Quantitative Approach in Assessing Standard Brachial Plexus MRI for the Diagnosis of Multifocal Motor Neuropathy and Lewis–Sumner Syndrome

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Abstract

Introduction. *Among various chronic polyneuropathies, there are conditions that pose challenges for differential diagnosis: multifocal motor neuropathy (MMN) and Lewis–Sumner syndrome (LSS). The potential of magnetic resonance imaging (MRI) to objectively assess pathological changes in the nerve structures of brachial plexuses remains highly relevant for the diagnosis and differential diagnosis of MMN and LSS.*

The aim *of this study is to determine the diagnostic value of quantitative methods for assessing MRI signal intensity and thickness measurements of brachial plexus neural elements in differentiating LSS and MMN.*

Materials and methods. *The study included 59 patients: 26 diagnosed with MMN, 33 with LSS, along with 15 healthy volunteers.*

Results. *When comparing the combined patient group (regardless of diagnosis) with healthy controls, both nerve thickness and signal intensity coefficient were significantly higher in patients. Additionally, disease-specific threshold values for signal intensity coefficient were established for each condition.*

Conclusion. *The analysis of signal intensity coefficients LSS and MMN demonstrates that quantitative assessment of MRI signal intensity from the anterior rami of spinal nerves forming brachial plexuses provides additional diagnostic information about pathological changes and facilitates accurate diagnosis. This approach enables earlier initiation of pathogenetic therapy, reduces disability rates, and shortens the duration of patient incapacitation.*

Keywords: *dysimmune neuropathies; Lewis–Sumner syndrome; multifocal motor neuropathy; brachial plexus magnetic resonance imaging; signal intensity coefficient*

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Роль количественного подхода в оценке стандартной МРТ плечевых сплетений в диагностике мультифокальной моторной нейропатии и синдрома Льюиса–Самнера

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Аннотация

Введение. *Среди множества хронических полинейропатий выделяют нозологические формы, трудно поддающиеся дифференциальной диагностике: мультифокальную моторную нейропатию (ММН)и синдром Льюиса–Самнера (СЛС).Актуальным для диагностики и дифференциальной диагностики ММН и СЛС является вопрос о возможности объективизации при магнитно-резонансной томографии (МРТ) изменений нервных структур плечевых сплетений.*

Цель *исследования – определение роли количественных методов оценки интенсивности МР-сигнала и толщины нервных элементов плечевых сплетений для диагностики и дифференциальной диагностики СЛС и ММН.*

Материалы и методы. *В исследование были включены 59 пациентов: 26 – с диагнозом ММН, 33 – с диагнозом СЛС, а также 15 здоровых добровольцев.*

Результаты. *При сравнении группы пациентов (без деления по нозологиям) сгруппой здоровых испытуемых толщина и коэффициент интенсивности были значимо больше у пациентов по сравнению с контрольной группой. Кроме того, были определены пороговые показатели коэффициента интенсивности для каждой нозологии.*

Заключение. *Полученные результаты анализа коэффициента интенсивности при СЛС и ММН позволяют сделать вывод о том, что использование предлагаемого количественного способа оценки интенсивности МР-сигнала от передних ветвей спинно-мозговых нервов, формирующих плечевые сплетения, может позволить получить дополнительную информацию о наличии патологических изменений и ускорить постановку верного диагноза. Это будет способствовать более раннему назначению патогенетической терапии, снижению уровня инвалидизации и сокращению срока нетрудоспособности пациентов.*

Ключевые слова: *дизиммунные нейропатии; синдром Льюиса–Самнера; мультифокальная моторная нейропатия; магнитно-резонансная томография плечевых сплетений; коэффициент интенсивности*

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Introduction

Among numerous chronic polyneuropathies, several conditions stand out that pose diagnostic challenges due to overlapping clinical and neurophysiological features, yet differ in pathophysiology and, most critically, therapeutic approaches: multifocal motor neuropathy (MMN) and atypical (multifocal) chronic inflammatory demyelinating polyneuropathy (CIDP) – Lewis-Sumner syndrome (LSS) [1, 2]. Until the 1980s, these conditions were not distinguished as separate pathological entities and were considered under a single nosological category [3].

Subsequent intensive research on MMN and LSS led to breakthroughs in understanding these chronic dysimmune polyneuropathies, revealing presumed pathophysiological distinctions and establishing definitive diagnostic criteria [2, 4]. Separate diagnostic criteria were formulated for LSS by the European Academy of Neurology and the Peripheral Nerve Society in 2021 [4], and for MMN by the European Federation of Neurological Societies and the Peripheral Nerve Society in 2010 [2].

The key advancement that has influenced both the classification of these conditions and therapeutic approaches is the elucidation of the immunopathological mechanisms underlying LSS and MMN. MMN is now classified as a nodo/paranodopathy, as its pathogenesis involves dysfunction of sodium and potassium channels at the nodes of Ranvier (nodal regions) with preserved axonal myelin sheaths. The condition is attributed to an autoimmune response involving IgM-class antibodies targeting GM1 ganglioside, which is localized to the nodal regions of peripheral nerve axons [5, 6]. Persistent local damage (demyelination) leads to secondary axonal impairment and muscle atrophy due to denervation. In LSS, an atypical chronic inflammatory demyelinating polyneuropathy, diffuse damage to myelin sheaths occurs in both proximal and distal segments of motor and sensory fibers without clear selectivity [7].

Despite differing pathophysiology involving distinct levels, scales, and consequences of autoimmune inflammation, nerve conduction studies using electroneuromyography in MMN and LSS may reveal similar patterns of conduction blocks due to the presence of a demyelinating component. To date, no specific sonographic findings differentiating these two conditions have been described. The clinical presentation may be limited to general manifestations such as asymmetric upper limb nerve involvement with asymmetric flaccid distal paraparesis, while the key consistent distinguishing feature between LSS and MMN lies in their differential response to disease-modifying therapies: high-dose corticosteroid therapy and intravenous immunoglobulin may be effective in LSS, whereas plasmapheresis and corticosteroids exacerbate MMN, with intravenous immunoglobulin being the only effective treatment [8, 9].

Thus, accurate diagnosis and differentiation of these two chronic dysimmune neuropathies are crucial for determining appropriate therapeutic strategies.

MRI findings are included only as supportive criteria for diagnosing these conditions. MRI can detect thickening of brachial plexus elements exceeding 5 mm and provide qualitative assessment of signal intensity from nerve trunks using fluid-sensitive sequences with fat suppression (STIR, T2FatSat, etc.) [2, 4]. Increased signal intensity from brachial plexus elements is considered a probable indicator of pathology. However, the absence of MRI changes does not exclude diagnoses of LSS and MMN [10–13], while the interpretation of signal alterations remains subjective, dependent on radiologist expertise, and particularly challenging in cases of symmetric brachial plexus involvement or when differentiating between diagnoses [10–13]. MR signal intensity represents a relative value that may vary between patients and even in the same patient during longitudinal studies when using identical sequences. Direct measurement of signal intensity from neural structures is therefore methodologically flawed and inadmissible for evaluating pathological changes [14]. This necessitates the clinical need for objective methods to detect and quantify brachial plexus abnormalities on MRI when diagnosing and differentiating between MMN and LSS.

Today, alongside standard scanning protocols that assess morphological parameters (thickness, homogeneity, and MR signal intensity), additional modern techniques widely used in other organ systems are being actively studied as potential biomarkers for various changes in peripheral neuropathies [15]. The most promising approach is diffusion-tensor MRI, which measures the magnitude and direction of water molecule diffusion in human tissues [16]. However, studies aimed at diagnosing and identifying differential biomarkers between LSS and MMN have not been conducted.

The aim of this study is to determine the diagnostic value of quantitative methods for assessing MRI signal intensity and thickness measurements of brachial plexus neural elements in differentiating LSS and MMN.

Materials and methods

The study included 59 patients (18 women and 41 men) aged 25–67 years with chronic polyneuropathy (26 patients with MMN 8, 33 patients with LSS) and 15 healthy subjects (7 women, 8 men) aged 23–60 years.

Sex and age were recorded for all participants. Body mass index (BMI) was calculated using the Quetelet formula: BMI = m/h^2, where m is body weight (kg) and h is height (m).

MRI was performed on a Siemens MAGNETOM Prisma 3T scanner. The standard scanning protocol included high-resolution 3D coronal sequences: 3D_T1_spc (TR = 1000 msec; TE = 30 msec; reconstructed voxel size 0.8 × 0.8 × 0.8 mm, FOV = 300 mm, 144 slices, scan duration 6 min 56 sec) and 3D_STIR (Short TI Inversion Recovery, TR = 3000 msec; TE = 281 msec; TI = 230 msec, reconstructed voxel size 0.4 × 0.4 × 0.9 mm, FOV = 350 mm, 144 slices, scan duration 7 min 27 sec).

To standardize MRI signal assessment, the calculation of an intensity coefficient (IC) [17] using 3D_STIR sequences was proposed, with the formula: IC = $(I_N - I_M)/I_M$, where I_N represents the directly measured MRI signal intensity from the most affected element of the brachial plexus, or from the left ventral ramus of the C7 spinal nerve in cases of symmetrical changes or in healthy subjects, while I_M denotes the MRI signal intensity from the left supraspinatus muscle. Muscle structure was preliminarily assessed for edema signs on 3D_STIR sequences and for atrophy/fatty replacement signs on 3D_T1_spc sequences. The C7 level was chosen for nerve element intensity measurements due to several factors: C5 and C6 ventral rami often course at more acute angles (~55°) to B0 magnetic field lines, potentially causing false signal hyperintensity from magic angle effects [18], while C8 and Th1 ventral rami lie close to major vessels and lung apices, making them more susceptible to pulsation and respiratory artifacts.

Additionally, 3D_STIR images were used to assess the thickness (transverse dimension measured in coronal plane) of spinal nerve ventral rami: maximal thickness at areas of focal enlargement or at 5–6 mm distance from the C7 spinal nerve ganglion in healthy volunteers and in cases with symmetrical diffuse nerve trunk thickening patterns.

Statistical analysis was performed using SPSS Statistics 23.0 (IBM, Armonk, NY, USA). Two-sided criteria were used in all cases. The null hypothesis was rejected at $p < 0.05$.

Results

According to the analysis results, all evaluated quantitative variables (IC, thickness, BMI, and age) were normally distributed. The calculated IC ranged from 0.88 to 4.05. When comparing the patient group (without diagnostic stratification) with healthy subjects, no significant differences were found in sex, age, or BMI; however, both thickness and IC were significantly greater in patients compared to the control group (Table 1).

ROC analysis identified threshold values for the differentiating parameters: 5.9 mm for thickness (probability of having a disease increases at ⩾ 5.9 mm) with 75.0% sensitivity and 86.7% specificity at this cutoff (Fig. 1), and 1.422 for the IC (probability of having a disease increases at IC ⩾ 1.422) with 86.4% sensitivity and 73.3% specificity at this cutoff (Fig. 2).

Spearman's rank correlation analysis in the combined MMN and LSS patient groups revealed a significant moderate inverse correlation between age and nerve element thickness ($p = 0.005$). A significant association was also observed between sex and BMI ($p = 0.005$), with males exhibiting higher BMI values. No significant correlations were found between IC and other characteristics, nor between thickness and any parameters except age.

When stratifying patients by diagnosis (MMN vs LSS), intergroup differences in sex, age, and BMI showed no statistical significance (unpaired T-test for age and BMI; Pearson's χ^2 test for sex; $p > 0.05$).

Analysis of thickness and IC values across MMN, LSS, and healthy controls revealed significant intergroup differences: both parameters were lowest in healthy volunteers, intermediate in MMN patients, and highest in LSS patients (Table 2). The Kruskal–Wallis test with post-hoc pairwise comparisons (Bonferroni-corrected for multiple comparisons) was used for comparing >2 independent groups with non-normal distributions. Post-hoc analysis demonstrated significant thickness differences between MMN vs controls and LSS vs controls, though MMN vs LSS differences became non-significant after Bonferroni correction: $p = 0.022$, $p_{adj} = 0.066$ (Table 3). For IC, significant differences were observed across all group pairs: MMN vs LSS, MMN vs controls, and LSS vs controls.

For parameters showing significant differences between MMN and LSS groups, threshold values were determined using ROC analysis. The area under the curve (AUC) for the thickness parameter was 0.678. The optimal threshold value was 7.2 mm, with sensitivity and specificity of 66.7% and 65.4%, respectively, at this cutoff (Fig. 3).

For the IC, the AUC was 0.707. The optimal IC threshold was 1.756, demonstrating 78.8% sensitivity and 57.7% specificity (Fig. 4).

Thus, thickness values for anterior ramus of the spinal nerve in MMN range between 5.9 and 7.2 mm, whereas IC values range from 1.422 to 1.756.

We present clinical cases where visual qualitative assessment of MR signal intensity fails to confirm pathological changes or differentiate LSS from MMN. However, calculated IC values enable diagnostic suggestions verified by clinical and instrumental methods per established criteria [2, 4].

Case 1. Healthy volunteer A., 35 years old. Fig. 5 shows a coronal STIR MRI of the brachial plexus (BP): the region of interest at the level of the anterior ramus of left C7 spinal nerve (Fig. 5, *A*, signal intensity $I_N = 87$) and the left supraspinatus muscle belly (Fig. 5, *B*, signal intensity $I_M = 39$). Substituting the values into the IC calculation formula yields IC = (87 – 39)/39 = 1.23, which falls within the normal range and indicates no pathological changes in BP (as evidenced by the absence of increased signal intensity on STIR sequences).

Case 2. Patient B., 48 years old, with multifocal motor neuropathy. Fig. 6 shows spinal BP MRI using STIR sequence in the coronal plane. Substituting the region of interest values

Table 1. Characteristics of patients diagnosed with LSS and MMN (without stratification by diagnosis) and healthy volunteers

Parameter		MMN and LSS (n = 59)	Control (n = 15)	p
Sex	male, n (%)	41 (69.5)	8 (53.3)	0.359
	female, n (%)	18 (30.5)	7 (46.7)	
Age, years	Median [Q_1; Q_3]	48.0 [38.0; 55.0]	30.0 [25.0; 52.0]	0.116
BMI, kg/m^2	Median [Q_1; Q_3]	26.5 [23.4; 27.9]	25.4 [21.9; 28.0]	0.228
Thickness, mm	Median [Q_1; Q_3]	7.2 [5.8; 8.0]	5.4 [4.7; 5.6]	< 0.001
IC	Median [Q_1; Q_3]	2.0155 [1.5431; 2.5307]	1.1915 [0.9457; 1.6333]	< 0.001

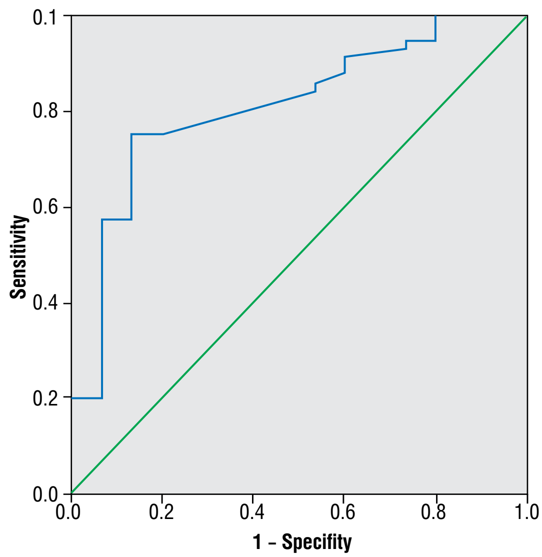

Fig. 1. ROC curve for the thickness parameter: normal/abnormal.
Significance level for curve deviation from the diagonal reference line: $p < 0.001$; area under the curve (AUC) = 0.81.

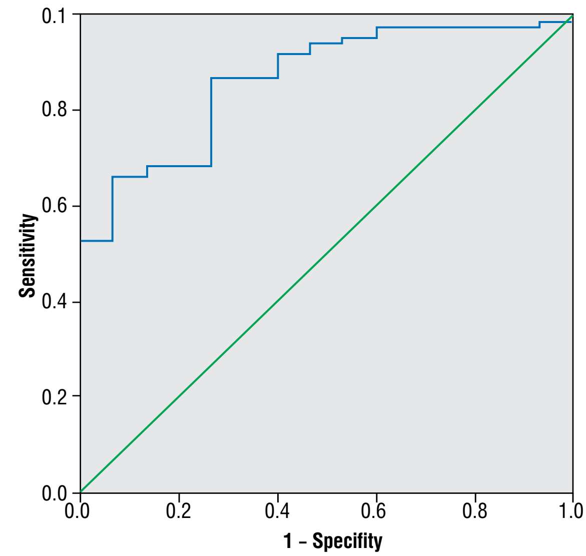

Fig. 2. ROC curve for IC: normal/abnormal.
Significance level for curve deviation from the diagonal reference line: $p < 0.001$; area under the curve (AUC) = 0.86.

Table 2. Characteristics of patients diagnosed with LSS and MMN (with stratification by diagnosis) and healthy volunteers

Parameter		Control ($n = 15$)	MMN ($n = 26$)	LSS ($n = 33$)	p
Sex	male, n (%)	8 (53.3)	15 (57.7)	26 (78.8)	0.119
	female, n (%)	7 (46.7)	11 (42.3)	7 (21.2)	
Age, years	Median [Q_1; Q_3]	30.0 [25.0; 52.0]	50.0 [44.0; 56.0]	46.0 [35.0 53.0	0.116
BMI, kg/m^2	Median [Q_1; Q_3]	25.4 [21.9; 28.0]	26.6 [22.3; 29.1]	26.4 [24.2; 27.8]	0.467
Thickness, mm	Median [Q_1; Q_3]	5.4 [4.7; 5.6]	6.2 [5.4; 7.5]	7.7 [6.2; 9.1]	< 0.001*
IC	Median [Q_1; Q_3]	1.1915 [0.9457; 1.6333]	1.6452 [1.3393; 2.1425]	2.2363 [1.7823; 2.7903]	< 0.001*

Table 3. Adjusted significance level (p_{adj}) for post hoc pairwise comparisons.

Parameter	MMN vs LSS	MMN vs control	LSS vs control
Thickness, mm	0.066	0.027	< 0.001
IC	0.021	0.015	< 0.001

(I_N = 92 and I_M = 36) into the IC calculation formula yields IC = (92 – 36)/36 = 1.55, which falls within the range of values indicative of STIR signal intensity changes (increased) in multifocal motor neuropathy.

Case 3. Patient V, 52 years old, with a multifocal chronic inflammatory demyelinating polyneuropathy – LSS. Fig. 7 shows spinal BP MRI using STIR sequence in the coronal plane. Substituting the region of interest values (I_N = 128 and I_M = 37) into the IC calculation formula yields IC = (128 – 37)/37 = 2.45, which falls within the range of values corresponding to signal intensity changes (increased) on STIR sequences in multifocal chronic inflammatory demyelinating polyneuropathy – LSS.

Discussion

Despite existing limitations of MRI for peripheral nerve evaluation, including high cost, specific technical requirements for equipment and scanning protocols, and the lack of clear diagnostic criteria, MRI has been increasingly used in clinical practice for assessing brachial plexus in recent years [19,20].

According to the aforementioned diagnostic criteria for MMN and LSS, nerve thickening is defined as an increase in the transverse diameter of the neural element in the coronal plane exceeding 5 mm. However, research data in this area are somewhat inconsistent. For instance, in a study assessing expert consensus involving 19 patients with a confirmed

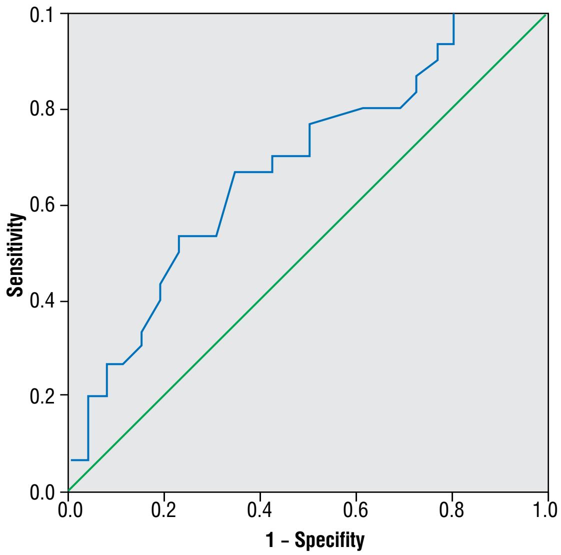

Fig. 3. ROC-curve for the thickness parameter: MMN/LSS.
Significance level for curve deviation from the diagonal reference line: $p < 0.022$; area under the curve (AUC) = 0.678.

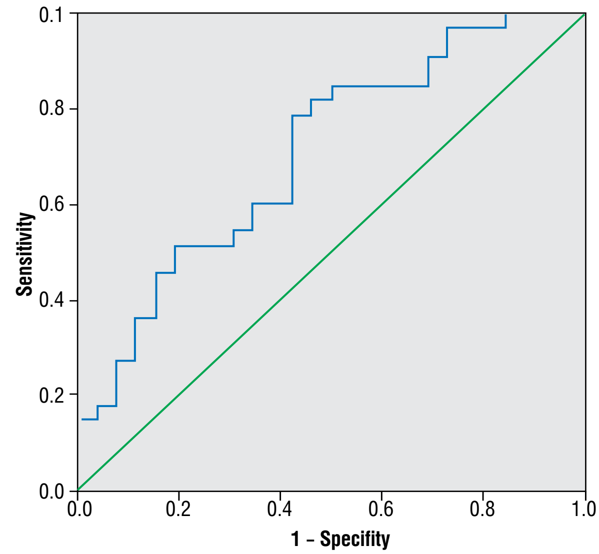

Fig. 4. ROC-curve for IC: MMN/LSS.
Significance level for curve deviation from the diagonal reference line: $p = 0.007$; area under the curve (AUC) = 0.707.

diagnosis of CIDP, 17 patients with MMN, and 14 healthy volunteers, an increase in peripheral nerve thickness greater than 5 mm was observed in 44–82% of patients, while the absence of this sign did not exclude or confirm the diagnoses of MMN and LSS [21]. In the patient sample included in the present study, significant differences in the thickness of the anterior spinal nerves forming the BP were found between the healthy volunteer group and the combined patient group, with a threshold value of 5.9 mm demonstrating sufficient sensitivity and specificity for predicting pathology. When comparing the two conditions based on this parameter, differences were also observed between MMN and LSS; however, post-hoc analysis with correction for multiple comparisons revealed no significant differences, which was further supported by low sensitivity and specificity values obtained in ROC analysis.

The second parameter evaluated when interpreting standard BP studies is the change in MR signal intensity in fluid-sensitive sequences with fat suppression, particularly when assessing the STIR sequence, as its use is the most adequate for BP visualization [22]. However, several studies have demonstrated that these parameters are nonspecific and often fail to distinguish between normal and pathologically altered peripheral nerves, for instance, in cases of symmetric bilateral involvement. This limitation primarily stems from the subjective nature of this parameter assessment, making it insufficient as a reliable marker of nerve fiber changes without further transformation. Calculating the IC using the formula proposed in this study could serve as a method for objectifying signal intensity changes in MRI studies of peripheral nerves. This work represents the first known study

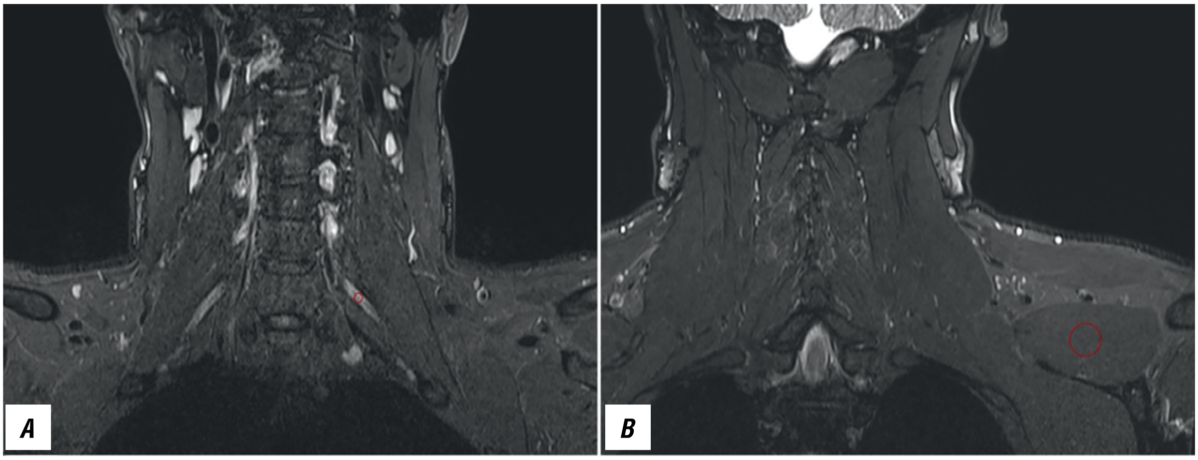

Fig. 5. Healthy volunteer A., 35 years old. Coronal projection, STIR sequence.
A — red outline indicates the region of interest at the level of the anterior ramus of the left C7 spinal nerve; *B* — red outline indicates the region of interest at the level of the left supraspinatus muscle belly.

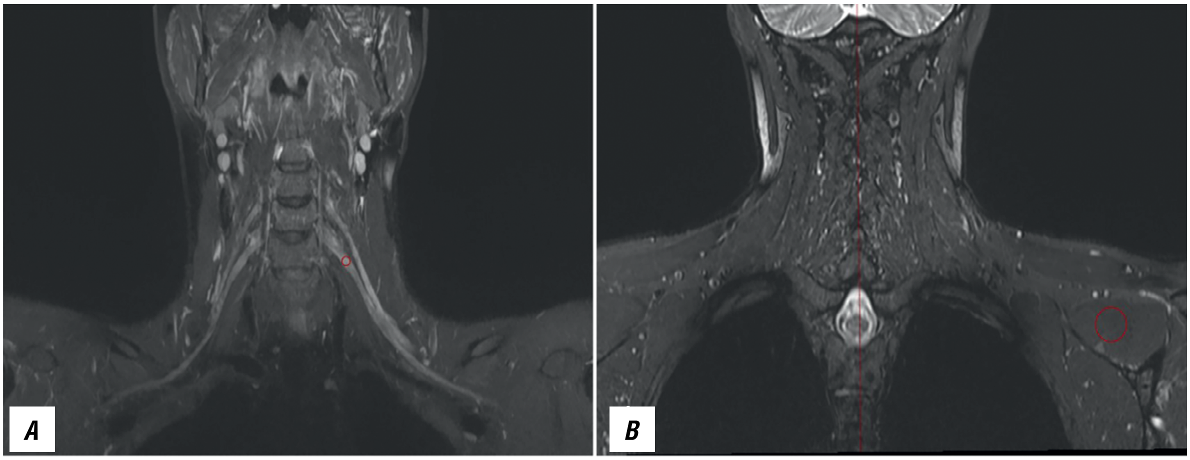

Fig. 6. Patient B., 48 years old, with a confirmed diagnosis of MMN established according to the EFNS/PNS 2010 criteria. Coronal projection, STIR sequence.
A – red outline indicates the region of interest at the level of the anterior ramus of the left C7 spinal nerve; *B* – red outline indicates the region of interest at the level of the left supraspinatus muscle belly.

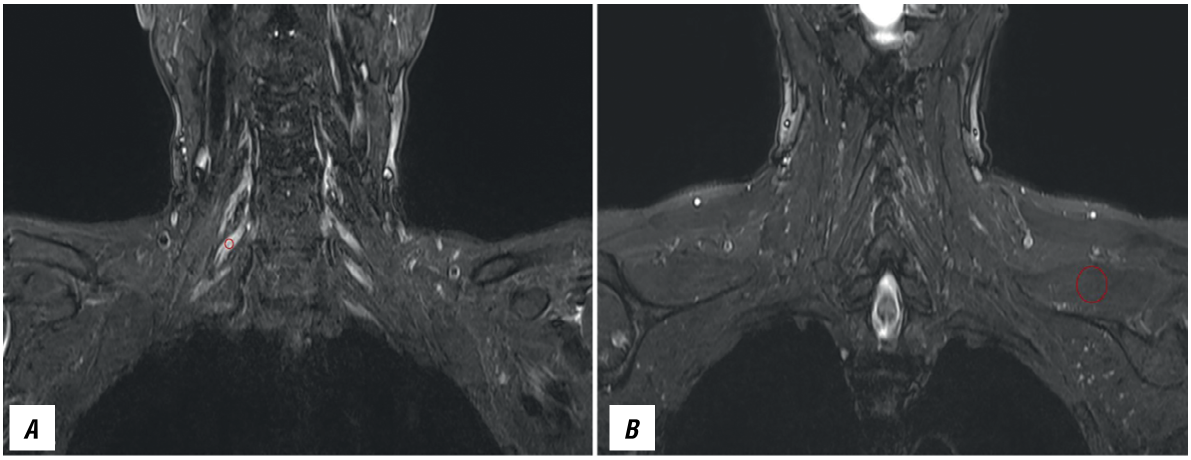

Fig. 7. Patient V, 52 years old, with a confirmed diagnosis of LSS established according to the EFNS/PNS 2021 criteria. Coronal projection, STIR sequence.
A – red outline indicates the region of interest at the level of the anterior ramus of the left C7 spinal nerve; *B* – red outline indicates the region of interest at the level of the left supraspinatus muscle belly.

in Russia and internationally to evaluate IC changes in MR signal intensity using STIR sequences for BP in a comparable patient cohort.

Given the relatively high sensitivity and specificity values (1.42) of the threshold IC between normal and pathological processes obtained in this study, the proposed method can be used to diagnose pathology. The specificity of the obtained IC threshold for differential diagnosis of MMN and LSS is insufficient for reliable discrimination between these pathologies, highlighting the need to search for other quantitative MRI markers, particularly using diffusion tensor MRI data, one of the most valuable techniques for assessing microstructural tissue changes.

The main limitation of this study, besides the relatively small sample size, is the use of the proposed method on a single MRI scanner with identical sequence parameters, which does not exclude possible threshold variations when using different scanners with alternative scanning parameters. Therefore, for widespread clinical implementation, the obtained data require validation in larger patient cohorts using different MRI scanners.

Conclusion

Based on the obtained data, it can be concluded that the proposed quantitative method for assessing MRI signal intensity in the anterior rami of spinal nerves forming the brachial plexus in MMN and LSS may provide additional information about pathological changes and facilitate faster accurate diagnosis. This will enable earlier initiation of pathogenetic therapy, reduce disability rates, and shorten the duration of patients' incapacity.

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