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ORIGINAL ARTICLE

Ultrasonographic evaluation of sciatic nerves in patients with spinal cord injury

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Study design: Cross-sectional, controlled study.
Objective: To evaluate the sciatic nerves of subjects with spinal cord injury (SCI) by using ultrasound (US) imaging and to explore whether US measurements are associated with clinical and electrophysiological findings.
Setting: National Rehabilitation Center in Ankara, Turkey.
Methods: Fifteen SCI subjects (12 male (M), 3 female (F)) and 23 (16 M, 7 F) healthy controls were included in the study. After clinical assessment of the subjects, lower limb nerve conduction studies and US imaging of the sciatic nerves were performed. Cross-sectional area (CSA) values of the sciatic nerves were correlated with the clinical and electrophysiologic data.
Results: Mean CSA values were lower in the patient group when compared with the control group ($P=0.042$). Reduced compound motor action potentials regarding tibial and peroneal nerves were observed in the patient group ($P=0.003$ and $P=0.005$, respectively). US measurements did not correlate with the electrophysiological findings. However, sciatic nerve CSA values were positively correlated with body mass index in the control ($r=0.534$, $P<0.05$) and patient ($r=0.482$, $P<0.05$) groups.
Conclusion: Sciatic nerves seem to be smaller in subjects with SCI. Together with our electrophysiological data, this preliminary finding could possibly be attributed to primary axonal loss.
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INTRODUCTION

The involvement of central nervous system with spinal cord injury (SCI) cases is well-documented.[1] Either due to the initial trauma or to other subsequent causes, structural damage in the upper motor neurons ensues. On the other hand, although in other neurological diseases—primarily involving the central nervous system—peripheral nerve problems have been reported,[2] similar studies concerning the paralytic limbs of subjects with SCI are few. Further, they are usually confined to electrophysiological assessment and axonal damage has been shown as the main pathological finding.[3–7]

Owing to its several advantages (that is, inexpensive, convenient, dynamic, radiation-free and has high resolution), ultrasound (US) imaging has been proven useful in the evaluation of peripheral nerves in various conditions.[8–11] In addition, majority of the peripheral nerves can easily be scanned throughout their anatomical courses.[12,13] Further, it has already been reported that peripheral nerves might undergo morphological changes (more enlargement in demyelinating neuropathies than axonal ones, and enlargement at proximal (and sometimes distal) to the site of an entrapment region) in clinical conditions whereby the primary pathology is actually elsewhere in the peripheral nervous system.[14,15] However, to the best of our knowledge, there is paucity of evidence suggesting the use of US imaging of lower limb peripheral nerves in subjects with SCI.

In this regard, keeping in mind the aforementioned pathological changes in relevant cases, we reasoned that morphological (in addition to electrophysiological) assessment of these nerves would be noteworthy as well. Accordingly, in this study, we aimed to evaluate the sciatic nerves of subjects with SCI using US imaging. We also tried to find out whether sciatic nerve measurements were related to clinical and electrophysiological findings of these subjects.

MATERIALS AND METHODS

This study consecutively included 17 (14 male (M), 3 female (F)) SCI subjects who had been hospitalized in our rehabilitation clinic between January 2013 and December 2013 and 23 (16 M, 7 F) healthy control subjects who had been referred to our electromyography laboratory for upper limb entrapment neuropathies in the same time period. Electrodiagnostic screening was performed in three limbs to rule out the diagnosis of polyneuropathy in control subjects. All paraplegic subjects with SCI were included but those who had a previous diagnosis of polyneuropathy (or pertinent diseases that could cause polyneuropathy) or a peripheral nerve entrapment syndrome, and lower motor neuron disease (that is cauda equina syndrome) were excluded. The study protocol was approved by the local ethics committee. We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research.

Subjects were clinically/functionally assessed according to the American Spinal Injury Association (ASIA) Impairment Scale (AIS) and Functional Ambulation Category.[16,17] Spasticity was evaluated using modified Ashworth scale.

Electrophysiological tests were performed by the same physiatrist using a Keypoint Dantec 4c machine (Dantec, Skovlunde, Denmark). Compound muscle action potential (CMAP), distal latency, motor nerve conduction velocity of tibial and peroneal nerves and sensory nerve action potential, latency and sensory nerve conduction velocity of sural nerves were measured using a standard protocol as described by Kamradt *et al.*[7] CMAP was recorded by using skin surface electrodes with a tendon-belly method. The peroneal nerve was stimulated at two different sites (ankle and distal to fibular head) and

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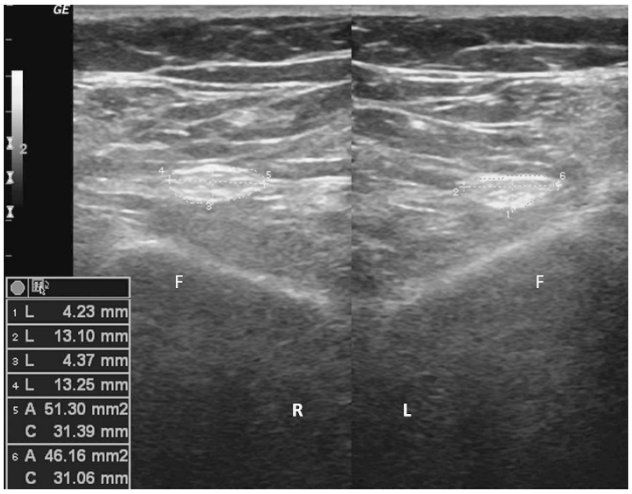

Figure 1 Bilateral ultrasonographic imaging (axial view, mid-thigh) of the sciatic nerves demonstrating the measurements of short axis, long axis and cross-sectional areas. F, femur; L, left; R, right.

CMAPs were recorded from the extensor digitorum brevis muscle. The tibial nerve was stimulated at two sites (medial malleolus and popliteal fossa), and CMAPs were taken from the abductor hallucis muscle. Sensory nerve action potential of the sural nerve was measured via antidromic stimulation of the sural nerve at the calves (recorded with skin surface electrodes from the posterior aspect of the lateral malleolus). Electrodiagnostic evaluations were performed bilaterally in SCI patients and unilaterally (from the nondominant side) in the control group. The reason why we have used the nondominant side was actually to avoid any possible extrinsic factors that might have been associated with limb overuse.

Ultrasonographic evaluations of the sciatic nerve were performed by the same physiatrist (with >10 years of experience in musculoskeletal US) using a 7–12-sMHz linear probe (Logiq P5, GE Medical Systems, Milwaukee, WI, USA). US measurements were taken bilaterally in SCI subjects and only using the nondominant side in the control group. During imaging, subjects lied in prone position on the examination table. Axial scanning of the nerve (perpendicular to its course) in a craniocaudal direction starting from the subgluteal fold to the popliteal fossa was done. Short and long axis diameters and cross-sectional area (CSA) of sciatic nerve were measured at mid-thigh (Figure 1).

Statistical analysis was performed by using SPSS 16.0 (Chicago, IL, USA). Data are expressed as mean ± s.d. Comparisons between patients and control subjects were performed using Mann–Whitney *U*-test (for nonparametric variables) and independent sample *t*-tests (for parametric variables). After exclusion of two subjects (with SCI below L1 level), sciatic nerve comparisons were made as 30 nerves (15 SCI subjects) vs 23 nerves (23 healthy controls). Correlations among clinical findings and electrophysiological/US measurements were evaluated by Pearson (for parametric variables) or Spearman (for nonparametric variables) rank coefficients, where appropriate. Statistical significance was set at $P<0.05$.

RESULTS

Demographic and clinical characteristics of the subjects are given in Table 1. All SCI subjects were paraplegic (two subjects with AIS-C had only anal contraction). Body mass index (BMI; 23.0 ± 2.3 vs 24.2 ± 2.6 kg m^{-2}) and gender (12 M, 3 F vs 16 M, 7 F) were similar between patients and control subjects ($P=0.152$ and $P=0.470$, respectively). Electrophysiological and ultrasonographic findings were similar between the right and left sides of SCI subjects (all $P>0.05$). The mean age of control subjects (51.7 ± 14.3 years; range: 25–72 years) was greater than that of SCI subjects' (34.5 ± 11.7 years; range: 20–60 years; $P=0.001$).

Table 1 Clinical characteristics of the patients

Age (years)	34.5 ± 11.7
BMI (kg m^{-2})	23.0 ± 2.3
Gender (F/M)	3/12
Duration of SCI (months)	10 (2–77)
Modified Ashworth scale	1 (1–3)
FAC	1 (0–2)
Level of injury	
C1–C8	2 (13.3)
T1–T12	11 (73.4)
L1	2 (13.3)
Completeness of injury	
Complete (AIS-A)	7 (46.7)
Incomplete (AIS-B)	6 (40)
Incomplete (AIS-C)	2 (13.3)

Abbreviations: AIS, American Spinal Injury Association Impairment Scale; BMI, body mass index; F, female; FAC, functional ambulation category; M, male.
The values are shown in mean ± s.d., median (min–max), number (%).

Table 2 Ultrasonographic measurements of the sciatic nerves (mean ± s.d.)

	SCI group (N = *30*)	*Control group* (N = *23*)	P-*value*
Short axis (mm)	5.1 ± 1.0	5.3 ± 2.1	0.642
Long axis (mm)	10.4 ± 2.1	11.7 ± 3.2	0.079
CSA (mm^2)	42.0 ± 10.3	49.1 ± 14.4	0.042

Abbreviations: CSA, cross-sectional area; SCI, spinal cord injury.

Table 2 summarizes the US measurements of the participants. Sciatic nerve CSA values were lower in the patient group when compared with the control group ($P=0.042$). Reduced CAMPs of tibial and peroneal nerves were observed in the patient group ($P=0.003$ and 0.005, respectively).

Although the US measurements did not have any correlation with the age, disease duration and clinical/electrophysiological findings, Spearman correlation analyses have revealed that CSA values were positively correlated with BMI in both the control ($r=0.534$, $P<0.05$) and patient ($r=0.482$, $P<0.05$) groups. CMAP values of tibial and peroneal nerve did not have any correlation with disease duration.

DISCUSSION

In this study, we aimed to explore whether lower limb peripheral nerves of SCI subjects were different from those of healthy controls. Our results have shown that sciatic nerve CSA values were smaller in the SCI group.

In subjects with SCI, sciatic neuropathy has been mentioned to be the most common neuropathy of the lower limbs.[3] On the other hand, it has also been reported that SCI could be related to polyneuropathy of the paralysed limbs and that peripheral motor axons below the level of the lesion showed degeneration.[4,6,7] The relevant mechanism (referred as trans-synaptic degeneration[4]) is thought to be due to the disconnection of the second motor neurons from the central nervous system.[4] Neurogenic disturbance and trans-synaptic changes ensue in spinal α-motor neurons after the central disconnection and these neurons become functionally inactive (unexcitable).[4,18] Depending on this dysfunction, distal axonal transport may also be impaired

leading to axonal degeneration/loss. Likewise, our results—decreased tibial and peroneal nerve CAMPs—are consistent with axonal loss. However, keeping in mind the fact that proximal nerve enlargement is usually observed in peripheral entrapment syndromes,[11,14] our finding of decreased CSA values seem to reflect primary axonal loss rather than secondary entrapment. Moreover, in our study neither CSA values nor CMAP amplitudes had any correlation with disease duration in subjects with SCI. According to our knowledge, there is the first study evaluating the correlation between CSA of sciatic nerves and disease duration. On the other hand, in a multicenter study evaluating CMAP amplitude changes in 345 patients with tetraplagia, it has been found that the lowest CMAP levels were found between 5 and 9 months post injury and during second 6 months following SCI, partial recovery of CMAP amplitudes was observed.[4] The absence of correlation between disease duration and CSA values or CMAP amplitudes could stem from the relatively wide range of disease duration (2–77 months) of the patients in our study and also the from small number of our subjects.

The relatively small number of subjects and statistical difference between the mean age values of the groups (higher in the control group) can be considered as the two major limitations of our study. Herewith, an overall analysis of the pertinent literature yields consistent results concerning the relationship between age, BMI and sciatic nerve CSA.[19,20] In one study including 60 participants (mean age: 45.9 years; range: 21–80 years), it has been reported that sciatic nerve CSA at mid-thigh was weakly correlated with age ($r=0.27$, $P=0.04$) and moderately correlated with BMI ($r=0.41$, $P=0.001$).[19] Another study with 60 healthy subjects (median age: 47 years; range: 18–81 years) revealed that age was weakly correlated with sciatic nerve CSA at piriformis level, whereas BMI was strongly correlated.[20] Similarly, our data also revealed that (mid thigh) sciatic nerve CSA values positively correlated with BMI but not with age. In addition, as patient and control groups were similar regarding BMI (with a narrower age range when compared with the aforementioned studies), we imply that the age difference between the groups would not significantly cloud our findings.

CONCLUSION

To summarize, in the light of our findings, we may conclude that sciatic nerves of SCI subjects seem to be adversely affected—both morphologically and electrophysiologically. Future studies with larger samples may provide better insight into understanding the association between those (acute/chronic) structural changes and the functional status of SCI subjects. Of note, US seems to be quite contributory in the evaluation of peripheral nerves in that sense.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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