The anterior tarsal tunnel syndrome

Report of two cases

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Two recently encountered patients with the anterior tarsal tunnel syndrome are presented. The various aspects of this probably under-recognized syndrome are discussed. Evidence is presented that this syndrome may be the result of abnormal stretch of the deep peroneal nerve.

KEY WORDS • peroneal nerve • nerve compression • tarsal tunnel syndrome

The term “tarsal tunnel syndrome” was established in 1962 by Keck to delineate a compression of the posterior tibial nerve in its fibro-osseous tunnel posterior to the medial malleolus. Subsequently, Kopell and Thompson identified a compression syndrome of the deep peroneal nerve underlying the inferior extensor retinaculum, which later was designated the “anterior tarsal tunnel syndrome,” while the syndrome affecting the posterior tibial nerve was renamed the “posterior or medial tarsal tunnel syndrome.”

Many reports have appeared delineating the various aspects of the posterior tarsal tunnel syndrome, but few have considered the anterior tarsal tunnel syndrome. Since the anterior tarsal tunnel syndrome may be a clinically under-recognized entity, we are reporting our recent experience with two patients treated for this disorder.

Case Reports

Case 1

This 66-year-old woman was well until 1971, when she began experiencing intermittent low-back discomfort and sciatica. This was managed conservatively, with gradual resolution. In 1972, she noted a circumferential tight feeling about the left ankle; it was brought on by sitting or lying down and occasionally awakened her at night. It was relieved by standing or walking. Associated with this discomfort was a peculiar inversion and extension of the left foot which she attempted to correct with a plastic orthosis. A peroneal nerve exploration at the fibular neck did not provide relief. Subsequently, she also noted intermittent paresthesias along the dorsum of the left foot radiating to the left first web space. Past medical history was remarkable for bilateral carpal tunnel releases in 1965.

On examination in 1978, she held the left foot in a plantar flexed and intermittently inverted posture. The left extensor digitorum brevis was atrophied and weak. Mild hypalgesia and hypesthesia to light touch were present in the dorsal first web space, and a Tinel's sign could be elicited from an area slightly anterosuperior to the left medial malleolus. Nerve conduction studies confirmed a deep peroneal nerve dysfunction on the left (Table 1). After an unsuccessful attempt at conservative therapy, the left deep peroneal nerve was explored and a tissue band, 2 mm in diameter, was encountered binding the epineurium
TABLE 1  

**Summary of electrophysiological studies**

<table>
<thead>
<tr>
<th>Study</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Normal</th>
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</thead>
<tbody>
<tr>
<td><strong>Nerve Conduction</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>left peroneal nerve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>distal motor latency (msec)</td>
<td>7.5</td>
<td>8.6</td>
<td>5.0</td>
</tr>
<tr>
<td>conduction velocity (m/sec)</td>
<td>48</td>
<td>49</td>
<td>43</td>
</tr>
<tr>
<td>right peroneal nerve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>distal motor latency (msec)</td>
<td>3.5</td>
<td>5.4</td>
<td>5.0</td>
</tr>
<tr>
<td>conduction velocity (m/sec)</td>
<td>52</td>
<td>52</td>
<td>43</td>
</tr>
<tr>
<td><strong>Electromyography</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>extensor digitorum brevis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>left</td>
<td>severe chronic denervation</td>
<td>severe acute &amp; chronic denervation</td>
<td></td>
</tr>
<tr>
<td>right</td>
<td>mild chronic denervation</td>
<td>mild chronic denervation</td>
<td></td>
</tr>
</tbody>
</table>

To the undersurface of the lower fibers of the extensor hallucis longus muscle (Fig. 1). This was excised and sent for pathological examination, which revealed dense fibrous tissue without amyloid. Thereafter, the inferior extensor retinaculum was divided, as the deep peroneal nerve was found to be widened as it passed underneath.

When seen 4 months postoperatively, the patient was without complaints referable to her left foot.

**Case 2**

This 48-year old man with Parkinson’s disease controlled by carbidopa and levodopa presented in April, 1977, for evaluation of pain in the dorsum of his left foot. He gave a history of several months of an intermittent tight feeling across the anterior aspect of his left ankle which made him want to move the ankle. Shortly thereafter, he began complaining of intermittent discomfort in the dorsum of the left foot, described as a dull, aching pain without paresthesias. He denied weakness of the left foot or leg.

Examination revealed normal strength in the feet and toes without atrophy. There was decreased appreciation to light touch and pinprick over the mid-dorsal and lateral dorsal surface of the left midfoot from the level of the cuneiform bones to the distal second to fourth metatarsals. The ankle jerks were 1+ bilaterally, and all other deep tendon reflexes were normal. The patient was thought to have a localized

![Diagram of the deep peroneal nerve](image-url)  
**FIG. 1.** Case 1. Diagram showing the deep peroneal nerve. The epineurium was found to adhere to the extensor hallucis longus muscle.
Anterior tarsal tunnel syndrome

compressive neuropathy in the left foot. Phenytoin was prescribed in an attempt to control the neuralgic pain but was of no benefit. However the administration of carbamazepine, 200 mg three times daily, was initially associated with some diminution of the pain.

Due to the persistence of the left foot pain, the patient underwent nerve conduction and electromyographic examination in February, 1978, the results of which are shown in Table 1. The findings were thought to be consistent with an entrapment of the deep peroneal nerve at the ankle beneath the inferior extensor retinaculum. The patient had orthopedic consultation, and an injection of 20 mg of triamcinolone hexacetonide in 1% lidocaine hydrochloride was made into the area of the deep peroneal motor branch to the extensor digitorum brevis. This did not alter the pain syndrome. The patient was then referred to a podiatrist who fashioned a wedged orthosis for the left shoe in order to correct an overpronation of the left foot. After 2 weeks of using this orthosis, the patient began noticing progressive improvement of the aching discomfort in the dorsum of the foot. After 2 months, the patient’s left foot was virtually painless. He wore the orthosis for approximately 1 year, and then discontinued it. In the ensuing year, he has had only rare twinges of dorsal left foot pain following exercise, and reports that he is asymptomatic greater than 95% of the time.

Electrophysiological Studies

Motor nerve conduction of the peroneal nerve was recorded with techniques similar to those of Behse and co-workers. Surface or needle electrode recording was made from the extensor digitorum brevis, and the nerve was stimulated at the ankle and at the fibular head with surface electrodes. The nerve conduction values were corrected 2 m/sec for each degree below 36°C. The distal motor latencies were corrected to 0.15 msec for each degree below 36°C. Nerve conduction in the posterior tibial nerve was tested with similar techniques, with recording electrodes over the abductor hallucis brevis and stimulation at the ankle and popliteal fossa. Medial plantar sensory action potentials were obtained using techniques proposed by Guiloff and Sherratt. The sensory conduction velocity was corrected 2 m/sec for each degree below 36°C. The sural nerve sensory action potential was recorded similar to the techniques of Burke, et al., using the “proximal segment.” Again, the sensory conduction velocity was corrected 2 m/sec for every degree below 36°C.

Discussion

Anatomy

Beginning between the neck of the fibula and the peroneus longus muscle, the deep peroneal nerve runs inferomedially on the fibula, joining the anterior tibial vessels on the surface of the interosseous membrane. Together they descend, overlapped by the extensor hallucis longus, onto the distal tibia. In coursing over the ankle joint the nerve is bounded dorsally by the fascia overlying the talus and navicular and ventrally by the extensor hallucis longus muscle fibers and tendon as well as the inferior extensor retinaculum. Within the anterior tarsal tunnel the deep peroneal nerve and anterior tibial vessels are ensheathed with a delicate areolar tissue which allows mobility of the overlying tendons without stretching the neurovascular structures. Slightly rostral to or underlying the inferior extensor retinaculum, the deep peroneal nerve divides into a medial and lateral branch which continue into the foot. The lateral branch passes under the extensor digitorum brevis to innervate this muscle and the nearby tarsal, tarsometatarsal, and metatarsophalangeal joints. Continuing onto the dorsum of the foot, the medial branch passes under the extensor hallucis brevis tendon to innervate the skin between the first and second toes.

Etiology of Compression

These anatomical considerations suggest that the anterior tarsal tunnel is a relatively unprotected region in which the deep peroneal nerve is susceptible to compression. Tightly worn shoes have been implicated as a primary etiological factor in this compression. However, during the anterior tarsal tunnel exploration in Case 1, it seemed that compression of the deep peroneal nerve underlying the inferior extensor retinaculum was a minor factor in any neural dysfunction. Therefore we were prompted to study further the relationship of the deep peroneal nerve to its surrounding structures.

Thirty anterior tarsal tunnels in 20 patients, aged 20 to 80 years, without evidence of neurological disease were studied during postmortem examinations. In its passage through the anterior tarsal tunnel, the deep peroneal nerve appeared flattened and widened by the overlying extensor retinaculum. This finding was consistent throughout all the specimens, but was somewhat more prominent in older than younger subjects. The degree of neural flattening observed in normal subjects was identical to that seen during exploration in Case 1.

After the deep peroneal nerve was exposed, the foot and toes were moved into different positions to determine the angle that caused maximum neural stretch. With the foot planter flexed and the toes dorsiflexed the nerve was placed under maximum stretch across a ridge formed by the hyaline cartilage overlying the fibronaviculare joint. This position is the same as that induced by wearing high-heeled shoes. While the normal nerve may be able to withstand this degree of stretch because of its mobility within the areolar compartment, pathological adhesions between the epi neurium and surrounding structures inhibit this mo-
bility and result in neural dysfunction. Presumably this explains the dysfunction in our first patient.

The exact pathophysiology of neural dysfunction in our second patient remains unknown, because the tarsal tunnel anatomy was not examined. However, since an orthosis restoring normal foot position provided resolution of symptoms, abnormal neural stretch is a likely etiology here also.

**Clinical Manifestations and Differential Diagnosis**

Although the deep peroneal nerve is a mixed sensorimotor nerve, symptoms are sensory in origin. Numbness and paresthesias can be noted in the first dorsal web space, and many patients also complain of an aching or tightness about the ankle and dorsum of the foot. This is worsened by certain positions and inactivity, often awakening the patient from sleep. In some instances, maintaining the affected foot in a certain position will minimize the discomforts.

Examination can reveal both sensory and motor signs. Hypalgesia and hypesthesia to light touch are found in the first dorsal web space. It may be possible to elicit Tinel’s sign over the area of nerve injury. The extensor digitorum brevis may be weak and atrophied.

While the fully developed syndrome is distinctive, similar presenting symptoms may be observed in common peroneal nerve and L-5 radicular dysfunction. Electrophysiological studies should always be obtained to assist with difficult diagnostic problems, as they may indicate the exact level of neural dysfunction. Characteristic electrophysiological abnormalities are illustrated in Table 1.

**Treatment**

Although the limited experience with this entity precludes an extensive discussion of therapeutic maneuvers, commonsense dictates that conservative measures are indicated initially. Avoiding footwear and activities that unduly stretch the deep peroneal nerve may result in prolonged resolution of symptoms, as was effective in our second case. Should further measures be required, the anterior tarsal tunnel may be infiltrated with steroids. Surgical exploration may be required in selected cases. If the deep peroneal nerve is explored, care should be taken to visualize the nerve for several centimeters above the inferior extensor retinaculum as well as underneath it to eliminate pathological adhesion which may alter its mobility. Merely sectioning the inferior extensor retinaculum is not sufficient, as the pathophysiology of this syndrome may be more related to neural stretch than compression.

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**References**


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