ANTERIOR TARSAL TUNNEL SYNDROME

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We have reviewed 10 patients treated for anterior tarsal tunnel syndrome produced by compression of the deep peroneal nerve or its branches, and we have studied the anatomy of the tunnel in 25 adult feet.

The causes of onset of the syndrome included contusion of the dorsum of the foot, tight shoe laces, talonavicular osteophytosis, ganglion, and pes cavus. The clinical signs were often diagnostic but electromyography was helpful. Operative decompression in nine feet of eight patients gave successful results at 1.5 to 4 years follow-up.

In 1963, Kopell and Thompson first identified a syndrome caused by compression of the deep peroneal nerve under the inferior extensor retinaculum on the dorsum of the foot and ankle. This was designated the ‘anterior tarsal tunnel syndrome’ by Marinacci (1968) to differentiate it from the better-known syndrome affecting the posterior tibial nerve below and behind the medial malleolus. Since 1985, we have seen 10 patients in whom 12 feet showed the syndrome. Nine feet were treated surgically. We report this experience and an anatomical study of 25 normal adult feet.

ANATOMY

The inferior extensor retinaculum is a Y-shaped band anterior to the ankle; its gross anatomy has been well described by Romanes (1964). We used micro-dissection of fresh specimens obtained from 25 Chinese adults to define its relationship to the terminal branches of the deep peroneal nerve.

The anterior tarsal tunnel is a flattened space between the inferior extensor retinaculum and the fascia overlying the talus and navicular. The deep peroneal nerve and its branches pass longitudinally through this fibro-osseous tunnel, deep to the tendons of extensor hallucis longus and extensor digitorum longus (Figs 1 and 2). The mean length of the tunnel is 15.7 mm laterally and 55.3 mm medially. At its superior border the average width of the tunnel is 46 mm and at the inferior border 64 mm. The mean distance between the retinaculum and the most prominent point of the head of the talus is 6.3 mm.

The stem of the Y-shaped retinaculum splits to enclose the tendons of peroneus tertius and extensor digitorum longus muscles, forming a compartment about 20 mm long by 18 mm wide with walls about 1 mm thick. The upper band of the medial part of the retinaculum

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splits to enclose the tendon of extensor hallucis longus forming another compartment which is about 11 mm long by 9 mm wide and has a thinner posterior wall. The lower medial band of the retinaculum is only about 0.5 mm thick.

In 23 of the 25 Chinese feet, the deep peroneal nerve divided into its medial and lateral branches at a mean distance of 15.5 mm (SD 5.3) proximal to the most prominent part of the articular surface of the head of the talus (Fig. 3). In the remaining two feet, the division was distal to the articular facet by 2 mm and 4 mm respectively.

Within the anterior tarsal tunnel the nerve trunk and its medial and lateral branches are oval in section, the main trunk being 2 to 3.5 mm wide and about 1 mm thick. The medial branch was slightly smaller than the lateral branch, which measured about 2.0 mm by 0.7 mm.

PATIENTS AND DIAGNOSIS

At the time of diagnosis, the age of the four male and six female patients ranged from 18 to 62 years (mean 35). The right foot was involved in five, the left in three, while two patients had bilateral symptoms. The length of history varied from one to five years. There were five manual workers, two farmers, one postman, one ballet dancer and one accountant.

The onset was related to a severe contusion by one patient, and to tight-laced footwear by two. Three patients had idiopathic pes cavus, and there was osteophyte protrusion into the anterior tarsal tunnel in one patient with talonavicular osteoarthritis (Fig. 4) and one with a navicular osteophyte. Two patients had ganglia in the anterior tarsal tunnel.

Symptoms. The patients complained of continuous paraesthesia with aching and numbness radiating from the dorsum of the foot into the hallux and second toe. There was weakness of extension of the hallux, and the foot was often liable to fatigue when walking. Symptoms were worse in plantar flexion, dorsiflexion relieved them. Wearing high-heeled or tightly laced shoes also made symptoms worse; some patients could not walk in them. Pain and paraesthesia were often severe and sometimes wakened patients during the night.

Signs. On examination there was weakness of extension of the hallux and wasting of the extensor hallucis brevis and extensor digitorum brevis muscles. Sensation was impaired in the skin of the first intermetatarsal space and the contiguous surfaces of the hallux and second toes. Percussion of the deep peroneal nerve over the anterior tarsal tunnel, on the lateral side of the tendon of the extensor hallucis longus, often elicited pain and paraesthesia radiating to the first intermetatarsal space and the first and second toes (Tinel’s sign was positive). Two-point discrimination in the region of numbness was reduced (> 40 mm).

Electrophysiological studies showed denervation in
the extensor digitorum brevis and reduction in its volitional activity. The motor conduction velocity of the nerve proximal to the ankle was normal, but distally motor latency was increased (Table I).

TREATMENT

The patient with severe contusion and the ballet dancer initially had conservative treatment. Corticosteroids were injected into the anterior part of the anterior tarsal tunnel once weekly for six weeks. Eight patients (nine feet) were operated on under local anaesthesia as follows.

A 6 cm longitudinal incision is made along the lateral border of the tendon of the extensor hallucis longus below the ankle. The lateral subcutaneous branch of the superficial peroneal nerve is retracted medially, and the inferior extensor retinaculum incised to expose the deep peroneal nerve and dorsalis pedis vessels. Decompression is performed either by simple incision of the inferior extensor retinaculum, or combining this with the removal of an osteophyte, ganglion or scar tissue. Repair of the extensor retinaculum is not necessary.

At follow-up between 1.5 and 4 years, only one of the two conservatively treated patients had improved, and then only by some slight relief of aching paraesthesia. By contrast, symptoms had been reduced or eliminated in all eight surgically treated patients, sensory recovery occurring within a day or two of operation. Motor recovery of extension of the hallux took longer, one to two weeks. Six patients eventually regained full power, the other two had only minor residual weakness.

Illustrative case. A 25-year-old postman had a four-year history of aching paraesthesia in the dorsum of his right foot which woke him at night and made him unable to wear tightly laced shoes.

Examination showed bilateral pes cavus with loss of sensation in the skin of the cleft between the hallux and second toes, weakness of extension of the big toe and wasting of extensor digitorum brevis. There was a positive Tinel’s sign and an EMG examination of extensor digitorum brevis showed denervation potentials and loss of volitional activity. The distal motor latency of the nerve was increased to 7 ms, but the motor conduction velocity of the nerve was normal from the head of the fibula to the ankle.

At operation the inferior extensor retinaculum was thick, and the head of the talus and the talonavicular joint protruded dorsally. Both branches of the deep peroneal nerve were thickened and pushed forward. Decompression was completed by incision of the articular capsule and removal of the bony protrusions. Numbness and paraesthesia had improved by the day after operation, and at review at 1.3 years, sensation and extension of the hallux were normal.

DISCUSSION

Aetiology. The cause of the syndrome is compression of the deep peroneal nerve in the anterior tarsal tunnel. This is in a relatively unprotected region of the foot, where the nerve is covered by the tendon of extensor hallucis longus as well as the inferior extensor retinaculum, and lies over the talonavicular joint, where it can be compressed by the head of the talus. In our anatomical

<table>
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<tr>
<th>Case</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Side</th>
<th>EMG Extensor digitorum brevis</th>
<th>Volitional activity</th>
<th>Distal motor latency* (ms)</th>
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<td>1</td>
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<td>25</td>
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<td>Reduced</td>
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<td>62</td>
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<tr>
<td>3</td>
<td>F</td>
<td>54</td>
<td>R</td>
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<tr>
<td>7</td>
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<td>51</td>
<td>L</td>
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<td>30</td>
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<td>35</td>
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<td>5.8</td>
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* normal distal motor latency = 4.53 ± 0.7 ms
study the nerve bifurcated at or below the level of the head of the talus in only two of 25 feet, however, the bifurcation was at this level in four of the eight surgical cases. When bifurcation is high, the two branches of the deep peroneal nerve run medial and lateral to the prominence of the head of the talus, thus being less vulnerable to compression.

Other possible factors in the aetiology are fractures, subluxations or sprains, and the wearing of high boots or high-heeled or tightly laced shoes (Marinacci 1968; Krause et al 1977; Gessini, Jandolo and Pietrangeli 1984). Two of our patients had a long history of wearing shoes with tight laces. One of them was a ballet dancer with bilateral symptoms, and, in her case, extreme plantar flexion of the foot may have increased the stretching of the nerve (Borges et al 1981).

Within the anterior tarsal tunnel the nerve is usually mobile within delicate areolar tissue: any adhesions between the epineurium and its surrounding structures will limit this mobility and result in neural dysfunction. Such adhesions were found in the ballet dancer and also in a patient with an ankylosed knee who needed to use tight shoe lacing. At operation in this case there was clear evidence of compression of the nerve by scar tissue.

Nerve compression by ganglia and by bony protrusion was conclusively shown at operation in our series. There was a low bifurcation of the nerve in relation to the head of the talus in three of the four cases. In two of them, the medial and lateral branches of the nerve were thickened and lacked lustre for 1 to 1.5 cm.

The three patients with bilateral pes cavus all had unilateral symptoms. The high longitudinal arches, with protrusion of the talonavicular and cuneonavicular joints, stretched the inferior extensor retinaculum and caused compression. The condition was aggravated by the use of high boots, tight fitting or tightly laced shoes.

**Diagnosis.** The clinical symptoms and signs are usually characteristic; pain may be made worse by plantar flexion.

The differential diagnosis includes L5 radicular dysfunction, especially that due to a prolapsed intervertebral disc, which may show similar presenting symptoms. Electromyography may help to solve difficult diagnostic problems, as it may indicate the exact level of neural dysfunction.

**Treatment.** Corticosteroid injections failed in two of our cases, operation was satisfactory in eight. However, in our experience, the injection of corticosteroids is worthwhile initially, with surgical treatment where this is unsuccessful. Operation must achieve complete decompression, with careful division of adhesions to allow the nerve to take up a relaxed position. Epineurial incision may be required where dense scar surrounds the nerve. There is no need to suture the inferior extensor retinaculum: we found no tendon bowstringing in our patients at follow-up.

In our view, the anterior tarsal tunnel syndrome is not uncommon, but is often overlooked clinically. The symptoms can be relieved by treatment.

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No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


