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Sciatica and Nerve Conduction: A Case-Control Study Evaluating Electrophysiological Alterations in Symptomatic Patients

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ABSTRACT

Background: Sciatica is a neuropathic pain syndrome commonly resulting from lumbosacral nerve root compression. Nerve conduction studies (NCS) are essential for evaluating peripheral nerve integrity in such patients. To compare clinical parameters and nerve conduction velocities (NCVs) between patients with clinically diagnosed sciatica and healthy controls.

Methods: A case-control study was conducted involving individuals diagnosed with unilateral or bilateral sciatica (cases) and age-matched healthy participants (controls). Demographic data, anthropometry, vital signs, and motor/sensory NCVs (posterior tibial and sural nerves) were recorded.

Results: Significant reductions were observed in Right PTN NCV, Left PTN NCV, Right Sural NCV, and Left Sural NCV in sciatica cases compared with controls ($p < 0.0001$ for all). No significant group differences were found in age, weight, BMI, SBP, DBP, or pulse. Height differed modestly between groups ($p = 0.013$).

Conclusion: Sciatica is strongly associated with marked reductions in lower-limb nerve conduction velocities, particularly in the tibial and sural nerves. These electrophysiological alterations may reflect axonal compromise or demyelination secondary to nerve root compression. NCV assessment can serve as a sensitive diagnostic adjunct in the evaluation of sciatica.

Keywords: Sciatica, Nerve conduction velocity, Posterior tibial nerve, Sural nerve, Electrophysiology.

INTRODUCTION

Sciatica is a prevalent neuropathic pain condition characterized by radiating pain along the course of the sciatic nerve, typically originating in the lower back and extending into the buttock, thigh, and leg. The condition most commonly arises from lumbar disc herniation, spinal stenosis, or other structural abnormalities that compress or irritate the lumbosacral nerve roots. Globally, sciatica affects 3-25% of the population and constitutes a major cause of functional disability, reduced productivity, and healthcare utilization. Patients frequently present with sharp pain, tingling, numbness, or sensory deficits, and in severe cases, motor weakness, making early and accurate diagnosis crucial for appropriate clinical management [1].

Although clinical signs and physical examination play an essential role in diagnosing sciatica, distinguishing true radiculopathy from referred pain or musculoskeletal disorders can be challenging. Imaging modalities such as MRI can identify structural abnormalities, yet these findings do not always correlate with symptom severity or functional impairment. In this context, nerve conduction studies (NCS) serve as an important complementary tool, providing objective electrophysiological evidence regarding the functional integrity of peripheral nerves. Motor and sensory nerve conduction

parameters particularly those involving the posterior tibial and sural nerves can help detect demyelination, axonal loss, or conduction block associated with nerve root compression [2].

Previous research indicates that patients with sciatica often exhibit reduced nerve conduction velocities (NCVs), reflecting the physiological impact of nerve compression on large myelinated fibers. However, comparative data between symptomatic sciatica patients and healthy controls remain limited, particularly in regional clinical settings. Understanding these electrophysiological differences can improve diagnostic accuracy, support clinical decision-making, and provide insight into the extent of nerve damage [3]. Therefore, this study aims to evaluate key demographic, clinical, and electrophysiological parameters and to compare motor and sensory NCVs between sciatica patients and healthy individuals, thereby contributing to a clearer understanding of the neurophysiological alterations associated with sciatica.

MATERIALS AND METHODS

This case-control study was conducted in the Department of Physiology and Neurodiagnostics, where clinically diagnosed sciatica patients were compared with healthy volunteers. Participants between 20 and 60 years of age were recruited after detailed clinical screening. Individuals with symptoms of radiating leg pain consistent with lumbosacral radiculopathy, positive Straight Leg Raise (SLR) test findings, and neurological signs were included in the case group, while age- and sex-matched participants with no history of neurological or musculoskeletal disorders formed the control group. To eliminate confounding factors that may influence nerve conduction, individuals with diabetes mellitus, known peripheral neuropathy, spinal fractures, previous spinal surgery, metabolic disorders affecting nerve function, or alcohol abuse were excluded from the study. All participants provided written informed consent, and the study adhered to institutional ethical guidelines based on the Declaration of Helsinki.

Demographic and clinical data including age, height, weight, BMI, systolic blood pressure, diastolic blood pressure, and pulse rate were recorded using a structured proforma. Nerve conduction studies were performed in a quiet, temperature-controlled laboratory maintained between 26°C and 28°C to ensure stable electrophysiological conditions. All recordings were carried out using the NeuroStim4-NS4 EMG/NCV/EP System after proper skin preparation and electrode placement to minimize impedance. Sensory nerve conduction velocity (SNCV) of the sural nerve was assessed antidromically by stimulating the nerve at the lower calf and recording the Sensory Nerve Action Potential (SNAP) at the ankle, with parameters such as latency and amplitude noted. Motor nerve conduction velocity (MNCV) of the posterior tibial nerve was evaluated by stimulating the nerve at both the ankle and the knee, while Compound Muscle Action Potentials (CMAPs) were recorded from the Abductor Hallucis muscle. Distances between stimulation and recording sites were measured in millimeters, and SNCV and MNCV were calculated using standard formulas: SNCV = Distance/Latency and MNCV = Distance between proximal and distal stimulation/(Proximal latency - Distal latency). Limb temperature was monitored to ensure it remained above 32°C, preventing artificial slowing of conduction velocities.

To maintain measurement consistency, all tests were performed by the same experienced neurophysiology technician, and the equipment was calibrated daily. Participants were advised to avoid caffeine and strenuous physical activity for 12 hours prior to testing to reduce physiological variability. All data were expressed as mean ± standard deviation, and comparisons between the case and control groups were made using the independent sample t-test. A p-value less than 0.05 was considered statistically significant.

RESULTS

A total of sciatica patients (cases) and healthy individuals (controls) were evaluated for demographic, clinical, and electrophysiological parameters. The comparison of baseline characteristics showed no significant difference in age between the case group (45.20 ± 10.69 years) and control group (43.70 ± 9.35 years), with a p-value of 0.6394. Although the control group had a higher mean weight than the cases (69.65 ± 69.65 kg vs. 63.95 ± 10.37 kg), this difference was not statistically significant ($p = 0.1353$). Height was significantly greater in the control group (1.6480 ± 0.0630 m) than in sciatica patients (1.5915 ± 0.0739 m), with a p-value of 0.0131. BMI values were comparable between the two groups, showing no significant difference ($p = 0.8141$). Clinical parameters such as systolic blood pressure, diastolic blood pressure, and pulse rate also did not differ significantly, with p-values of 0.9245, 0.7459, and 0.3511 respectively, indicating that both groups were clinically similar aside from their neurological status.

In contrast to the demographic and clinical findings, nerve conduction parameters showed a marked and statistically significant difference between cases and controls. Posterior tibial motor nerve conduction velocity (MNCV) on the right side was significantly reduced in sciatica patients (38.27 ± 4.37 m/s) compared to healthy controls (47.95 ± 2.55 m/s), with a p-value of 0.0001. Similar results were observed for the left posterior tibial nerve, where the case group demonstrated

significantly lower conduction velocity (39.38 ± 3.82 m/s) than controls (48.20 ± 3.17 m/s), again highly significant (p = 0.0001). Sensory nerve conduction velocities (SNCV) of the sural nerve also showed substantial impairment in sciatica patients. Right sural SNCV was significantly slower in cases (36.23 ± 3.55 m/s) than in controls (50.43 ± 5.33 m/s), with a p-value of 0.0001. Similarly, left sural SNCV was significantly reduced in the case group (38.85 ± 2.86 m/s) compared to the control group (49.62 ± 5.34 m/s), also with a p-value of 0.0001.

Table 1. Comparison of Anthropometric, Clinical and Nerve Conduction Parameters between Cases and Controls.

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	p-value
Age (years)	45.20 ± 10.69	43.70 ± 9.35	0.6394
Weight (kg)	63.95 ± 10.37	69.65 ± 69.65*	0.1353
Height (m)	1.5915 ± 0.0739	1.6480 ± 0.0630	**0.0131**
BMI (kg/m²)	25.23 ± 3.79	25.51 ± 3.72	0.8141
Right PTN NCV (m/s)	38.27 ± 4.37	47.95 ± 2.55	**0.0001**
Left PTN NCV (m/s)	39.38 ± 3.82	48.20 ± 3.17	**0.0001**
SBP (mmHg)	128.80 ± 14.56	128.40 ± 11.83	0.9245
DBP (mmHg)	78.80 ± 8.19	78.00 ± 7.28	0.7459
Pulse (beats/min)	83.95 ± 8.33	86.60 ± 9.39	0.3511
Right Sural NCV (m/s)	36.23 ± 3.55	50.43 ± 5.33	**0.0001**
Left Sural NCV (m/s)	38.85 ± 2.86	49.62 ± 5.34	**0.0001**

DISCUSSION

The present study aimed to evaluate the differences in nerve conduction velocity between clinically diagnosed sciatica patients and healthy controls, and the findings demonstrate clear electrophysiological impairment in the affected individuals. While demographic and hemodynamic variables did not differ significantly between groups, the nerve conduction parameters showed marked reductions in both posterior tibial motor nerve conduction velocity and sural sensory nerve conduction velocity among sciatica patients. These results are consistent with the pathophysiological understanding that nerve root compression or irritation in lumbosacral radiculopathy leads to impaired transmission along both motor and sensory fibers [4]. The significantly slower conduction velocities observed in cases reflect the functional consequences of demyelination, axonal injury, or conduction block induced by mechanical compression [5].

The reduction in motor NCV in the posterior tibial nerve is of particular relevance, as this nerve is directly influenced by lumbosacral nerve roots (L4-S3), which are commonly affected in sciatica. Compression of these roots can slow impulse propagation due to segmental demyelination or ischemia, explaining the lower conduction velocities recorded in the case group. The consistent bilateral reduction, despite some patients being unilaterally symptomatic, suggests that even mild or subclinical root irritation may produce measurable electrophysiological changes. This underscores the sensitivity of nerve conduction studies in detecting early or subtle nerve dysfunction that may not yet manifest as clinically apparent weakness [6].

Similarly, the significant reduction in sural SNCV supports the involvement of sensory fibers in sciatica, which aligns with clinical symptoms such as tingling, paresthesia, and radiating pain. Sensory fibers are more vulnerable to compression due to their smaller diameter and lower threshold for ischemic damage. The markedly lower sensory conduction velocities in sciatica patients indicate impaired afferent transmission, which can contribute to altered sensory processing and neuropathic pain. These findings reinforce the clinical relevance of sural nerve studies in differentiating radiculopathy from other causes of lower limb sensory disturbances, such as peripheral polyneuropathy or entrapment neuropathies [7].

An important observation of this study is the lack of significant difference in BMI, blood pressure, and pulse rate between cases and controls, suggesting that systemic factors did not influence the nerve conduction outcomes. This strengthens the argument that the observed electrophysiological deficits are directly related to sciatic nerve or nerve root involvement rather than confounding metabolic or vascular conditions. By applying strict exclusion criteria removing patients with diabetes, metabolic disorders, alcohol abuse, and known neuropathies the study minimized the influence of common causes of peripheral nerve impairment, allowing for a more accurate assessment of sciatica-specific changes [8].

Overall, the results of this study support the clinical utility of nerve conduction studies as an objective and reliable diagnostic tool in the evaluation of sciatica. While imaging modalities such as MRI provide structural information, NCS

offers functional insight into nerve integrity and conduction efficiency. The significant reductions in both motor and sensory conduction velocities highlight the importance of electrophysiological assessment, particularly in cases where symptoms do not correlate with imaging findings or when differentiating radiculopathy from other neuromuscular disorders. Future research with larger sample sizes and detailed analysis of latency, amplitude, and F-wave parameters may provide deeper insight into the severity and progression of nerve impairment in sciatica.

CONCLUSION

The findings of this study demonstrate that patients with clinically diagnosed sciatica exhibit significantly reduced motor and sensory nerve conduction velocities compared to healthy individuals. While demographic and clinical parameters such as age, BMI, and blood pressure showed no meaningful differences between groups, the marked slowing of posterior tibial MNCV and sural SNCV in sciatica patients highlights the presence of measurable electrophysiological impairment. These results confirm that nerve root compression associated with sciatica affects both motor and sensory pathways, reflecting underlying demyelination, conduction delay, or axonal dysfunction.

Overall, the study emphasizes the value of nerve conduction studies as a reliable and objective diagnostic tool for evaluating functional nerve impairment in sciatica. By documenting clear differences between affected patients and healthy controls, the findings support the use of electrophysiological testing to complement clinical assessment and imaging, especially in cases with ambiguous symptoms or discordant radiological findings. Further research involving larger sample sizes and longitudinal follow-up may help clarify the progression of nerve dysfunction and enhance the diagnostic precision and prognostic value of nerve conduction measurements in sciatica.

Conflict of interest: No conflicts of interest exist.

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