Case Report

Anterior Tarsal Tunnel Syndrome: Electrophysiological and Clinical Evaluation of Five Cases

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Summary

Anterior tarsal tunnel syndrome is entrapment of deep peroneal nerve between the inferior extensor retinaculum and fascia overlying the talus and navicular bones that is an infrequent and probably overlooked condition. We report five cases which were referred to EMG laboratory with different diagnosis other than anterior tarsal tunnel syndrome. In our cases, ground sitting habits such as legs crossed position and practising “namaz” is thought to be etiological factors. As our opinion, hand dominance and special sitting position during namaz probably determined the side of the affected limb. It is important to keep in mind anterior tarsal tunnel syndrome during the EMG examination especially in patients complaining foot ache with ambiguous diagnosis.

Key words: Anterior tarsal tunnel syndrome, electrophysiology, etiological factor

Anterior Tarsal Tünel Sendromu: Beş Olgunun Elektrofizyolojik ve Klinik Değerlendirmesi

Özet


Anahtar Kelimeler: Anterior tarsal tünel sendromu, elektrofizyoloji, etiyolojik neden

INTRODUCTION

In 1963, Koppel 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 called as “the anterior tarsal tunnel syndrome (ATTS)” by Marinacci to differentiate it from classical tarsal tunnel syndrome(11). The anterior tarsal tunnel (ATT) is a flattened space between the inferior extensor retinaculum and fascia overlying the talus and navicular bones(10). The deep peroneal nerve (DPN) and its branches pass longitudinally through this fibro-osseous tunnel. The lateral terminal branch supplies extensor digitorum brevis (EDB) and extensor hallucis brevis muscles while the medial gives the sensory fibers to the skin of first toe interspace and the adjacent sides of the first and second
toes. Symptoms are primarily sensory and include numbness and paresthesia in the first web space as well as paresis and atrophy of the extensor digitorum brevis muscle. A partial syndrome due to compression of the sensory branch also has been reported\(^{(12)}\).

Compression and stretching of deep peroneal nerve in the anterior tarsal tunnel under the inferior extensor retinaculum in a relatively unprotected area is the cause of anterior tarsal tunnel syndrome. The causes might be due to any abnormality of the elements surrounding the structure such as the presence of bony protrusion, ganglia, tightening and thickening of retinaculum. Other possible factors which causes acute trauma or chronic compression of nerves are edema, postpartum period\(^{(1)}\), fractures, subluxations, ankle sprains, wearing high heel\(^{(7)}\) or ankle boots or tightly fitting shoes, talus fracture\(^{(4)}\), osteophytes, pressure from the extensor hallucis brevis muscle, various special jobs such as dancers\(^{(9)}\), pes cavus and “namaz” (kneeling and prone posture for prayer)\(^{(3,7,11)}\).

Five patients diagnosed as ATTS with different etiologies from our hospital's neurophysiology department will be presented in this report. As above mentioned, compression was the most frequent etiological factor but “namaz” especially in men was the second most seen etiology in ATTS patients. As all known, ATTS is a rare entrapment neuropathy, we would like to emphasize regional and sociocultural differences of the etiological factors affecting the evolution while reviewing electrophysiological and clinical features.

**CASE PRESENTATION**

**Case 1:**

Twenty-nine years old right handed women who had numbness of the left foot, left toe in particular for two years referred to our hospital with a diagnosis of polyneuropathy. She had no risk factor for polyneuropathy. When the history was questioned she reported to sit legs crossed when she was at home. Neurological examination disclosed hypohesthesia over the left foot and weakness of toe dorsiflexion (4/5). In electrophysiological examination, left fibular motor nerve distal latency (DL) was found prolonged (16.4 msec). CMAP amplitude was very low, therefore a needle electrode was used for recordings (Figure-1). Left deep peroneal sensory nerve action potential (DPN-SNAP) could not be elicited while the contralateral (DPN-SNAP) and superficial peroneal SNAP were normal. Other nerve conduction properties were in normal limits (Table-1).

During the needle EMG examination acute and chronic denervation findings and decreased recruitment and volitional activity were observed in extensor digitorum brevis muscle (EDB).

**Case 2:**

Thirty-six years old right handed women complaining left knee ache and numbness on plantar surface of left foot for one year was referred to our electrophysiology department for suspected polyneuropathy. In history she said that she always sat legs cross especially during the meals. She was not diabetic. She was on antidepressive treatment for one year (escitalopram). Neurological examination revealed hypohesthesia of sole of the foot and weakness of toe dorsal flexion (4/5). In electrophysiological examination, left fibular motor nerve DL was 12.5 msec, MCV was 49.5 m/sec in the segment from the fibular head to the ankle. Recordings were carried out with needle electrode because CMAP amplitude was to small to be detected by superficial electrode. DPN-SNAP nerve could not be produced while the other sensory and motor nerve conduction properties were in normal limits (Table-1) (Figure-2 and 3). During electromyographic examination chronic neurogenic MUP changes and and decreased volitional activity were observed in EDB muscle.
Table 1: Electrophysiological documentation of five cases. EDB: extensor digitorum brevis muscle, NCV: nerve conduction velocity, DL: distal latency, EMG: electromyography.

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Peroneal motor NCV/DL (right)</th>
<th>Peroneal motor NCV/DL (left)</th>
<th>Peroneal sensory NCV/DL (deep) (right)</th>
<th>Peroneal sensory NCV/DL (deep) (left)</th>
<th>Peroneal sensory NCV/DL (superf.) (right)</th>
<th>Peroneal sensory NCV/DL (superf.) (left)</th>
<th>Needle EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case-1</td>
<td>49 m/sec 3.6 msec</td>
<td>42.2m/sec 16.4 msec (needle electrode recording)</td>
<td>48.1m/sec 2.7 msec No response</td>
<td>69 m/sec 2.1 msec 53 m/sec 1.8 msec</td>
<td>Active and chronic denervation in EDB muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case-2</td>
<td>48 m/sec 4.1 msec</td>
<td>49.5m/sec 12.5 msec (needle electrode recording)</td>
<td>43.8m/sec 3.2 msec No response</td>
<td>52m/sec 2.5 msec 50m/sec 2.4 msec</td>
<td>Chronic neurogenic MUP changes and decreased voluntary activity in EDB muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case-3</td>
<td>54.1 m/sec 4.9 msec</td>
<td>50 m/sec 7.9 msec</td>
<td>45.8m/sec 3.5 msec No response</td>
<td>47.6m/sec 2.9msec 50m/sec 3.8msec</td>
<td>One chronic neurogenic MUP in EDB muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case-4</td>
<td>50.9m/sec 4.0 msec</td>
<td>48.4m/sec 7.3 msec (needle electrode recording)</td>
<td>44.3m/sec 2.7 msec No response</td>
<td>50.5m/sec 2.7 msec 59.3m/sec 2.8 msec</td>
<td>Denervation potentials and one chronic neurogenic MUP in EDB muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case-5</td>
<td>42 m/sec 7.1 msec</td>
<td>55 m/sec 4.0 msec</td>
<td>No response</td>
<td>45.2 m/sec 3.1 msec 47 m/sec 2.8 msec 56 m/sec 2.6 msec</td>
<td>Chronic neurogenic MUP changes and MUP loss with denervation potentials in EDB muscle</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1: Left fibular motor response recording by needle electrode from EDB muscle of Case-1, stimulation from ankle and caput fibula.
Figure 2: Left superficial peroneal sensory recording- Case 2. 1msec, 10 µV.

Figure 3: Right deep peroneal sensory recording- Case 2.

Figure 4: Normal electrophysiological results of right fibular nerve of Case-4.
Case 3:
A thirty eight years old right handed women was referred to our hospital with possible diagnosis of fibular entrapment neuropathy and polyneuropathy. She has had ankylosing spondylitis for ten years and taking medicine. Because rheumatologist did not think that left foot ache and numbness due to arthritis, patient was referred to clinical neurophysiology department. She reported that she was used to sit cross legged position while sitting around a ground table during the meals and other times. The physical and neurological examination revealed left plantar and dorsal hypoesthesia, mild weakness of toe dorsiflexion (4/5). In electrophysiological examination, left fibular motor nerve DL was 7.9 msec,. All the sensory recordings were normal but left DPN-SNAP could not be recorded. In needle EMG only one large polyphasic MUP was recorded in EDB muscle. Other examined muscles were normal.

Case 4:
Twenty-six years old right handed male was referred to our department with possible diagnosis of polyneuropathy. He had complaints of left plantar and knee ache for two years. He reported an increase of complaints last 2-3 months while he was performing his military service. He was practising “namaz”. He was not diabetic. In neurological examination, EDB muscle was atrophic, toe dorsiflexion was 2/5. In electrophysiological examination, left fibular motor nerve DL was 7.3 msec. All the sensory recordings were normal but left deep peroneal sensory response could not be recorded. In needle EMG only one large polyphasic MUP and denervation potentials were recorded in EDB muscle. Other examined muscles were normal.

Case 5:
Fifty-three years old, right handed male was referred to clinical neurophysiology department from our neurology outpatient clinic for electrophysiological examination to explain his right foot numbness, tingling and weakness of toe dorsiflexion. The possible diagnosis was entrapment neuropathy of right foot or polyneuropathy. The skin over the anterior aspect of ankles was thick and callused. When patient was questioned about the calluses he said he spent prolonged time sitting over his feet during praying (namaz). He had no diabetes. He complained about continuously tingling and numbness of right foot. Neurological examination revealed dorsiflexion weakness of toes on right side (4/5). In electrophysiological examination, right fibular DL was 7.1 msec. (Table-1), right deep peroneal sensory response could not be recorded. Electromyographic examination of EDB muscle showed fibrillation potentials, positive sharp waves, decreased volitional activity with only the presence of one large polyphasic MUP. Electromyographic examination of the other muscles was normal.

DISCUSSION
Anterior tarsal tunnel syndrome is a rare type of entrapment neuropathy localized between the inferior extensor retinaculum and fascia overlying the talus and navicular bones(2). Entrapment neuropathy is a frequent diagnosis in many patients who are referred to electrophysiological examination. But the site of entrapment or affected nerve are seldom specified. ATTS remains poorly diagnosed in many cases(6). Although two of five patients in our series had two prodagnosis; entrapment neuropathy and polyneuropathy, polyneuropathy was leading diagnosis in all patients. It seems like polyneuropathy was usually suspected in all patients who complained numbness or tingling sensations and ache. When clinician did not make a through questioning and physical examination a nonspecific diagnos is most likely. In this situation electrophysiological investigation should
be carried out cautiously and carefully to unveil the real problem. When prolonged distal motor latency of peroneal nerve present while NCV is normal it should be suspected. The examination protocol should include deep sensory peroneal nerve examination as well as routine nerve conduction examinations. It would be best to search for other entrapment neuropathies and polyneuropathy since in polyneuropathies nerves are more susceptible to entrapment lesions. The differential diagnosis should also include L5 radicular dysfunction, which may show similar symptoms especially in the presence of a protruded intervertebral disc. Electromyography may help to solve difficult diagnostic problems, as it may indicate the exact level of neural dysfunction.

The reported women cases traditionally prefer to sit in legs crossed position during their daily life. All of the patients were right handed. Because of that it is possible that they might use right leg preferentially and when they sit they take left leg under their right leg. Their complaints might be due to preferred use of dominant limb. An interesting point is that sitting position in context of hemispheric dominancy has not been discussed before in literature. Four of our five cases had left sided symptoms, and findings whereas only one patient had right sided symptom. In literature only asymmetrical sport activity was reported to cause nerve conduction abnormalities in the dominant extremity. Even its aim is different, “namaz” also can be considered a kind of ground sitting position. During the namaz as a rule men sit on their left foot in dorsally flexed position while right is in planter flexion plastered on to floor (Figure 5). This position puts both peroneal nerves in a vulnareble position. Both of male cases had been practising namaz. The complaints of case 4 increased during his military service because of wearing high boots whereas case 5 only had “namaz” history to explain clinical picture. Possibly sitting over the feet on kneeling position stretched the nerve and the body weight compressed the nerve between the floor and talus with concomitant hardening of connective tissues.

CONCLUSION

In conclusion even it is rarely encountered, anterior tarsal tunnel syndrome should be kept in mind when evaluating patients with numbness or pain as well as weakness of the toes especially if there is no clear clinical diagnosis. Concerning hemispheric dominance in the etiology of the asymmetrical nerve lesions, a controlled study would be valuable.

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