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ORIGINAL ARTICLE

Anterior tarsal tunnel syndrome: a misunderstood and a misleading entrapment neuropathy

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Abstract Anterior tarsal tunnel syndrome (ATTS) is a rare entrapment neuropathy of the deep peroneal nerve beneath the extensor retinaculum on the top of the ankle. ATTS is often asymptomatic or olygosymptomatic. There are few reports describing the ATTS. We describe the clinical and electrophysiological features of 85 patients with unilateral or bilateral ATTS prospectively collected between January 2000 and December 2010 in our laboratory of Clinical Neurophysiology. This entrapment neuropathy remains poorly diagnosed and it might be misleading when performing a diagnostic EMG-ENG examination for suspected polyneuropathy or lumbosacral radiculopathy.

Keywords Anterior tarsal tunnel · Lumbosacral radiculopathy · Entrapment neuropathy · Neurophysiology

Introduction

Anterior tarsal tunnel syndrome (ATTS) is a chronic entrapment neuropathy involving the deep peroneal nerve beneath the thin Y-shaped inferior extensor retinaculum (cruciate ligament) on the top of the ankle [1–5]. Initially, the existence of a true anatomic "anterior tarsal tunnel" was debated but it has been subsequently detailed [6–8]: the inferior extensor retinaculum forms the roof of the route covered by the deep peroneal nerve at the ankle before dividing in its lateral (motor) and medial (sensory) branches, while the floor is the fascia overlying the talus and navicular bones. In the tunnel there are four tendons, the dorsal pedis artery, the anterior tibial veins, and the deep peroneal nerve [1]. Therefore, the anterior tarsal tunnel is a flattened and a relatively unprotected space in the ankle. As a result, any external pressure could traumatize the underlying nerve. Several factors may contribute to the development of ATTS. ATTS may be unilateral or bilateral. The most common cause of compression is trauma to the dorsum of the foot (fractures, subluxation or sprains), which could cause local fibrosis or modify the configuration of the "tunnel". ATTS may also be related to talanavicular osteophytosis, fibrous band, high-heeled and/or tight fitting shoes, localized oedema, long-standing abnormal posture, non-neurogenic pes cavus and ganglion in various combinations [2, 4, 8–12].

If it is symptomatic, ATTS could cause dull ache, numbness and paresthesias on the dorsum of the foot, radiating to the first interdigital space. These symptoms could be increased by certain positions (plantar flexion of the foot) or inactivity, or they could have nocturnal exacerbation and proximal radiation. In some symptomatic patients, we could find hypoesthesia over the first web space. In extremely serious cases, weakness of toe extension and atrophy of the extensor digitorum brevis (EDB) muscle have been found. However, the motor symptoms are generally mild because the function of the EDB is replaced by the extensor digitorum longus and the extensor hallucis longus muscles [1, 5].

We report the clinical and electrophysiological characteristics of our prospectively collected 85 patients who were diagnosed with ATTS.

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Materials and methods

We performed 19,582 EMG–ENG examinations, using Nicolet Viking IV equipment in all cases, in our laboratory of Clinical Neurophysiology between January 2000 and December 2010.

Peroneal motor nerve conduction studies and antidromic superficial sensory studies, at least sural nerve and superficial peroneal sensory nerve bilaterally, like other conduction studies, were performed according to standard conduction techniques especially maintaining a fixed distance to calculate distal motor latency (such as 7 cm for distal motor latency peroneal nerve motor conduction study) [13, 14]. Skin temperature was monitored just above the ankle and maintained above 32 °C. The patients were laid in supine position, and the room temperature was 23–24 °C in all seasons. Concentric needle electrode (CNE) examination was performed bilaterally on the EDB muscle and on other muscles, according to an individualized needle EMG study based on the clinical findings and the differential diagnosis requested, nevertheless always including bilateral NCS of sural, superficial peroneal and deep peroneal nerves.

In our laboratory, ATTS was diagnosed when: (1) deep peroneal motor distal latency was more than 3 SD above the mean (normal value 3.97 ± 0.44 ms) and, if unilateral, more than 130 % of the contralateral value (in our laboratory the normal value of intratrial intraindividual asymmetry was 2.44 ± 7.52 %) [15] or (2) the M response of EDB muscle was absent and (3) there were electromyographic abnormalities (abnormal spontaneous activity and/or abnormal motor unit action potential morphology and firing pattern and reduction in volitional activity) confined to the EDB muscle and in excess of the normal electromyographic findings in the EDB muscle in healthy subjects, especially the older ones, according to our laboratory references (none healthy subject presents fibrillation potentials in EDB muscle; standardizated interference pattern in subjects aged 15–65 years and in subjects aged >65 years). We excluded patients with polyneuropathy, ipsilateral lumbosacral radiculopathy, common or deep peroneal neuropathy at the fibular neck, and deep peroneal neuropathy at the ankle produced by penetrating wounds. The presence of an accessory deep peroneal nerve was excluded in all patients who were diagnosed with ATTS in order to avoid errors in interpreting the data on peroneal nerve conduction velocity [16–18].

Results

According to previously described electrophysiological criteria, we excluded 19 patients satisfying the above electrophysiological criteria for ATTS with co-existing axonal polyneuropathy and 25 patients with co-existing ipsilateral lumbosacral radiculopathy. Isolated deep peroneal mononeuropathy at the ankle (i.e., ATTS) was diagnosed in 85 patients, 46 females, 39 males (0.43 % of the patients examined between January 2000 and December 2010). At the time of the diagnosis, the age of patients ranged from 27 to 82 years (mean 53.4 ± 12.8 years). ATTS was unilateral in 73 patients and bilateral in 12 patients. Nerve conduction studies revealed an increased peroneal motor distal latency with a mean value of 7.42 ms (range from 5.5 to 11.6 ms in all cases). Stimulating the deep peroneal nerve at the ankle and recording with both surface electrodes and CNE, M response was absent in five patients. In all patients with ATTS, CNE examination disclosed a chronic neuropathic pattern of variable gravity in the EDB muscle; abnormal spontaneous activity (fibrillation potentials and positive sharp waves) was found in 34 cases. All patients were asymptomatic for ATTS, also if specifically inquired, except for 19 patients who reported a dull ache on the top of the foot was also. There was an asymptomatic hypotrophy or atrophy of the EDB muscle in all patients with ATTS; in 59 patients there was also a mild hypoesthesia in the first interdigital space. The etiology of ATTS was unknown in 33 patients; miscellaneous causes were recorded in 52 patients (ankle sprains treated with a compression bandage or plaster cast, remote contusions, tight fitting shoes often associated with non-neurogenic pes cavus).

Conclusions

This entrapment neuropathy was identified for the first time in 1963 by Kopell and Thompson [3] and was designated as “the anterior tarsal tunnel syndrome” by Marinacci [19] to distinguish it from the better known entrapment neuropathy affecting the posterior tibial nerve at the medial side of the ankle. There are few reports and little investigation regarding the electrophysiologic features, the frequency and the etiology of this entrapment neuropathy [1, 2, 4, 5, 8, 11, 12]. We described the clinical and electrophysiological data of a prospectively collected series of patients who were diagnosed with ATTS. Clinical features of our patients proved that this condition is often asymptomatic. For this reason, it is often difficult to discover its etiology retrospectively in individual patients. Various etiologies have been reported since the first description of ATTS. For example, in a study of 10 patients, published by Zonghzao et al. [8], two subjects presented with a deep peroneal mononeuropathy at the ankle caused by osteophyte protrusion while in two cases ganglia was found in the anterior tarsal tunnel; in two patients, the onset was

related to tightly laced footwear, while three subjects presented with an idiopathic pes cavus. In one patient, the cause was severe contusion. Akyüz et al. [10] reported 14 cases of ATTS: all patients had been doing Namaz (the kneeling/prone posture of prayer) for many years, suggesting that chronic prolonged stretching of the deep peroneal nerve at the ankle may cause this entrapment neuropathy. All our patients presented with atrophy and paresis of the EDB muscle (but its weakness was often undetectable in spite of evident atrophy because its function is replaced by extensor hallucis longus and extensor digitorum longus muscles); only 19 patients had vague sensory disturbance on the dorsum of the foot. In all patients, a conservative treatment was recommended (rest, shoe modifications, physical therapy and/or steroid injection) because the symptoms were mild.

EMG–ENG examination was performed for suspected polyneuropathy or lumbosacral radiculopathy, or because non-specific pain/paresthesia in lower limbs in most part of the patients was observed. The ATTS may then be an unforeseen and misleading event because it involves a nerve (the peroneal nerve) and a muscle (the EDB muscle) frequently examined in routine EMG–ENG studies. In these patients, a high degree of attention is necessary in order to avoid a wrong electrophysiological diagnosis and a wearisome EMG–ENG examination. Moreover, deep peroneal mononeuropathy at the ankle, as we reported in this study, is a rare (0.43 % among 19.582 subjects examined in 10 years) but non-exceptional chronic entrapment neuropathy, and "pure" ATTS (we excluded patients with polyneuropathy and/or lumbosacral radiculopathy) has nearly the same frequency as that of the posterior tarsal tunnel syndrome in our laboratory. It can also be difficult to diagnose ATTS if fibrillations in the EDB are attributed to "age" or "foot trauma" and a prolonged peroneal distal latency to cool extremities [1, 20, 21]. ATTS may co-exist with polyneuropathy or radiculopathy, and its incidence may turn out to be higher like all other entrapment neuropathies of the lower limbs.

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