellent
8	78/M	11	L	Foot sole numbness and hypaesthesia	+	+	4M	TTO + NVD	10M	Good
9	72/M	12	R	Foot sole numbness and hypaesthesia	+	–	14M	TTO + NVD	9M	Good

M=Months; TTO=tarsal tunnel opening; NVD=neurovascular decompression.

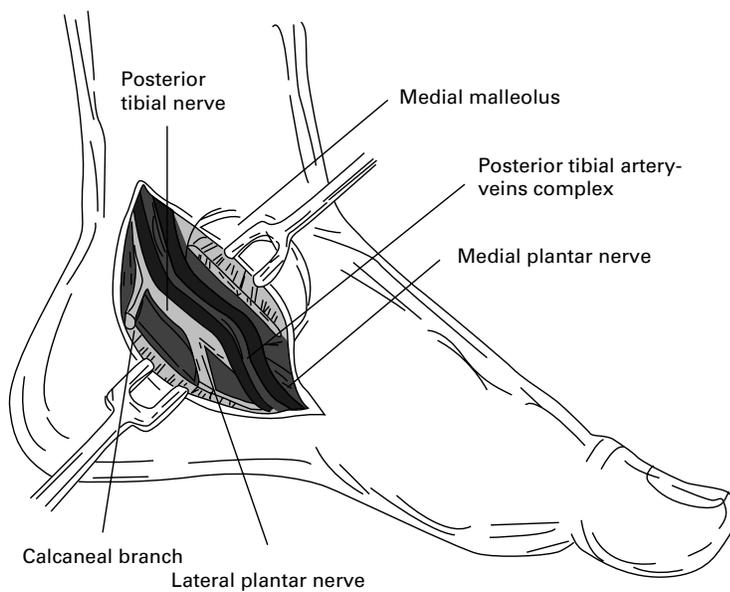


Figure 1 Diagram of operative procedure for left tarsal tunnel syndrome.

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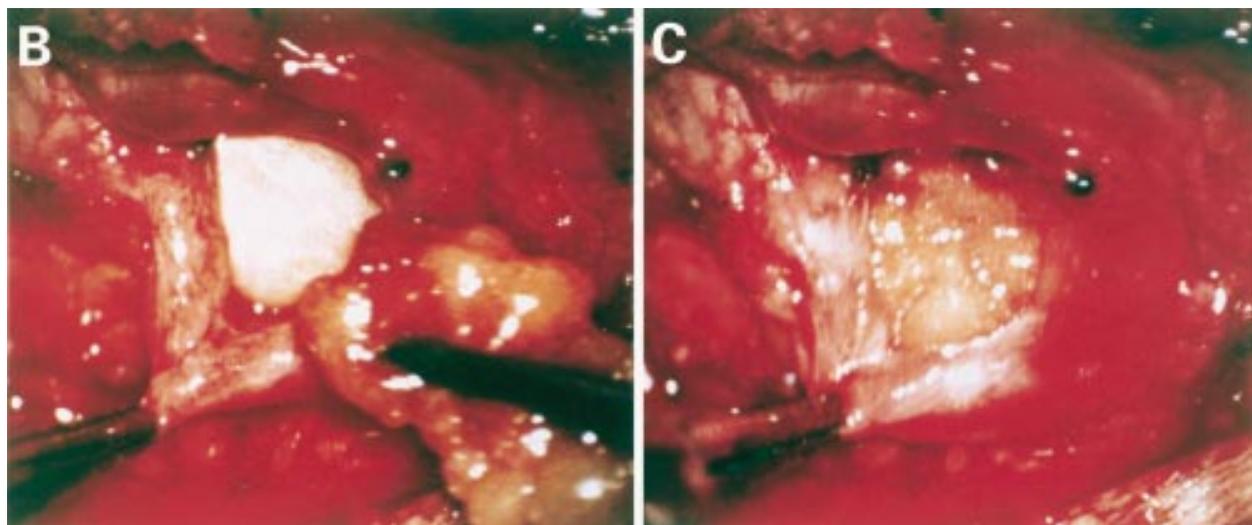
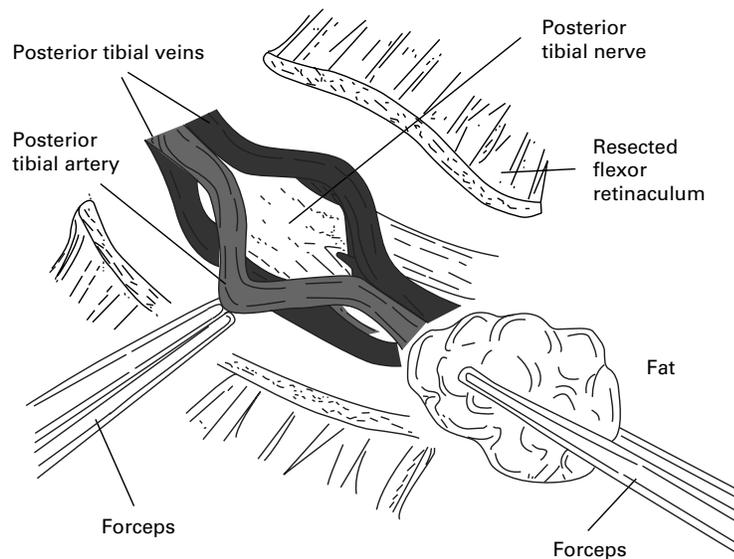


Figure 2 Intraoperative scheme and photographs of the left foot of patient 4. The posterior tibial nerve can be identified through the divided posterior tibial artery and veins. Note the grafted fat held by forceps (A, B). Fat was placed between the posterior tibial nerve and vessels (C).

Three feet showed no response and the others showed a slow SCV, less than 35 m/s. The disease was diagnosed on the basis of both the positive Tinel's sign and the effectiveness of tarsal tunnel blocking, and considering the SCV findings in a patient complaining of painful dysaesthesia localised at the sole of the foot. Surgical treatment was indicated for patients who wished it, and whose symptoms were severe and not tolerable for daily life and who had shown no response to conservative medication for at least 3 months after establishing the diagnosis.

SURGICAL METHOD

Under local anaesthesia and mild sedation, a 9 cm bow-like skin incision was made 1.5 cm below the medial malleolus. A tourniquet was not used. After full resection of the flexor retinaculum along the skin incision, the posterior tibial artery and veins (arteriovenous complex) were exposed. Under the surgical microscope, the posterior tibial nerve was identified after dissection of the posterior tibial arteriovenous complex (fig 1). The posterior tibial nerve, which is attached to the arteriovenous complex, was freed from the dissected posterior tibial artery and veins, after confirming that no nerve compressing structures such as a ganglion or an osseous prominence, had developed around the nerve.

During dissection of the nerve and vessels, the perineurium was preserved intact. The dissected nerve had a flattened appearance in all of our patients, suggesting the occurrence of nerve compression by the attached arteriovenous complex and by the flexor retinaculum superficially. A subcutaneous graft of fat was inserted between the freed vessels and nerve to allow neurovascular decompression (fig 2). A subcutaneous drain was then placed, and subcutaneous and skin closures were performed by layer leaving the resected retinaculum open.

Results

Postoperative results were evaluated based on the resolution of subjective complaints and



Figure 3 Follow up CT in patient 3. Note separated branches of the right posterior tibial nerve (arrowheads) and vessels (thin arrow) 18 months after surgery, by contrast with the neurovascular complex (thick arrow) in the left tarsal tunnel.

improvement of objective neurological symptoms. Postoperative resolution of painful dysesthesia and sensory disturbance was estimated as excellent, marked improvement of both as good, slight improvement of both as fair, and no postoperative improvement as unchanged (table). The postoperative follow up period was 9–61 months (mean 26.8 months).

Patient 1, who underwent single tarsal tunnel opening, showed no improvement of painful dysaesthesia in the sole (foot 1). The idea of reoperation was rejected. In subsequent patients, neurovascular decompression was performed in addition to tarsal tunnel opening. Burning pain and hypaesthesia of the sole were resolved in patient 2 (feet 2 and 3) and patient 7 (feet 9 and 10); numbness and hypaesthesia of the foot sole were also resolved in patient 3 (foot 4) and were markedly improved in patients 8 (foot 11) and 9 (foot 12). In patient 4, dysaesthesia and hypaesthesia of the sole were markedly improved in the right foot (foot 5), and sole dysaesthesia was resolved and the movement of the first toe was improved in the left foot (foot 6). In patient 5 (foot 7), slight dysaesthesia remained postoperatively, although foot sole pain was resolved. In patient 6 (foot 8), burning foot sole pain and hypaesthesia improved partially. Neither wound infection nor symptom recurrence was found in these patients. Long term follow up CT and MRI showed that the remaining vascular and nervous groups had separated (fig 3).

Discussion

Tarsal tunnel syndrome was first named by Keck¹³ and Lam¹⁴ in 1962 and was defined as a syndrome of compression of the posterior tibial nerve in the tarsal tunnel, which consists of the flexor retinaculum, calcaneus, and talus.^{9 15} In this syndrome, there are no sex differences,^{3 4 8} and the symptoms include dysaesthesia or pain

in the foot sole, and very often excludes the heel.^{2 4 6 8} Motor weakness is only rarely recognised.^{3 5 6}

The aetiology of tarsal tunnel syndrome has been attributed to the existence of a ganglion, an osseous prominence with tarsal bone coalition, trauma, varicose vein, hypertrophy of the flexor retinaculum, neurinoma, tenosynovitis, an accessory or hypertrophic abductor hallucis muscle, or is sometimes idiopathic.^{1 2 6 8–11} The distribution of the causes has varied among reported series. Satoh *et al*¹ reported that the causes were idiopathic in 29 (56%) of the 52 feet they investigated, a ganglion in 19%, trauma in 14%, tarsal coalition in 8%, and varicose vein in 4%. Takakura *et al*⁶ stated that a ganglion was the most frequent cause (36%), followed by tarsal coalition (30%), idiopathic (18%), and trauma (10%) in their study of 50 feet. Lam³ found no compressing mass (idiopathic) in nine out of 13 (69%) patients. By contrast, Ricciardi-Pollini *et al*⁷ found some causes in all of the eight feet they studied. Radin⁶ reviewed previously reported cases in 1983, and noted that 33 out of 74 feet (45%) were affected by idiopathic tarsal tunnel syndrome. Linscheid *et al*⁸ reported a series in which idiopathic tarsal tunnel syndrome represented only three of 24 feet (13%). However, they found an enlarged posterior tibial vein in 13 feet (54%), which was tortuous in at least six feet and varicose in three. The tortuosity they reported may be considered a normal variation, as we saw tortuous posterior tibial veins in all of our patients.

Idiopathic tarsal tunnel syndrome includes the condition in which no evident nerve compressing lesion is recognised. However, neurovascular compression may exist, as the nerve and arteriovenous complex run together in the tarsal tunnel, a different condition from that in the carpal and cubital tunnels.

In fact, the flexor retinaculum is usually much thinner in tarsal tunnel syndrome than in carpal tunnel syndrome, and resection of the tarsal flexor retinaculum is much easier than the tough carpal one, suggesting that nerve compression is not attributable to the tarsal flexor retinaculum alone. The posterior tibial nerve and arteriovenous complex are combined tightly together in the tarsal tunnel. Therefore, we think that it is possible for the nerve to become compressed by these vessels. In fact, after dissection of the nerve and the vessels, the nerve did have a flattened appearance in all of our patients. Linscheid *et al* and Lam suspected a mechanism of nerve compression by the engorged vein,^{3,4} suggesting the neurovascular compression that we think occurs.

DIAGNOSIS OF TARSAL TUNNEL SYNDROME

Electrophysiological studies including the terminal latency of the motor nerve, SCV, and EMG have been reported to be useful for diagnosing tarsal tunnel syndrome.^{1,2,4,5,11,12,16,17} However, these examinations tend to yield positive results less often in this disease than in other forms of peripheral entrapment neuropathy producing motor nerve symptoms such as carpal or cubital tunnel syndrome.^{1,2,4,8,11} By contrast, Tinel's sign is usually positive, and is thought to be the most reliable criterion for diagnosis.^{1,4-6,8} We consider that tarsal tunnel blocking⁴ in the presence of positive Tinel's sign is diagnostically useful with electrophysiological examinations as supportive evidence.

SURGICAL METHOD

In a patient with a ganglion or a tumour, surgical removal is usually performed.^{10,11,18} Surgical resection is usually performed for a varix,^{10,15} and bony resection for an osseous prominence with tarsal bone coalition.¹⁵ For idiopathic tarsal tunnel syndrome, several surgical methods have been used, including single resection of the flexor retinaculum (tarsal tunnel opening),^{9,10,18} nerve dissection of the posterior tibial nerve,^{5,11} and resection of the veins or arterial branches.^{4,13} In our method, neurovascular decompression is performed by inserting a graft of fat between the nerve and the vessels to act as a cushion and prevent them from adhering to each other.

The use of a tourniquet^{4,10,11} is considered to be effective for the control of intraoperative bleeding. However, this method is not advisable as it would then be impossible to estimate the degree of neurovascular compression because of the unusual arteriovenous conditions produced by the tourniquet.

Although general anaesthesia during surgical treatment of tarsal tunnel syndrome is recommended in the literature,¹⁰ we consider that a the combination of local anaesthesia and mild sedation is appropriate. In fact, even in a patient with tarsal bone coalition, who was not

included in this series, resection of an osseous prominence using a rongeur under local anaesthesia was not uncomfortable for the patient. Furthermore, local anaesthesia has an advantage in that the patient is able to warn the surgeon of radiating pain, which may save a nerve branch difficult to identify from injury. However, this advantage is reduced when using a tourniquet because of the ischaemia that occurs subsequently in the nerves of the lower foot.

SURGICAL OUTCOME

The surgical outcome is generally good.^{2,3,5-7} However, patients in whom only a partial improvement or no improvement at all have also been reported.^{2,4,6-8,10,12} In the series reported by Linscheid *et al*,⁴ the surgical outcome was less than ideal, and we speculate that the resection of the veins or arterial branches performed as part of their surgical method influenced the vascular circulation of the nerve and was therefore undesirable. Takakura *et al* found that the surgical outcome of idiopathic as well as traumatic tarsal tunnel syndrome was worse than that attributable to tarsal coalition, tumour, or ganglion.⁸ Thus, further improvement of the surgical treatment for idiopathic tarsal tunnel syndrome is considered necessary.

If during surgery, a ganglion or osseous prominence is not recognised, and no nerve contacting structures except for the arteriovenous complex are evident, then neurovascular compression should be suspected and decompression with a graft of fat should be added to the tarsal tunnel opening.

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