



Neuroimaging Markers for Differential Diagnosis Between Multifocal Motor Neuropathy and Multifocal Acquired Demyelinating Sensory and Motor Neuropathy

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Abstract

Introduction. Similar asymmetric patterns of motor disorders and neurophysiological changes complicate the differential diagnosis between multifocal motor neuropathy (MMN) and multifocal acquired demyelinating sensory and motor neuropathy (MADSAM) as two chronic dysimmune neuropathies with significantly different treatment approaches. The lack of specific paraclinical markers often result in misdiagnosis and selection of ineffective specific therapy. Identification of specific neuroimaging biomarkers to differentiate these conditions may improve diagnostic approaches.

Objective: To identify neuroimaging markers for the differential diagnosis between MMN and MADSAM.

Materials and methods. The study included 65 participants, particularly 30 individuals with MMN and 35 individuals with MADSAM followed up in the Center of Peripheral Nervous System Diseases, Research Center of Neurology, Moscow, Russia. We retrospectively analyzed their clinical and epidemiological characteristics as well as ultrasonography and magnetic resonance imaging (MRI) findings.

Results. Ultrasonography was performed on the peripheral nerves of the upper extremities, the spinal nerves, and the brachial plexus. The results showed that participants with MADSAM had significantly greater cross-sectional areas (CSAs) and a higher incidence of intraneural ultrasonographic abnormalities compared to participants with MMN. CSA thresholds of the median nerves were identified using ROC analysis to differentiate between MMN and MADSAM. MRI scans of the brachial plexus revealed no abnormalities in 41.4% of the individuals with MMN and 27.3% of the individuals with MADSAM. Meanwhile, STIR hyperintense signal from the brachial plexus was most typical (> 70%) for the MADSAM group.

Conclusions. This was the first detailed comparative analysis of neuroimaging findings in a large sample of patients with either MMN or MADSAM in Russia. Ultrasonographic markers for differential diagnosis have been determined. The advantages and limitations of ultrasonography and MRI of the brachial plexus and the spinal and peripheral nerves in diagnosing multifocal chronic dysimmune neuropathies have been demonstrated.

Keywords: multifocal motor neuropathy, multifocal acquired demyelinating sensory and motor neuropathy, ultrasonography of peripheral nerves, magnetic resonance imaging of the brachial plexus, dysimmune neuropathies

Ethics approval. The study was conducted with the informed consent of the patients. The research protocol was approved by the Ethics Committee of the Research Center of Neurology (protocol No. 10-4/21, November 17, 2021).

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Нейровизуализационные дифференциально-диагностические маркеры при мультифокальной моторной нейропатии и мультифокальном варианте хронической воспалительной демиелинизирующей полинейропатии

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Аннотация

Введение. Одинаковый асимметричный паттерн двигательных нарушений и однонаправленные нейрофизиологические изменения, регистрируемые при мультифокальной моторной нейропатии (ММН) и мультифокальном варианте хронической воспалительной демиелинизирующей полинейропатии (мХВДП), усложняют проведение дифференциального диагноза между этими двумя хроническими дизиммунными нейропатиями, терапевтическая тактика которых существенно различается. Отсутствие отличительных специфических параклинических маркеров зачастую приводит к ошибочному суждению о диагнозе и выбору неэффективной патогенетической терапии. Актуален прицельный поиск внутригрупповых нейровизуализационных различий.

Цель исследования: определить нейровизуализационные дифференциально-диагностические маркеры при ММН и мХВДП.

Материалы и методы. В исследование были включены 65 пациентов: 30 – с диагнозом ММН и 35 – с диагнозом мХВДП, наблюдающиеся в Центре заболеваний периферической нервной системы ФГБНУ «Научный центр неврологии». Проведены ретроспективный анализ клинико-эпидемиологических характеристик пациентов, сонографическое и магнитно-резонансное (МРТ) обследование.

Результаты. У пациентов с мХВДП по сравнению с ММН при УЗИ длинных периферических нервов рук, спинномозговых нервов и стволов плечевых сплетений отмечены значимо большие величины площади поперечного сечения и частота регистрации интраневральных сонографических изменений. С помощью ROC-анализа определены пороговые величины площади поперечного сечения срединного нерва, значимые для дифференциальной диагностики ММН и мХВДП. В 41,4% случаев у пациентов с ММН МРТ-картина исследования плечевых сплетений была сопоставима с нормой, при мХВДП патологические изменения не выявлены в 27,3% случаев. При этом наличие STIR-гиперинтенсивного сигнала от плечевых сплетений наиболее характерно для пациентов с мХВДП и встречалось более чем 70% случаев.

Заключение. В ходе настоящего исследования впервые в России на большой выборке пациентов проведён детальный сравнительный анализ данных нейровизуализационных методов исследования у пациентов с ММН и мХВДП; определены сонографические дифференциально-диагностические маркеры, показаны преимущества и ограничения ультразвукового и МРТ-исследований плечевых сплетений, спинномозговых и периферических нервов в диагностике мультифокальных хронических дизиммунных нейропатий.

Ключевые слова: мультифокальная моторная нейропатия; мультифокальный вариант хронической воспалительной демиелинизирующей полинейропатии; ультразвуковое исследование периферических нервов; магнитно-резонансная томография плечевых сплетений; дизиммунные нейропатии

Этическое утверждение. Исследование проводилось при добровольном информированном согласии пациентов. Протокол исследования одобрен Этическим комитетом Научного центра неврологии (протокол № 10-4/21 от 17.11.2021).

Источник финансирования. Авторы заявляют об отсутствии внешних источников финансирования при проведении исследования.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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Introduction

Similar asymmetric patterns of motor disorders and neurophysiological changes complicate the differential diagnosis between multifocal motor neuropathy (MMN) and multifocal acquired demyelinating sensory and motor neuropathy (MADSAM) as two chronic dysimmune neuropathies (CDNs) with significantly different treatment approaches [1, 2]. Despite continuous improvement of the chronic inflammatory demyelinating polyneuropathy (CIDP) and MMN diagnostic criteria, the lack of specific paraclinical markers result in misdiagnosis and selection of ineffective specific therapy [1–3].

The significance of neuroimaging, namely ultrasonography (USG) and magnetic resonance imaging (MRI) of peripheral nerves and brachial plexus (BP), for CIDP diagnosis has been demonstrated [4–6]. However, possible use of the methods for differential diagnosis between MMN and MADSAM and their interchangeability are still being discussed.

Objective: To identify neuroimaging markers for the differential diagnosis between MMN and MADSAM.

Materials and methods

The study included individuals with MMN ($n = 30$) and with MADSAM ($n = 35$), followed up in the Center of Peripheral Nervous System Diseases, Research Center of Neurology, Moscow, Russia.

Inclusion criteria:

- age > 18 years;
- CIDP diagnosed according to the European Academy of Neurology/Peripheral Nerve Society (EAN/PNS) 2021 diagnostic criteria;
- MMN diagnosed according to EFNS/PNS 2010 diagnostic criteria;
- signed informed consent to participate in the study.

Exclusion criteria:

- age < 18 years;
- the diagnosis did not comply with MMN or CIDP diagnostic criteria;
- contraindications for USG and MRI;
- decompensated severe medical conditions;
- patient refused to provide an informed consent.

We retrospectively analyzed clinical and epidemiological characteristics of the participants (sex, age at onset and enrollment, disease duration, and onset-to-treatment duration).

USG was performed in B mode with 4–18 MHz frequency on PhilipsElite scanner with a linear probe. We assessed long peripheral nerves of the upper extremities (median, ulnar, and radial nerves) at 23 points bilaterally and BPs in 7 areas bilaterally. At each point, we measured the nerve

cross-sectional area (CSA) either elliptically or, in irregular cross section, manually along the internal hyperechoic nerve border. We considered CSA parameters published by A. Kerasnoudis et al. [7] and A. Grimm et al. [8]. At each point, we also evaluated intraneural abnormalities classified according to L. Padua et al. [9].

MRI scan was performed under the standard protocol with 3T induction by Magnetom Siemens Prisma. We used high-resolution STIR 3D sequence to measure slice thickness and signal intensity (TR = 3000 msec, TE = 281 msec, TI = 230 msec, reconstructed voxel size $0.4 \times 0.4 \times 0.9$ mm, FOV = 350 mm, number of slices 144, scan time 7 min 27 sec). In each participant, we measured thickness of C4–C7 (N5–N7) anterior branches equidistantly from the ganglia on both sides and at maximum and used the highest result for statistical analysis. We also assessed BP MR signal intensity qualitatively at the entire visible level. MRI assessment was based on EAN/PNS guidelines (2021), with threshold coronary thickness of 5 mm. This paper presents an assessment of this parameter (thickened or not) based on the generally accepted reference. In addition, we performed a qualitative assessment of MRI signal intensity (hyperintense/not hyperintense) from BPs, based on the clinical radiologist's experience, without using quantitative methods.

Statistical analysis was performed using SPSS Statistics 23.0 (IBM). We used paired tests for all comparisons. Distribution of quantitative data was estimated using frequency histogram analysis. Quantitative variables were described using medians (Me), quartiles [Q1; Q3], means, and standard deviations (in Gaussian distribution), while categorical variables were described using frequencies and percentages.

We used independent samples t-test to compare two independent data groups by quantitative variables in Gaussian distribution and Mann–Whitney U test to compare them in non-Gaussian distribution. We used Pearson's χ^2 test to compare two independent data groups by categorical variables and Fisher's exact test under constraints. We performed ROC analysis to assess the possible use of median nerve CSA as a diagnostic marker. We determined optimal thresholds with the Youden's index calculated as sensitivity and specificity sum minus 1.

Results

The study included 65 participants, particularly 30 individuals, of them 12 (40%) women and 18 (60%) men aged 34–68 (Me = 49.0 [41.0, 56.0]), with MMN (Group 1) and 35 individuals, of them 9 (25.7%) women and 26 (74.3%) men aged 25–78 (Me = 52.0 [40.0, 61.0]), with MADSAM (Group 2) (Table 1). No gender or age differences was documented. The disease duration was significantly higher in the MMN population than in the MADSAM population ($p = 0.001$). Both groups included pre-treated and treat-

Table 1. Epidemiological, clinical and medical history data of patients included in the study

| Characteristic | | Patients with MMN | Patients with MADSAM | <i>p</i> |
|-------------------------|---------------------------------------|-------------------|----------------------|----------|
| Number of participants | | 30 | 35 | – |
| Sex | male. <i>n</i> (%) | 18 (60.0%) | 26 (74.3%) | 0.290 |
| | female. <i>n</i> (%) | 12 (40.0%) | 9 (25.7%) | |
| Enrollment age. years | <i>M</i> ± <i>SD</i> | 49.7 ± 10.1 | 51.5 ± 12.4 | 0.519 |
| | Me [Q ₁ ; Q ₃] | 49.0 [41.0; 56.0] | 52.0 [40.0; 61.0] | |
| Disease duration. years | Me [Q ₁ ; Q ₃] | 10.0 [7.0; 13.0] | 6.0 [4.0; 8.0] | 0.001 |

ment-naive patients. The MMN group included 24 (80%) pre-treated individuals and 6 (20%) treatment-naive patients, while the MADSAM group included 20 (57%) pre-treated individuals and 15 (43%) treatment-naive patients.

Comparative evaluation of USG findings on the long peripheral nerves of the upper extremities in patients with MMN and MADSAM

Comparative evaluation of USG findings at 23 points on each side of the long peripheral nerves of the upper extremities revealed intergroup differences in 34 (73.9%) of 46 possible points with a threshold significance < 0.05. Therefore, the significance threshold was elevated to 0.005, which enabled us to decrease the number of the points with statistically significant intergroup differences to 12 (26%) (Table 2).

Compared to the patients with MMN, the mean unilateral CSA of the median nerve in the patients with MADSAM was significantly higher at the antecubital fossa, the lower and upper brachium thirds, and bilaterally in the axillary area ($p < 0.005$). Ulnar nerve imaging demonstrated similar changes at the upper antebrachium third, the brachium, and in the axillary area unilaterally ($p < 0.005$), while radial nerve imaging showed them at the middle and upper brachium thirds unilaterally ($p < 0.005$; Table 2).

Considering that intergroup difference was most often found in the median nerve at various levels, we performed ROC analysis to evaluate the possible use of the median nerve CSA for differential diagnosis between MMN and MADSAM. We took the models with area under the ROC curve (AUC) > 0.700 into account. The CSA thresholds of the median nerve, assessed unilaterally and significant for the differential diagnosis between MMN and MADSAM, in favor of the latter, were the following:

- $\geq 8.10 \text{ mm}^2$ at the lower antebrachium third (AUC = 0.741, sensitivity 74%, specificity 73%; Fig. 1, A);
- $\geq 7.25 \text{ mm}^2$ at the upper antebrachium third (AUC = 0.766, sensitivity 71%, specificity 70%; Fig. 1, B);
- $\geq 9.9 \text{ mm}^2$ at the antecubital fossa (AUC = 0.731, sensitivity 63%, specificity 73%; Fig. 1, C);
- $\geq 11.65 \text{ mm}^2$ at the lower brachium third (AUC = 0.712,

sensitivity 71%, specificity 70%; Fig. 1, D);

- $\geq 12.55 \text{ mm}^2$ at the upper brachium third (AUC = 0.707, sensitivity 71%, specificity 77%; Fig. 1, E);
- $\geq 12.6 \text{ mm}^2$ in the axillary area (AUC = 0.760, sensitivity 71%, specificity 70%; Fig. 1, F);

USG changes were asymmetric in both groups as expected with the pathophysiology and the clinical characteristics of the studied CDNs.

Comparative evaluation of the USG patterns of intraneural changes in the assessed arm points based on L. Padua's classification demonstrated that the above changes were significantly more often revealed in the patients with MADSAM though only in isolated MMN cases. So, in the MADSAM patients, class 1/2 intraneural changes (enlarged CSAs, enlarged single fascicles) were significantly more often revealed on the median nerves at the antecubital fossa ($p = 0.003$), at the lower brachium third ($p = 0.012$), and in the axillary area ($p = 0.019$); on the ulnar nerves in the lower (right: $p = 0.013$; left: $p = 0.007$) and middle ($p = 0.008$) brachium third and in the axillary area ($p = 0.003$); and on the radial nerves in the middle ($p = 0.013$) and upper ($p = 0.017$) brachium thirds.

Thus, USG of the long peripheral nerves of the upper extremities showed a significantly larger CSA and a higher incidence of documented intraneural USG abnormalities in the patients with MADSAM than in the MMN patients. We determined significant thresholds of the median nerve CSA at various levels for differential diagnosis between MMN and MADSAM.

Comparative evaluation of USG findings of the spinal nerves and the BPs in patients with MMN and MADSAM

USG detected enlarged diameters of the spinal nerves and BP trunks in 26 (87%) patients with MMN and 32 (94%) patients with MADSAM, including unilateral ones in 6 (23%) of 26 patients with MMN and 3 (9%) of 32 patients with MADSAM (Fig. 2).

As compared to the patients with MMN, the mean unilateral diameters of the spinal nerves and CSAs of the BP

Table 2. Comparative evaluation of USG findings on the long peripheral nerves of the upper extremities in patients with MMN and MADSAM, mm² (Me [Q₁; Q₃])

| Nerve and assessment level | | Reference CSAs | Side | Patients with MMN | Patients with MADSAM | <i>p</i> | |
|----------------------------|--------------|-------------------|--------------|----------------------|----------------------|--------------------|---------|
| Peripheral nerves | Median nerve | radiocarpal joint | Right | 8.35 [7.50; 9.70] | 8.90 [7.30; 11.30] | 0.598 | |
| | | | Left | 8.45 [7.50; 9.90] | 9.30 [7.90; 10.30] | 0.298 | |
| | | antebrachium | lower third | Right | 7.45 [6.30; 8.70] | 8.60 [7.00; 10.10] | 0.120 |
| | | antebrachium | lower third | Left | 6.60 [5.80; 8.50] | 9.30 [7.80; 11.10] | 0.001 |
| | | antebrachium | middle third | Right | 6.75 [6.00; 8.20] | 7.70 [6.70; 10.70] | 0.011 |
| | | antebrachium | middle third | Left | 7.45 [6.30; 8.80] | 8.90 [7.10; 12.40] | 0.036 |
| | | antebrachium | upper third | Right | 6.35 [5.20; 7.40] | 8.70 [6.70; 13.20] | < 0.001 |
| | | antebrachium | upper third | Left | 7.50 [6.10; 9.20] | 9.10 [6.70; 12.20] | 0.039 |
| | | antecubital fossa | Right | 8.30 [7.10; 10.70] | 11.00 [8.10; 13.30] | 0.006 | |
| | | | Left | 8.10 [6.90; 10.10] | 12.20 [7.90; 16.60] | 0.001 | |
| | brachium | lower third | Right | 11.00 [9.00; 15.20] | 13.70 [9.80; 20.00] | 0.040 | |
| | | | Left | 10.75 [8.60; 12.30] | 14.30 [11.30; 23.00] | 0.003 | |
| | | middle third | Right | 12.50 [9.10; 15.20] | 14.30 [10.10; 20.30] | 0.069 | |
| | | | Left | 10.70 [8.70; 12.80] | 13.80 [9.80; 20.10] | 0.006 | |
| | | upper third | Right | 11.10 [9.80; 12.40] | 14.70 [12.10; 20.40] | 0.004 | |
| | | | Left | 10.55 [9.70; 12.60] | 13.70 [8.70; 19.80] | 0.035 | |
| | | axillary fossa | Right | 12.05 [10.20; 15.30] | 17.10 [12.30; 26.70] | 0.004 | |
| | | | Left | 10.30 [9.10; 13.10] | 16.60 [11.10; 22.20] | < 0.001 | |
| | Ulnar nerve | radiocarpal joint | Right | 5.35 [4.30; 6.60] | 5.40 [4.60; 6.70] | 0.693 | |
| | | | Left | 5.30 [4.20; 6.30] | 6.10 [5.20; 6.90] | 0.053 | |
| antebrachium | | lower third | Right | 6.10 [4.70; 6.90] | 6.60 [4.90; 7.60] | 0.241 | |
| antebrachium | | lower third | Left | 5.40 [4.70; 6.60] | 6.60 [5.30; 8.90] | 0.015 | |
| antebrachium | | middle third | Right | 6.30 [5.30; 7.50] | 7.60 [5.50; 9.00] | 0.047 | |
| antebrachium | | middle third | Left | 6.00 [5.10; 6.70] | 6.90 [5.30; 10.70] | 0.033 | |
| antebrachium | | upper third | Right | 6.00 [5.20; 8.30] | 7.10 [5.80; 8.70] | 0.107 | |
| antebrachium | | upper third | Left | 5.90 [5.10; 7.00] | 7.50 [6.30; 10.00] | 0.001 | |

End of the Table 2.

| Nerve and assessment level | | Reference CSAs | Side | Patients with MMN | Patients with MADSAM | <i>p</i> |
|----------------------------|--------------|-----------------------|--------------------|---------------------|----------------------|----------|
| Peripheral nerves | Ulnar nerve | antecubital fossa | Right | 8.15 [6.70; 11.00] | 9.30 [7.10; 10.40] | 0.608 |
| | | | Left | 8.35 [6.20; 9.50] | 9.80 [6.80; 12.00] | 0.016 |
| | | brachium lower third | Right | 7.65 [6.50; 9.30] | 9.90 [6.80; 15.20] | 0.024 |
| | | | Left | 7.40 [5.80; 9.60] | 8.70 [7.10; 13.00] | 0.017 |
| | | brachium middle third | Right | 8.40 [6.70; 9.20] | 10.70 [7.30; 14.40] | 0.045 |
| | | | Left | 8.40 [6.30; 10.60] | 9.90 [6.80; 13.30] | 0.100 |
| | | brachium upper third | Right | 7.60 [6.30; 9.80] | 10.90 [7.70; 15.60] | 0.003 |
| | | | Left | 8.10 [7.00; 9.10] | 10.10 [7.80; 13.70] | 0.007 |
| | | axillary fossa | Right | 7.75 [6.40; 11.00] | 11.60 [7.90; 17.90] | 0.002 |
| | Left | | 8.55 [6.60; 9.80] | 10.80 [8.10; 17.20] | 0.008 | |
| | Radial nerve | antecubital fossa | Right | 6.70 [5.00; 7.80] | 8.60 [5.70; 10.10] | 0.010 |
| | | | Left | 8.10 [6.00; 11.40] | 9.70 [7.40; 12.00] | 0.041 |
| | | brachium lower third | Right | 6.80 [5.40; 8.40] | 8.30 [5.70; 9.20] | 0.111 |
| | | | Left | 7.30 [5.60; 8.40] | 8.10 [6.30; 11.20] | 0.067 |
| | | brachium middle third | Right | 6.30 [5.00; 8.10] | 7.70 [5.50; 10.50] | 0.039 |
| | | | Left | 6.00 [5.30; 7.40] | 8.60 [6.30; 11.00] | 0.002 |
| | | brachium upper third | Right | 7.85 [6.10; 9.90] | 9.40 [7.90; 14.20] | 0.004 |
| | | | Left | 7.70 [6.40; 8.90] | 9.60 [7.10; 13.30] | 0.044 |
| axillary fossa | | Right | 7.80 [6.70; 10.70] | 10.80 [7.00; 16.10] | 0.031 | |
| | Left | 7.75 [6.70; 10.30] | 9.60 [8.20; 15.30] | 0.006 | | |

trunks were statistically significantly larger in the MADSAM population (Table 3). USG revealed significant differences of the mean CSAs at the middle and lower BP trunks unilaterally and in the cross-scanned supraclavicular fossa ($p < 0.01$).

Thus, USG of the spinal nerves and the BP trunks revealed significantly larger diameters and CSAs respectively in the patients with MADSAM than in those with MMN.

Comparative evaluation of BP MRI findings in the patients with MMN and MADSAM

BP MRI was conducted in 29 patients with MMN and 33 patients with MADSAM. The most common reason for refusal was claustrophobia (i.e. a fear of confined spaces). BP MRI findings were apparently normal in 41.4% of the patients with MMN and no changes were found in 27.3% of the patients with MADSAM (Table 4). Enlarged BP trunks were detected with the same frequency in both groups ($p > 0.05$). Documented in 70% of the cases, STIR hyperintense BP signal was more typical for the patients with MADSAM.

Qualitative evaluation showed several changed BP trunk patterns in the assessed sample:

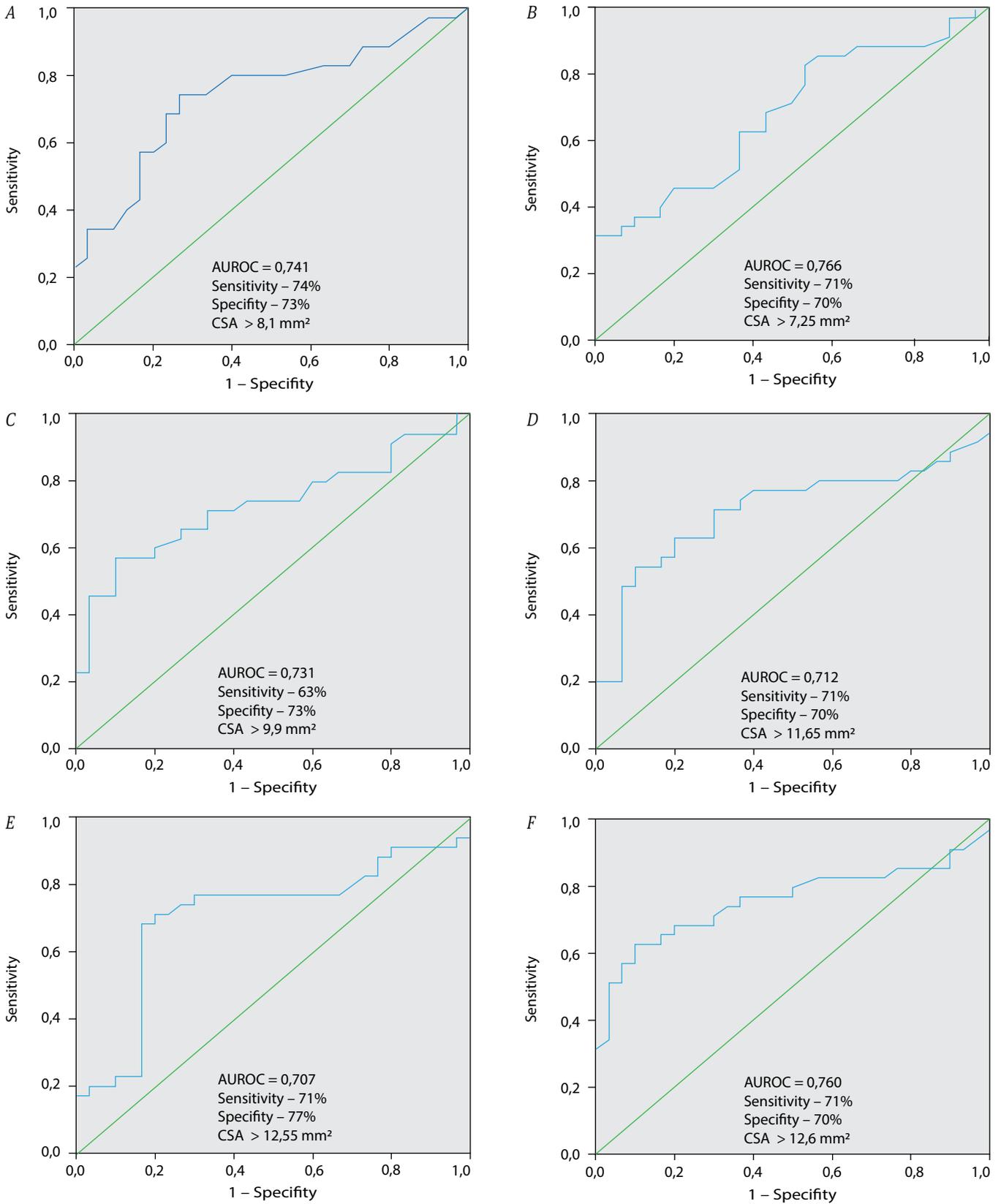


Fig. 1. ROC analysis of the significance of the median nerve CSA at various levels for the differential diagnosis between MMN and MADSAM.

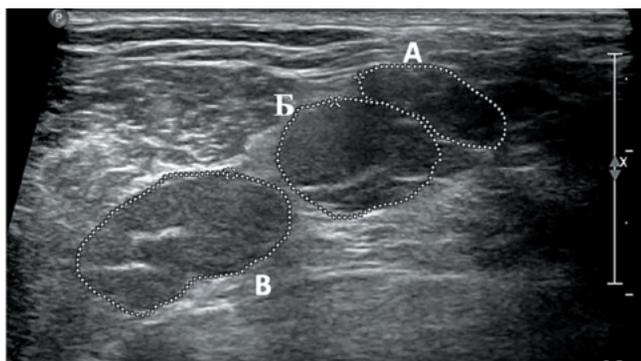


Fig. 2. USG of BP trunks in a patient with MADSAM (8-year follow-up history, pre-therapy assessment).

In the cross section, three primary trunks are seen in the scalene part, with enlarged upper ($\leq 33.6 \text{ mm}^2$; A), middle ($\leq 68.9 \text{ mm}^2$; B), and lower ($\leq 94.8 \text{ mm}^2$; C) primary trunks (reference $< 8 \text{ mm}^2$).

- significant symmetric bilateral diffuse BP thickening in 10 (34.5%) patients with MMN and 17 (51.5%) patients with MADSAM (Fig. 3);
- asymmetric diffuse BP thickening in 3 (10.3%) patients with MMN and 2 (6%) patients with MADSAM (Fig. 4);
- local BP thickening in 4 (13.8%) patients with MMN and 5 (15.2%) patients with MADSAM (Fig. 5);

- isolated hyperintense STIR MRI signal without enlarged BP trunks in 5 (17.2%) patients with MMN and 6 (18.2%) patients with MADSAM (Fig. 6)

Therefore, qualitative evaluation of the BP MRI findings demonstrated rather uniform changes that did not differentiate reliably between MADSAM and MMN.

Discussion

N. Taniguchi et al. were first to describe a CIDP USG pattern [10]. Routine thyroid USG found thickened peripheral nerves and BP proximal parts in a patient with a 3-year follow-up CIDP history [10]. Being widely available and non-invasive, peripheral nerve USG was then further investigated in a cohort of patients with polyneuropathies of various origin.

First studies were conducted in limited samples. In 2004, N. Matsuoka et al. assessed 13 patients with CIDP and documented enlarged cervical nerves in 69% cases [11]. In 2009, C. Zaidman et al. assessed 36 patients with CIDP, found over 2-fold diffusely enlarged median and ulnar nerves as compared to controls, and discovered direct correlation between the USG pattern and disease duration, with inverse correla-

Table 3. Comparative evaluation of USG findings of the spinal nerves and the BPs in patients with MMN and MADSAM, Me [Q₁; Q₃]

| Assessed level | | Side | Patients with MMN | Patients with MADSAM | <i>p</i> |
|--|------------------------------|-------|----------------------|-----------------------|----------|
| Spinal nerves. mm | C5 | Right | 8.15 [7.50; 9.40] | 9.30 [8.20; 14.80] | 0.022 |
| Spinal nerves. mm | C5 | Left | 8.65 [7.30; 11.20] | 10.20 [7.00; 16.70] | 0.171 |
| Spinal nerves. mm | C6 | Right | 11.40 [9.00; 17.70] | 14.70 [10.00; 20.50] | 0.134 |
| Spinal nerves. mm | C6 | Left | 12.60 [9.50; 14.50] | 14.20 [11.90; 26.60] | 0.023 |
| Spinal nerves. mm | C7 | Right | 10.75 [9.40; 16.40] | 13.50 [10.90; 19.80] | 0.040 |
| Spinal nerves. mm | C7 | Left | 13.20 [9.60; 15.70] | 15.60 [12.00; 27.20] | 0.039 |
| BP trunk CSA. mm ² | upper trunk (<i>n</i> < 8) | Right | 7.80 [6.20; 11.10] | 9.80 [7.10; 15.60] | 0.124 |
| BP trunk CSA. mm ² | upper trunk (<i>n</i> < 8) | Left | 7.15 [5.40; 11.60] | 11.00 [6.90; 21.30] | 0.069 |
| BP trunk CSA. mm ² | middle trunk (<i>n</i> < 8) | Right | 12.10 [9.30; 15.70] | 16.50 [11.20; 26.10] | 0.040 |
| BP trunk CSA. mm ² | middle trunk (<i>n</i> < 8) | Left | 10.25 [8.40; 15.90] | 16.70 [11.70; 29.10] | 0.009 |
| BP trunk CSA. mm ² | lower trunk (<i>n</i> < 8) | Right | 12.05 [9.10; 14.70] | 15.40 [10.10; 20.80] | 0.095 |
| BP trunk CSA. mm ² | lower trunk (<i>n</i> < 8) | Left | 13.25 [9.90; 15.30] | 17.50 [11.70; 26.50] | 0.004 |
| Supraclavicular fossa CSA. mm ² | | Right | 66.15 [58.80; 98.00] | 83.50 [66.20; 115.00] | 0.024 |
| Supraclavicular fossa CSA. mm ² | | Left | 70.35 [54.90; 90.60] | 101.0 [74.60; 125.00] | 0.002 |

Table 4. Comparative evaluation of BP and spinal nerve MRI changes in the patients with MMN and MADSAM, n (%)

| Characteristic | | Side | Patients with MMN | Patients with MADSAM | p |
|--|--------------|-------|-------------------|----------------------|-------|
| Enlarged BP trunks | upper trunk | Right | 9 (31.0%) | 16 (48.5%) | 0.200 |
| Enlarged BP trunks | upper trunk | Left | 8 (27.6%) | 17 (51.5%) | 0.072 |
| Enlarged BP trunks | middle trunk | Right | 13 (44.8%) | 20 (60.6%) | 0.308 |
| Enlarged BP trunks | middle trunk | Left | 13 (44.8%) | 20 (60.6%) | 0.308 |
| Enlarged BP trunks | lower trunk | Right | 13 (44.8%) | 18 (54.5%) | 0.611 |
| Enlarged BP trunks | lower trunk | Left | 12 (41.4%) | 17 (51.5%) | 0.456 |
| STIR hyperintense BP signal (total) | | Right | 16 (55.2%) | 24 (72.7%) | 0.188 |
| STIR hyperintense BP signal (total) | | Left | 14 (48.3%) | 27 (81.8%) | 0.007 |
| STIR hyperintense BP signal without enlarged BP trunks | | – | 5 (17.2%) | 6 (18.2%) | 1.000 |
| No changes | | – | 12 (41.4%) | 9 (27.3%) | 0.289 |

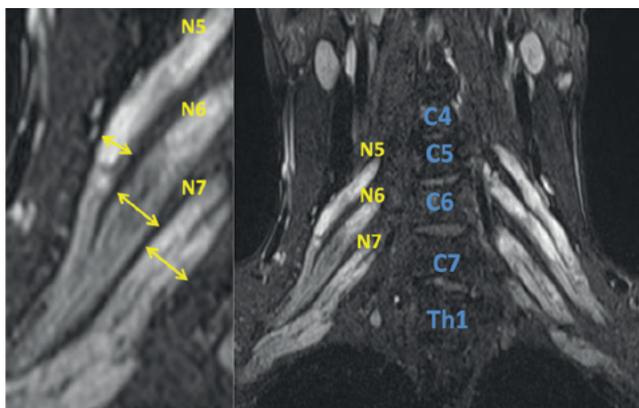


Fig. 3. MRI of BPs in a MMN patient (13-year follow-up history; assessed on maintenance therapy: intravenous immunoglobulin 1 g/kg every 4 weeks).
 The coronal STIR MRI showed significant (≤ 8 mm) bilateral uniform symmetric BP thickening, with hyperintense signal.

tion between assessed peripheral nerve CSA and motor fiber conduction velocity [12]. Several studies were limited as small case series with an idea to establish correlation between enlarged peripheral nerve CSA on one hand and CIDP and MMN symptom severity, electroneuromyographical conduction blocks, and response to specific therapy on the other hand [13–18]. There were no reliable evidence that USG and electroneuromyography abnormalities correlate with neurological deficit distribution and severity [19, 20]. A number of studies showed that the peripheral nerve CSA is smaller in relapsing disease than in progressive disease [16]. Besides, the patients with peripheral nerve CSAs that exceed reference values and with hypoechoic USG signal tended to be better responders to specific treatment than those without any enlarged CSA and with hyperintense USG signal [9, 21].

L. Padua et al. established enlarged CSAs and described three patterns of intraneural USG abnormalities in patients with CIDP: the thickened nerve with hypoechoic fascicles (Class 1); the thickened nerve with hypo- and hyperechoic fascicles (Class 2); the normal nerve CSA with the hyperechoic signal (Class 3) [9].

Further research was aimed at developing USG protocols for differential diagnosis between polyneuropathies of various origin with disease follow-up [17, 22–25]. Particularly, the S. Goedee et al., showed that the median nerve CSA enlarged at the antebrachium > 13 mm² and at the brachium > 10 mm² as well the enlarged CSA of any BP bundle > 8 mm² is 99% specific for CIDP diagnosis [4].

Additionally, D.S. Druzhinin et al. obtained noteworthy findings by peripheral nerve USG in patients with MMN ($n = 13$) and CIDP ($n = 7$) [26]. They showed that similarly enlarged BP and peripheral nerve CSAs were detected in both CIDPs, while asymmetric USG changes were more typical for patients with MMN and symmetric and diffuse changes were seen in those with CIDP.

In 2021, as a result of 20-year retrospective analysis of accumulated data, EAN and PNS recognized peripheral nerve USG as a significant supportive modality of CIDP diagnosis [1]. The MMN criteria have not included USG yet [2].

MR neuroimaging traces back to the early 1990s. First publications were focused on the MRI of the cauda equina [27–29]. In 1997, the Netherlandic clinicians were first to demonstrate BP MRI diagnostic performance in patients with DNs [30].

A study published in 1999 included 14 patients with CIDP

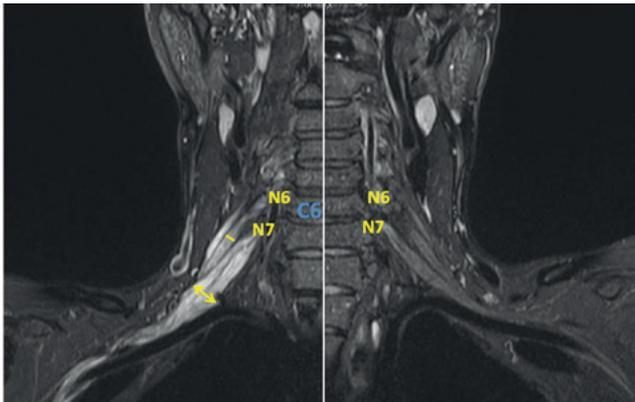


Fig. 4. MRI of BPs in a MADSAM patient (6-year follow-up history; assessed on maintenance therapy: intravenous immunoglobulin 1 g/kg every 12 weeks for 2 years).

The coronal STIR MRI showed right-sided significant (≤ 12 mm) diffuse N7 thickening, with hyperintense signal. Hyperintense MRI signals from other right-sided BP elements were registered at the entire visible level with unchanged thickness. No changes on the left side.

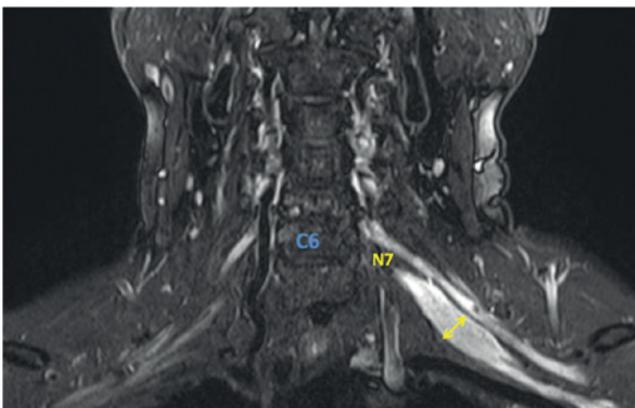


Fig. 5. BP MRI in a MMN patient (10-year follow-up history, pre-therapy assessment).

The coronal STIR MRI showed left-sided local N7 (≤ 11 mm) primary trunk thickening, with hyperintense signal. Thickness of other BP elements remained unchanged; however, hyperintense MRI signal was registered bilaterally.

[31]. Brachial and lumbar plexus MRI showed enlarged BPs in 8 (57%) patients and enlarged lumbar plexus in 6 (43%) patients. Further, enhanced MRIs revealed signal hyperintensity in 5 patients with hypertrophic plexus and 1 patient without any signs of hypertrophy for researchers to conclude contrast agent accumulation directly depended on disease activity [31].

Currently the use of an intravenous contrast agent in MR neuroimaging has lost its diagnostic value and might be utilized in few peripheral nerve abnormalities (primary masses, metastases) [32]. Sequences that suppress fat signals (e.g. STIR) have become methods of choice to assess changed peripheral nerves. With good signal/noise ratios (SNRs), optimal contrast parameters, and sufficient spatial resolution ob-

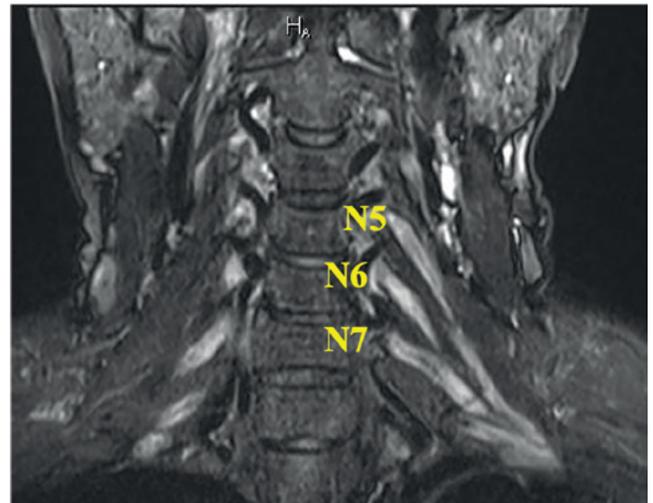


Fig. 6. MRI of BPs in a MADSAM patient (6-year follow-up history; assessed during 2-year glucocorticosteroid therapy).

Hyperintense STIR MRI signal bilaterally at the entire visible level without any thickened BP trunks.

tained on modern high-field (≥ 1.5 T) scanners, we can obtain high-quality selective images of tiny or tortuous structures, minimize respiration and vasculature/musculature artifacts, and come closer to BP qualitative description and DN differential diagnosis [33].

Thus, USG and MRI have been shown to provide accurate information for the diagnosis of CDN. However, intra-group neuroimaging differences are still a current target. Thus, the differential diagnosis between two multifocal CDNs, MMN and MADSAM, remains a challenging issue.

Unlike D.S. Druzhinin et al. [26], we complicated our task with comparison of MMN and multifocal (atypical) CIDP, having increased the number of patients and adding MRI findings.

We demonstrated that mean nerve CSAs in the MADSAM group were larger than those in the MMN group. Established USG patterns must be based on relevant CDN underlying mechanisms including demyelination and, as a consequence, more apparent edema of the peripheral nerves in the patients with MADSAM and affected though less edematous nodal and paranodal areas of the nerve trunks in the patients with MMN.

Like H.S. Goedee et al. [4], we found that it is the median nerve, particularly its proximal part (above the antecubital fossa), that it is the most diagnostically informative among three upper-extremity peripheral nerves including the median, ulnar, and radial nerves. We were first to conduct ROC analysis and calculate median nerve CSAs in the assessed levels that can be used for differential diagnosis between the investigated CDNs. Noteworthy, sensitivity and specificity of the obtained CSA thresholds varied, depending on the assessed points and

the side. Therefore, AUC [95% confidence interval] was < 0.7 in the median-nerve CSA ROC model for the antebrachium and brachium middle thirds. However, these points constitute the basis of the Ultrasound Pattern Sum Score [22] and the abbreviated ultrasound protocol [4] widely used for the diagnosis of dysimmune neuropathies. Considering these results, we recommend broadening the scope of assessment with antebrachium and brachium lower and upper thirds for MMN and MADSAM differential diagnosis.

USG typically revealed full-length thickened peripheral nerves in the patients with MADSAM and mostly asymmetrically changed segments in the patients with MMN, which was described above [26, 34, 35].

Spinal nerve and BP MRI did not demonstrate any significant differences between the patients with MMN and MADSAM. Furthermore, MRI BP qualitative characteristics (thickened or not; STIR-hyperintense signal or not) have a low diagnostic value in patients with CDN, especially with non-pronounced changes. In USG, qualitative analysis is cheaper and enables us to assess changes quantitatively (as diameters and CSAs) unlike that in MRI. Therefore, further research should focus

on the determination of quantitative MRI-parameters and evaluation of their significance for differential diagnosis between multifocal CDNs.

As demonstrated earlier, combined USG and MRI increased diagnostic value of both methods up to 83% in patients with MADSAM while, according to the authors, the methods are substitutable [4]. Our study showed that the patients with normal peripheral-nerve CSAs on USG can demonstrate only isolated hyperintense MRI signal from the BP trunks in the STIR mode (2/29 [6.9%] in the MMN population, 1/33 [3.0%] in the MADSAM population), which can indirectly indicate CDN.

Conclusion

This is the first study in Russia's that provided a detailed comparison of neuroimaging findings obtained in a large sample of patients with MMN and MADSAM; determined ultrasonographic markers for differential diagnosis; and demonstrated advantages and limitations of USG and MRI of BPs, spinal and peripheral nerves in the diagnosis of multifocal chronic dysimmune neuropathies.

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