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ORIGINAL RESEARCH ARTICLE

# Bilateral sciatic nerve impairment in patients with spine-related leg pain: a case-control study

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## ABSTRACT

**Introduction:** Unilateral spine-related leg pain is a common peripheral neuropathic pain condition. Patients with unilateral spine-related leg pain may present with bilateral nerve impairments. Comparing both sides of a patient with a matched control could clarify whether the sciatic nerve is impaired bilaterally. This study aimed to compare the cross-sectional area (CSA) and skin-to-nerve distance (SKN) of the sciatic nerve between patients with spine-related leg pain and matched asymptomatic controls.

**Methods:** A case-control study was conducted with 31 participants presenting with unilateral chronic probable peripheral neuropathic pain, and 31 matched asymptomatic controls. Ultrasound evaluation of the sciatic nerve was performed at the posterior mid-thigh under two tension conditions: hip flexed with knee flexed (lesser tension position), and hip flexed with knee extended plus ankle dorsiflexion (greater tension position). Group comparisons were performed with linear mixed-effects models.

**Results:** Patients with unilateral spine-related leg pain demonstrated significantly greater CSA bilaterally compared to controls ($p < 0.01$). Within-patient comparisons revealed no significant differences in CSA between symptomatic and asymptomatic sides (p-values from 0.38 to 0.74). CSA decreased under greater neural tension, which was statistically significant in the spine-related leg pain group ($p < 0.01$) but not in controls ($p = 0.06$). SKN analysis showed significant group and positional effects ($p < 0.01$), with controls presenting smaller values than patients in both positions.

**Conclusion:** Patients with chronic unilateral spine-related leg pain demonstrated bilateral impaired morphology (increased CSA) and impaired response to increased tension (decreased SKN) compared to matched controls.

**Keywords:** Case-control studies, Sciatic neuropathy, Sciatic nerve, Ultrasonography

**What's already known about this topic?**

- *Ultrasound can detect sciatic nerve morphological changes in unilateral spine-related leg pain. Contralateral limbs are commonly used as a normal reference. Bilateral nerve impairment has been reported in other peripheral neuropathic conditions, such as carpal tunnel syndrome and cervical radiculopathy.*

**What does this study add?**

- *Patients with chronic unilateral spine-related leg pain show bilateral sciatic nerve enlargement and impaired neurodynamic response, indistinguishable between sides. Clinicians must be aware that neurological impairments may be present in asymptomatic limbs in certain conditions.*

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## Introduction

Chronic low back pain with neuropathic characteristics represents a significant clinical challenge, accounting for approximately one-third of the point prevalence of physician-diagnosed probable neuropathic pain in Brazil (1). Within this spectrum, spine-related leg pain (i.e., sciatica) constitutes a common and clinically relevant entity (2),


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typically resulting from compression of the lumbosacral nerve root (3,4). Establishing a neuropathic component in sciatica requires integrating clinical features with confirmatory neurological signs, in accordance with established international consensus-based diagnostic frameworks for neuropathic pain (5). These criteria classify neuropathic pain as possible, probable, or definite based on the level of available clinical and neurophysiological evidence. The diagnostic frameworks support clinicians in diagnosing neuropathic pain to facilitate the best pain management.

Ultrasound imaging (USI) has emerged as a valuable tool for the structural and dynamic assessment of peripheral nerves. Structural alterations in the peripheral nerves detected by USI include changes in cross-sectional area (CSA). For instance, USI frequently reveals pathological enlargement of the nerve, associated with increased intraneural vascularisation, fascicular architectural disruption, and reduced neural mobility within the surrounding tissues (6). Recent studies establishing reference values for the CSA of the sciatic nerve have reported mean values of approximately 44-53 mm², depending on the anatomical level of measurement, typically between 1 cm and 4 cm proximal to the sciatic nerve bifurcation (7). Nonetheless, there is no universally accepted set of values to define abnormal sciatic nerve CSA due to individual anatomical differences, population characteristics, and methodological variability in USI (8,9). Accordingly, sciatic nerve CSA interpretation should be performed preferentially by comparing the symptomatic side with the asymptomatic contralateral side, an approach considered more sensitive than using absolute reference values (10,11). In patients with spine-related leg pain, significant side-to-side differences in sciatic nerve CSA have been reported, with larger values on the symptomatic side than on the contralateral limb (12). Similarly, studies in other unilateral neuropathic conditions (e.g., moderate carpal tunnel syndrome) showed bilateral impairment in motor function when compared to controls (13). These findings suggest that unilateral nerve involvement may induce broader neural adaptations extending beyond the clinically affected limb (14).

Measurement of the skin-to-nerve distance (SKN) using USI reflects the anteroposterior displacement of the nerve under tension, functioning as an indicator of neurodynamic mobility (15). In response to movements that increase neural tension, such as increased dorsiflexion, the nerve shifts to a more superficial position, representing an adaptation to mechanical loading (15). Some studies in anaesthesia or interventional techniques have reported skin–nerve distances for the sciatic nerve ranging from 3.0 to 5.5 cm using specific approaches (16). However, there is no reliable, widely accepted normative value for SKN at the mid-thigh level in healthy populations, as reported values vary substantially with anthropometric characteristics (17).

Although CSA and SKN have been previously investigated in patients with spine-related leg pain, it remains unclear how these parameters behave or adapt when the condition progresses to a chronic phase. Previous research has focused on CSA in upper-limb conditions and cervical radiculopathy. Similar bilateral CSA values were observed at the peripheral nerve level in patients with chronic cervical radiculopathy (18), suggesting that morphological changes in peripheral nerves may be symmetrical in some conditions. However, whether the sciatic nerve exhibits comparable adaptive or compensatory remodelling in individuals with chronic spine-related leg pain remains to be established, particularly regarding potential alterations in its morphology (measured by CSA) and response to increased tension (measured by SKN). Therefore, the present study compared the CSA and SKN of the sciatic nerve between participants with spine-related leg pain and matched asymptomatic controls. We hypothesized that individuals with spine-related leg pain would show bilateral increases in the CSA of the sciatic nerve and reduced SKN compared to asymptomatic controls, as well as a different response to neural tension variation between lower- and higher-tension positions compared to asymptomatic controls.

## Materials and methods

### *Study design*

A case-control study was conducted, comparing a sample of patients with spine-related leg pain from a previous study by our group (19) with a matched sample of asymptomatic individuals. We prospectively recruited asymptomatic adults matched for age, sex, and assessment side. This study was approved by a Research Ethics Committee (number CAAE: 94093225.0.0000.5235), and all participants included signed the written informed consent form before enrolment. This study is reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist for case–control studies (20).

### *Participants with spine-related leg pain*

Participants aged between 18 and 65 years were recruited from April 2022 to October 2023. We screened participants with at least moderate unilateral leg pain (i.e., score greater than three on the Numerical Pain Scale out of ten) (21), persistent or recurrent, lasting more than three months, and greater leg pain intensity than in the lumbar region, with signs and symptoms related to L4, L5, or S1 nerve root distribution. In addition, participants were eligible if they were classified as having unilateral chronic probable peripheral neuropathic pain based on clinical examination findings, according to the International Association for the Study of Pain Special Interest Group on Neuropathic Pain (the coexistence of symptoms and objective neurological signs—such as hypoesthesia, allodynia, motor deficits, or reflex abnormalities—suggesting involvement of the somatosensory system) (5).

We excluded patients with self-reported: a) economic compensation due to sick leave; b) undergoing treatment due to low back pain; c) pregnant or breastfeeding women on the date of assessment; d) history or presence of serious spinal pathologies, such as cauda equina syndrome, infection, cancer or fracture; e) history of previous surgery on the lumbar spine, lower limbs, or abdominal region in the last year; f) history of neuropathies secondary to trauma, surgery, or cancer treatment; g) current diagnosis of metabolic

or autoimmune diseases, herpes zoster, rheumatological pathologies in the acute inflammatory phase, oncological or psychiatric diseases.

### *Matched asymptomatic control group*

Asymptomatic adults were prospectively recruited and individually matched to each patient based on age, sex, and assessment side from February to September 2024. The matched control was evaluated on the same limb (left or right) as the patient's symptomatic side. For the control group, exclusion criteria included the presence of low back pain or lower limb pain within the past 12 months, the presence of mechanosensitivity assessed by sciatic nerve–related radiating pain below the knee at a straight leg raise angle of less than 70° (22), a history of systemic disease (such as metabolic or autoimmune diseases, herpes zoster, rheumatologic conditions in the acute inflammatory phase, oncological diseases, among others), and pregnant or breastfeeding women on the date of assessment.

### *Procedures*

Participants' characteristics (e.g., age, biological sex, body mass index) were registered. The current study focused on data comparing the morphology (measured by CSA) and response to increased tension (measured by SKN) from patients with spine-related leg pain and matched controls.

### *Ultrasound Imaging Acquisition*

All imaging acquisitions were performed by a research assistant with four years of experience in peripheral nerve imaging. Participants were positioned in a lateral decubitus posture. Based on previous studies demonstrating that combined knee extension and ankle dorsiflexion increase neural tension and modify sciatic nerve excursion (23,24), and knee flexion reduces tension (25), ultrasound images were acquired in two lower-limb positions, maintaining the hip flexed to 60°: (1) Lesser neural tension position - knee flexed to 90° with the ankle in a neutral position, and (2) Greater neural tension position - knee fully extended with the ankle in maximal dorsiflexion. Figure 1 illustrates the positioning used to evaluate the sciatic nerve.

Ultrasound parameters were acquired using the musculoskeletal preset of a linear transducer (Philips Lumify, Maryland, USA), 4–10 MHz in B-mode, depth of 10.0 cm, and a gain of 50 dB, positioned transversely over the sciatic nerve at the posterior mid-thigh level. The examiner measured the distance between the popliteal fossa and the ischial tuberosity and placed a Velcro strap at the midpoint of the thigh. When the sciatic nerve was difficult to identify in the short axis, scanning began at the tibial nerve in the popliteal fossa and progressed from distal to proximal (26). The sciatic nerve was identified, and two images were stored; one image was digitally annotated to facilitate subsequent confirmation of its localization.

### *Ultrasound Image Processing*

This study utilized an imaging database established in a previous investigation (19). Because the patient-group images originated from that earlier study, all ultrasound images from the patient group were re-analyzed. A single evaluator, who had not participated in the prior analysis and was blinded to symptomatic and asymptomatic sides, conducted the new assessment. This methodology ensured that all measurements were performed by one rater under standardized conditions, thereby addressing the operator-dependent variability inherent to ultrasound. Ultrasound image processing was conducted independently using ImageJ software (version 1.43; National Institutes of Health, Bethesda, MD, USA). ImageJ supports multiple image formats, enables area calculation, provides pixel-based statistics, and allows calibration in centimetres or millimetres (27). Prior to analysis, each image was calibrated by measuring the pixel count over a known 10 mm distance. The nerve CSA was delineated using the freehand selection tool, with tracing guided by the inner margin of the hyperechoic epineural layer. Area measurements were automatically computed based on the prior calibration (Fig. 1A). The SKN corresponded to the perpendicular

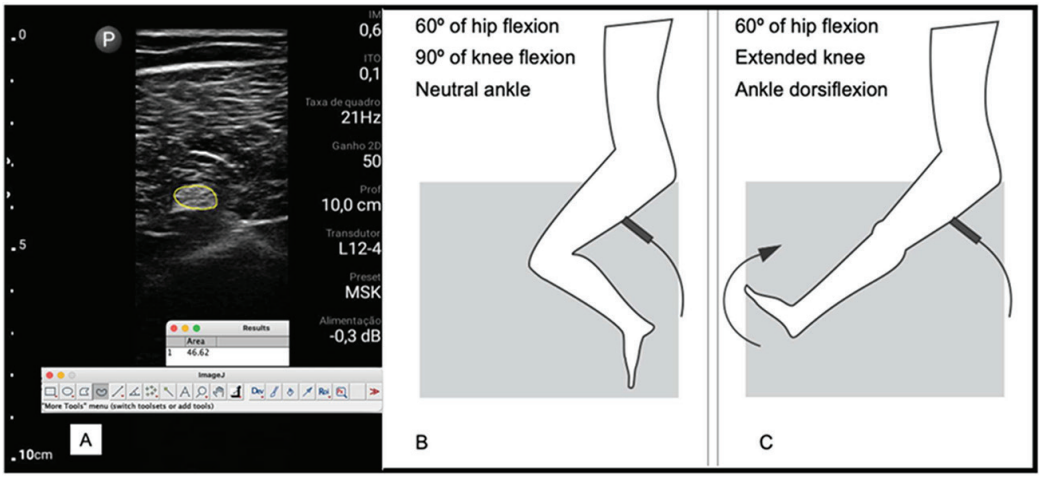



**FIGURE 1 -** Image acquisition of the sciatic nerve with transverse positioning of the transducer. Panel A shows image acquisition of the sciatic nerve with the area delineated. Panels B and C show positioning of the transverse transducer at mid-thigh during examination of the lesser and greater neural tension positions, respectively.

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distance from the skin surface to the superior inner margin of the hyperechoic epineural layer of the sciatic nerve, assessed with the straight-line selection tool and recorded in millimetres.

### *Sample size*

The sample size calculation was performed in the software G*Power version 3.1.9 (Heinrich-Heine-Universität, Düsseldorf, Germany) to determine a sufficient sample size. An effect size f of 0.25 (equivalent to $\eta p^2 = 0.059$, considered a medium effect) was estimated for the group-by-position interaction on sciatic nerve CSA. Based on a repeated-measures ANOVA with a within-between interaction, an alpha level of 5%, statistical power of 80%, two groups, two repeated measurements (lesser and greater tension positions), and a correlation among repeated measures of 0.1, a total of 60 participants (30 per group) was required. The final sample of 62 participants (31 per group) satisfied this requirement.

### *Statistical analysis*

Descriptive analysis was used to describe the participants' sociodemographic and clinical data. Continuous variables were expressed as means and standard deviations, while categorical variables were reported as absolute frequencies and percentages. Between-group differences in age and body mass index were compared using independent-samples t-tests, and sex distribution was compared using a chi-square test. The mean values of CSA ($mm^2$) and SKN (mm) were compared among the groups using a linear mixed-effects model to account for the non-independence of repeated measurements obtained from the same individual (e.g., bilateral measurements). The group × position interaction was assessed to examine whether the effect of tension position on outcome differed according to group (i.e., whether the symptomatic side, asymptomatic side, and controls responded differently to the change from lesser to greater tension). The main effects of group and position were examined to assess differences among the three groups (symptomatic side, asymptomatic side, controls) and between tension positions, respectively. The Tukey post hoc test was applied to identify the comparison pairs. All statistical tests were two-tailed, with the significance level pre-established at $p < 0.05$. Data analysis was performed using the JASP software (version 0.14.1, Amsterdam, The Netherlands) and GraphPad Prism software (version 10; GraphPad Software, San Diego, CA, USA) for Mac.

We conducted a sensitivity analysis due to an imbalance in body mass index between patients with spine-related leg pain and matched controls. Differences in the outcomes (CSA and SKN) between groups were analyzed using a linear mixed-effects model. The outcomes were specified as the dependent variable, with group (three levels), condition (lesser tension or greater tension), and body mass index included as fixed effects. To account for the non-independence of repeated measurements obtained from the same individual (e.g., bilateral measurements), participant identification (ID) was included as a random intercept. Models were estimated using restricted maximum likelihood (REML). Effect estimates are presented as regression coefficients with 95% confidence intervals, and statistical significance was set at $p < 0.05$.

## Results

A total of thirty-one matched asymptomatic controls were prospectively assigned to the 31 patients with chronic unilateral spine-related leg pain (Fig. 2). Groups had similar mean age (Participants in the spine-related leg pain group = 43.12 ± 10.85 years; asymptomatic group = 39.83 ± 11.29 years; $p = 0.244$). Body mass index was significantly higher in the spine-related leg pain group (30.72 ± 8.88 $kg/m^2$) than in the control group (25.80 ± 3.92 $kg/m^2$; $p = 0.007$). Most of the sample was composed of females [n = 22 (77.4%)].

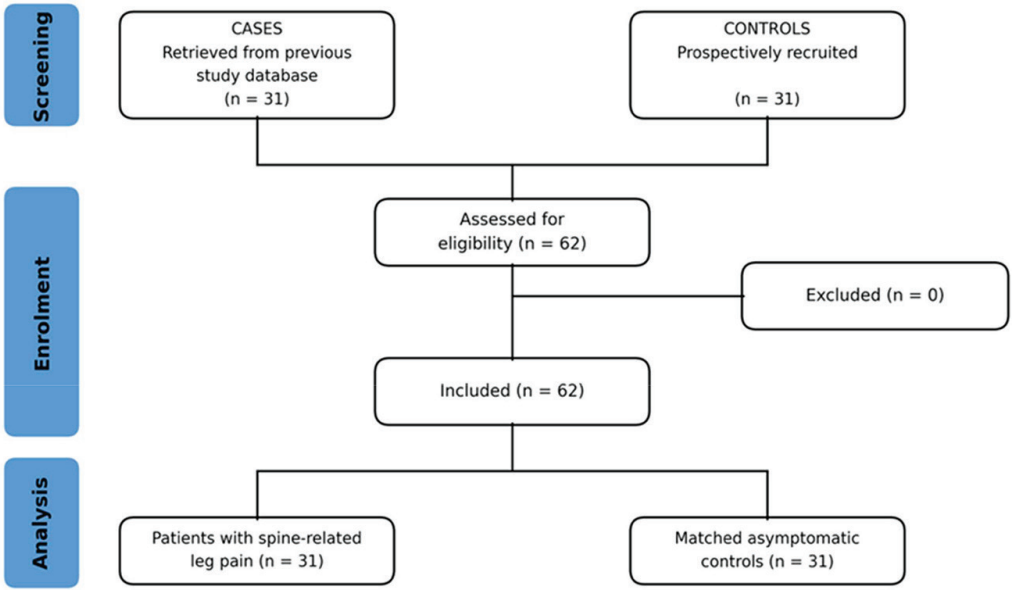



**FIGURE 2 -** Study flowchart. Participant flowchart from screening to analysis, following STROBE reporting guidelines for case-control studies.

The CSA means were higher in both sides of patients with spine-related leg pain (symptomatic side = 50.76 ± 10.03 $mm^2$ and asymptomatic side = 46.98 ± 13.89 $mm^2$) than in matched controls (31.36 ± 8.47 $mm^2$) under a less-tensioned position. The CSA means were also higher in both sides of patients with spine-related leg pain (symptomatic side = 40.75 ± 11.45 $mm^2$ and asymptomatic side = 38.68 ± 13.80 $mm^2$) than in matched controls (28.25 ± 7.29 $mm^2$) under a greater tension position. A linear mixed-effects model revealed a significant group x position interaction effect for CSA [$F(2,90) = 5.015$, $p < 0.01$], indicating that the change in CSA between tension positions differed across groups. Participants with spine-related leg pain showed significantly greater CSA than matched controls, irrespective of side or position (Tukey's post hoc test $p \leq 0.01$). No significant differences were observed between asymptomatic and symptomatic sides within participants in the two positions (Tukey's post hoc test $p > 0.30$). CSA decreased significantly from lesser to greater tension in participants with spine-related leg pain (Tukey's post hoc test $p < 0.01$), whereas the positional change in controls was not significant (Tukey's post hoc test $p = 0.06$) (Fig. 3).

Under the lesser tension position, SKN values were also higher on both sides of participants with spine-related leg pain compared with the matched asymptomatic controls. The asymptomatic side presented a mean SKN of 54.60 ± 9.86 mm, while the symptomatic side showed 51.75 ± 8.56 mm, in contrast to the lower mean value observed in the control group (39.07 ± 9.55 mm). This pattern was maintained under the greater tension position, where the asymptomatic side demonstrated a mean SKN of 48.60 ± 7.91 mm and the symptomatic side 47.51 ± 7.99 mm, both exceeding the value obtained in the matched controls (33.15 ± 7.28 mm). These results indicate consistently increased SKN in individuals with spine-related leg pain relative to asymptomatic participants, irrespective of the level of neural tension. A linear mixed-effects model revealed a non-significant group x position interaction effect for SKN [$F(2,90) = 1.469$, $p = 0.24$], indicating that the change in SKN between tension positions did not differ across groups. Significant main effects of position ($F(1,90) = 128.8$, $p < 0.01$) and group ($F(2,90) = 32.30$, $p < 0.01$) were found for the SKN. Post-hoc comparisons revealed that SKN was greater under lesser neural tension than under greater tension in all within-group comparisons. All statistical significance values obtained from comparisons between and within groups for both outcomes (CSA and SKN) are presented in Supplementary File 1.

Figure 3 shows the sciatic nerve CSA (left panel) and the SKN (right panel) in the symptomatic side, asymptomatic side, and matched controls under the two positions of lesser and greater neural tension. Participants with unilateral symptoms demonstrate bilaterally increased CSA compared to controls, suggesting morphological alterations that are not limited to the painful side. Regarding SKN, matched controls show a clear reduction in SKN under greater neural tension, consistent with normal anterior displacement of the nerve. In contrast, this tension-related reduction appears attenuated in the neuropathic pain group, indicating a potentially altered mechanical response to neural loading.

A sensitivity analysis was performed to examine whether the body mass index imbalance could account for the between-group differences in CSA and SKN reported above. In this adjusted model, body mass index was not significantly associated with CSA (β = 0.30, 95% CI −0.07 to 0.67; p = 0.115), indicating that the CSA differences between groups are unlikely to be explained by body mass index. In contrast, body mass index was significantly associated with

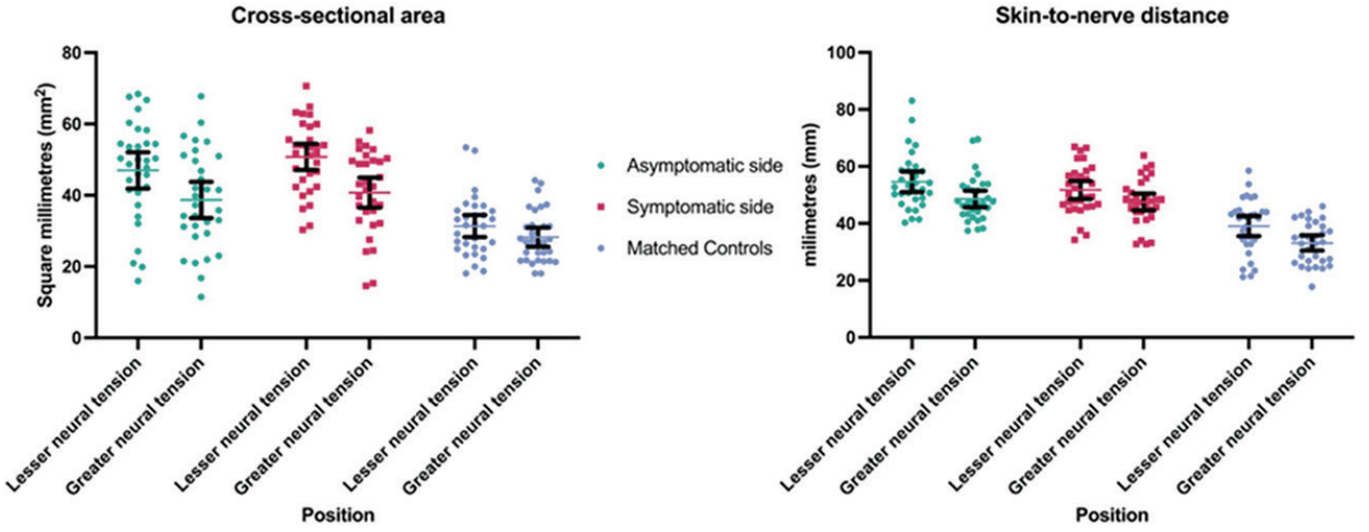



**FIGURE 3 -** CSA and SKN of nerves under lesser and greater neural tension across groups. Scatter plots show individual data points for sciatic nerve CSA (left) and SKN (right) under lesser and greater neural tension positions. Green circles represent the asymptomatic side, red squares the symptomatic side of patients with spine-related leg pain, and blue circles the matched controls. Horizontal lines and error bars represent the mean and 95% confidence interval for each group and position.

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SKN (β = 0.49, 95% CI 0.19-0.80; p < 0.01), suggesting that the observed group difference in SKN may be partly attributable to body mass index rather than to group status itself.

## Discussion

This study examined sciatic CSA and SKN in individuals with spine-related leg pain compared with matched asymptomatic controls. Participants with spine-related leg pain exhibited bilaterally significantly greater CSA than controls (i.e., without spine-related leg pain) in both positions. The control group showed the sciatic nerve to be more superficial in both positions when compared to the patients' symptomatic and asymptomatic sides. The control group sciatic nerve demonstrated a different response to the increase in tension in the CSA and a similar response to the SKN when compared to the patients' sides. The similarities between both sides of the patients and the differences compared to the control group suggest bilateral sciatic morphological alterations in patients with chronic spine-related leg pain.

Our findings align with previous reports in patients with chronic cervical radiculopathy (mean age of 39 ± 14.4 years; mean body mass index of 27.6 ± 5.2 kg/m²), who had similar peripheral nerve (median, ulnar, and radial nerves) CSA between the affected and unaffected sides (18). Experimental models of unilateral inflammatory and peripheral nerve injury have shown that a unilateral lesion may induce contralateral changes in the nervous system (28). These effects are mediated by neuroimmune processes, including cytokine-driven signalling between dorsal root ganglia (29,30), and involve both peripheral and central neural mechanisms (31).

Imaging evidence of neural alterations associated with unilateral radiculopathy has been described. Inflammatory signal and enlargement of the area of the median nerve of patients with whiplash-associated disorders were described (32). In patients with cervical radiculopathy, increased inflammatory activity at the level of the affected nerve root and dorsal root ganglion was described (33). Similarly, in chronic lumbar radicular pain, increased inflammatory signalling was identified in affected nerve roots and the spinal cord compared with healthy controls (34). These findings indicate that chronic spine-related pain is associated with quantifiable neuroinflammatory activity involving neural structures beyond simple mechanical compression. Taken together, the structural alterations observed on magnetic resonance imaging (32) and the inflammatory activity demonstrated through molecular imaging (33,34) support the interpretation that radiculopathy involves biological processes affecting neural tissue. Within this framework, the bilateral sciatic morphological alterations observed in our sample may reflect broader neural involvement in chronic spine-related leg pain rather than solely local structural compromise.

This pattern of bilateral or subclinical nerve adaptation has also been described previously in chronic neuralgic amyotrophy (35). In chronic neuralgic amyotrophy, comparisons between affected and unaffected sides revealed less pronounced but still significant CSA differences in C6 roots, the brachial plexus at interscalene and supraclavicular levels, and the median and radial nerves at the upper arm. Notably, when unaffected sides were compared with healthy controls, enlargements were observed in the C5 root and more robust differences in the median and radial nerves at the upper arm (35). Such observations demonstrate that morphological nerve changes can extend beyond clinically affected regions and even involve contralateral limbs, highlighting the potential for widespread adaptations in chronic peripheral neuropathic conditions.

The observed difference in SKN between groups should be interpreted with caution due to the imbalance in body mass index. Sensitivity analysis demonstrated a significant association between body mass index and SKN, with higher body mass index corresponding to greater SKN. Therefore, the SKN difference between patients and controls is partly attributable to variations in body mass index rather than solely to the presence of spine-related leg pain. This observation is consistent with previous ultrasound studies showing that peripheral nerve depth is influenced by body composition, particularly subcutaneous adipose tissue thickness, for which body mass index is commonly used as a surrogate marker (17,36,37). Therefore, although between-group differences in SKN were observed, the present data do not allow determination of whether these differences reflect nerve-related alterations or primarily variations in body composition and surrounding tissue characteristics.

We acknowledge the strengths and limitations of the present study. The inclusion of matched asymptomatic controls allowed for robust between-group comparisons, while within-subject assessments of symptomatic and contralateral limbs provided insights into potential bilateral adaptations. By evaluating both sciatic nerve CSA and SKN under different limb positions, the study captured complementary morphological and functional aspects of neural health, highlighting dynamic changes in nerve behaviour. However, the cross-sectional design precludes causal inferences regarding the relationship between spine-related leg pain and neural changes. Although the sample size was adequate to detect group differences, larger cohorts are needed to deeply explore potential moderating factors, such as pain duration, severity, and comorbidities. Participant classification relied solely on clinical assessment, which may have failed to detect structural abnormalities in control subjects or inaccurately represented symptom severity in cases. Future research should incorporate imaging-confirmed spinal pathology in both groups to address this potential confounding factor. Finally, while ultrasound assessment of SKN is reliable (24), it may not fully capture the three-dimensional complexity of nerve movement.

Future studies should explore longitudinal designs to clarify causal relationships between neuropathic pain and changes in nerve CSA and SKN. Besides, more diverse populations should also be studied to understand the influence of age, body mass index, comorbidities, health condition, and pain duration on the nerves' morphologic changes. Additionally, further investigation of bilateral or subclinical nerve adaptations may improve assessment strategies and guide targeted interventions in neuropathic conditions.

The present study findings have direct clinical relevance, as they demonstrate that neural alterations may extend beyond the symptomatic side, even in unilateral presentations.

Given that CSA and SKN alterations were observed bilaterally, our findings suggest that the contralateral (asymptomatic) limb may not serve as an appropriate normative reference when evaluating sciatic nerve morphology or neurodynamic behavior in patients with unilateral spine-related leg pain. Recognizing the possibility of bilateral or widespread neural adaptations can inform more accurate evaluations and support the design of targeted, individualized rehabilitation strategies, ultimately improving patient management and outcomes.

## Conclusion

Patients with chronic unilateral spine-related leg pain demonstrated bilateral impaired morphology (increased CSA) and impaired response to increased tension (decreased SKN) compared to matched controls.

## Disclosures

**Conflict of Interest**: The authors declare that they have no conflict of interest.

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**Data availability statement**: The non-identifiable data supporting this study's findings will be made available upon reasonable request by researchers and/or academic entities.

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