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Prognostic value of nerve ultrasound and electrophysiological findings in traumatic sural neuropathy

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ABSTRACT

Introduction: We report on the prognostic role of cross sectional area (CSA) enlargement and axonal damage in traumatic sural neuropathy (TSN).
Methods: Reference values were defined in 23 healthy subjects. 13patients with TSN underwent evaluation (Thessaloniki Hypesthesia Score (THS), ultrasound, electrophysiology). All patients were followed up with THS 6 months after initial evaluation.
Results: During initial evaluation, the 13 patients showed a mean THS of 2.6 (SD ± 0.9). 7 patients showed pathological (pUS) and 6 normal CSA (nUS). 8 patients showed axonal affection (pCS) and 5 no axonal affection (nCS). During follow up, mean THS was 3.1 (SD ± 0.9) in pUS, and 1.8 (SD ± 0.7) in the nUS group ($p < 0.001$). Mean THS was 2.8 (SD ± 0.7) in pCS, and 2.1 (SD ± 0.9) in nCS group ($p = 0.035$).
Discussion: CSA enlargement, but not axonal loss, seems to have a negative prognostic role in patients with TSN.

1. Introduction

The sural nerve is usually characterised by a great anatomical and topographical variability (Amoiridis et al., 1997). Sural mononeuropathy is considered as rare, but may occur in a variety of pathological conditions, such as stripping or venous surgery, peripheral nerve tumors, or even traumatic injury. The largest sample reported in the literature included 36 patients, among them 50% cases were of non-surgical, non-traumatic origin (Yuebing and Lederman, 2014). Traumatic sural neuropathy (TSN) can be caused by fractions, iatrogenic lesions during Achilles tendon reconstructive surgery, stretch injury due to ankle sprain, or even chronic external compression due to tight shoe wearing (Fabre et al., 2000; Yuebing and Lederman, 2014). Electrophysiological evaluation of the sural and lower/upper extremity nerves is used not only to confirm the presence and severity of the neuropathy, but also to differentiate a sural mononeuropathy from an affection of the same nerve in the context of a polyneuropathy.

To our knowledge, no sufficient literature data exist on the prognostic value of nerve ultrasound and electrophysiological findings in TSN. In this study, we evaluated the prognostic value of the cross-sectional area (CSA) enlargement and axonal damage in patients with TSN of different etiology.

2. Methods

2.1. Subjects and patients

The ethics committee of the local council (St Luke's Hospital, Thessaloniki) approved our study protocol and all healthy subjects and patients signed an informed consent form. Inclusion criteria included: (1) patients aged > 18 years old, with history of trauma or surgery of the lower extremities in the last 6 months, (2) the presence of symptoms and signs compatible with isolated sural neuropathy, (3) electrophysiological evidence of sural nerve affection, defined as sensory conduction velocity of < 40 m/sec and/or amplitude of the sensory action potential (sNAP) < 6.0 μV.

Exclusion criteria included: (1) symptoms, clinical or electrophysiological signs referable to other peripheral nerve disease (for example polyneuropathy), (2) history of diabetes, alcoholism or any other systematic disease, that could potentially affect peripheral nerves, (3) severe obesity, defined as body-mass index (BMI) > 40.

2.2. Study protocol

This prospective study was divided in two phases. In the first phase,

Abbreviations: CSA, cross sectional area; f.e, for example; TSN, traumatic sural neuropathy; SNAP, sensory nerve action potential

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Table 1
Thessaloniki hypesthesia score.

Thessaloniki Hypesthesia Score	
Symptom	Points
No hypesthesia	1
Mild hypesthesia, not limiting facilities in everyday life	2
Severe hypesthesia, limiting facilities in everyday life	3

Overview of the scoring system, that helps in the quantification of the severity of hypesthesia in patients with traumatic sural neuropathy.

all patients underwent a double blinded ultrasound and electrophysiological examination of the sural nerve. In addition, a scoring system developed at our department (Thessaloniki Hypesthesia Score, THS, Table 1) addressed the severity of hypesthesia in the patients' group. All healthy subjects underwent electrophysiological and ultrasound evaluation of the sural nerve on both sides, in order to exclude bilateral, even subclinical affection of the nerve.

The patients were divided into two groups, according to the presence of CSA enlargement (group with pathological CSA (pUS) and group with normal CSA (nUS)) or the presence of axonal affection in the sensory conduction studies (group with axonal affection (pCS) and group without axonal affection (nCS)).

During the second phase, all patients underwent clinical evaluation with the help of the THS 6 months after initial evaluation. There was no difference in the treatment strategy between the study groups. A statistical comparison of the acquired THS data was performed in order to detect statistically significant differences.

2.3. Clinical examination

A scoring system was used to address the severity of hypesthesia caused by sural neuropathy (Thessaloniki Hypesthesia Score, THS, Table 1). Patients with no hypesthesia received 1 point, patients with mild hypesthesia, not limiting facilities in everyday life, received 2 points, patients with severe hypesthesia, limiting facilities in everyday life received 3 points.

To avoid selection bias between physicians and patients, the questionnaires were administered by a medical assistant (P.N.) of our department, who was not involved in the patient selection or management.

2.4. Ultrasound examination

Ultrasonography was performed by a board certified radiologist (G.B.). All ultrasound studies have been performed using a Mindray M7 ultrasound system (Mindray medicals, China). For the bilateral sonography of the sural nerve, a 14-MHz linear array transducer was used. The transducer was always kept perpendicular to the nerves, to avert anisotropy. No additional force was applied other than the weight of the transducer, and the extremities were kept in neutral position, to avoid causing any artificial nerve deformity. Cross-sectional area measurements were performed at the inner border of the thin hyperechoic epineural rim by the continuous tracing technique, and the average values were calculated after serially measuring three times. Cross-sectional area enlargement was defined as CSA values > mean CSA + 2SD. The sural nerve was evaluated in all healthy subjects bilaterally at 3 anatomical sites: (1) between the two heads of the gastrocnemic muscle (14 cm proximal to malleolus externus), (2) 7 cm proximal to malleolus externus, (3) at malleolus externus (Fig. 1). Only the anatomical sites showing cross sectional area enlargement were recognised as possible sites of lesion.

The intrarater reliability of the examiner (G.B.) was determined with the help of the dependability coefficient (phi) after measuring the CSA of the sural nerve in a single healthy control over 5 consecutive days.

2.5. Nerve conduction studies

All electrophysiological studies were performed by a board certified neurologist (A.K.) with the use of a 4-channel electromyography device (Deymed, Czech Republic). All testing was done while maintaining a skin temperature of 34 °C. Sensory conduction studies included bilateral sural sensory nerve, superficial fibular sensory nerve, and/or medial plantar mixed nerve action potentials. All sensory nerve action potentials (SNAP) were recorded after antidromic stimulation and signal averaging, in order to improve the signal-to-noise ratio.. The SNAP was defined as abnormal when it was absent unilaterally or if the involved/healthy side amplitude ratio was < 50%. Skin temperature was maintained at > 30C.

2.6. Statistics

An Anderson-Darling Normality Test was performed in all study groups, to check the distribution of the samples. All study groups (pUS, nUS, pCS, nCS) showed a normal distribution according to the Anderson–Darling Normality Test ($p = 0.142$, $p = 0.201$, $p = 0.125$ and $p = 0.152$ respectively), so that statistical comparison of groups and correlation analysis were performed with the help of the Student's *t*-test using SPSS 17.0 for Windows. Only $p < 0.001$ values were accepted as statistically significant.

3. Results

Overall, 23 healthy subjects (mean age 49.3, SD ± 12.3, 11 women) and 46 sural nerves were examined. The CSA reference values of the sural nerve are reported in Table 2.

Overall, 15 patients with symptoms and clinical signs of TSN, that were referred to our department between January 2014 and September 2017, were initially sonographically and electrophysiologically evaluated. Among them, 1 patient having paraneoplastic polyneuropathy and 1 patient with severe obesity (BMI = 42) were excluded from the study. 13 patients fulfilling the inclusion criteria of TSN (mean age 51.2, SD ± 10.7, 5 women) were enrolled in the study.

During initial evaluation, the 13 patients showed a mean THS of 2.6 (SD ± 0.9) (see Table 3). Among the 13 patients with TSN, 7 showed a pathological CSA enlargement (pUS group), while 6 patients showed normal CSA (nUS group). In the pUS group the site of lesion was recognised in 2 patients 7 cm proximal to malleolus and in 5 patients 14 cm proximal to malleolus, while no patient showed signs of lesion at the malleolus externus (see Fig. 1). The causative lesion was in 5 patients iatrogenic (4 cases with neuroma formation after stripping of the small saphenous vein and 1 case with neuroma formation after achilles tendon surgery), while in 2 patients direct trauma without neuroma formation after ankle sprain was documented. The electrophysiological evaluation showed an axonal affection of the sNAP in 8 patients (pCS group), while 5 patients had normal amplitude of the sNAP (nCS group).

During phase 2, the pUS group showed a mean THS of 3.1 (SD ± 0.9), and the nUS group a mean THS of 1.8 (SD ± 0.7) ($p < 0.001$). On the other hand, the pCS group showed a mean THS of 2.8 (SD ± 0.7), and the nUS group a mean THS of 2.1 (SD ± 0.9) ($p = 0.035$).

4. Discussion

In this study, we evaluated the prognostic value of cross-sectional area enlargement and axonal damage in patients with TSN.

The sural nerve is characterised by great anatomical and topographical variability. Normally, it arises as a cutaneous branch from the medial popliteal nerve, in the distal half of the popliteal fossa. It is then joined by the peroneal communicating branch of the common peroneal