Spine

SPINE Volume 45, Number 21, pp 1506–1512

DIAGNOSTICS

OPEN

The Noninvasive Diagnostic Value of MRN for CIDP: A Research from Qualitative to Quantitative

Yuan Feng, PhD,[a] Xiaoyun Su, PhD,[b] Chuansheng Zheng, PhD,[b] and Zuneng Lu, PhD[a]

Study Design. We examined the chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) patients and non-CIDP patients who have similar symptoms and difficult to differential diagnosis with CIDP by magnetic resonance neurography to find the difference among them.

Objective. To investigate the differential diagnostic value of magnetic resonance neurography (MRN) for CIDP and other peripheral neuropathies.

Summary of Background Data. Thirty-two consecutive patients with CIDP and 22 non-CIDP patients with symptoms similar to CIDP and difficult to be discriminate were recruited and imaged as a control group between May 2017 and May 2019.

Methods. In this prospective study, the brachial plexus and lumbosacral plexus of 32 CIDP patients and 22 non-CIDP patients were examined by MRN. The clinical features and the nerve roots cross-sectional area (CSA) of the brachial plexus and lumbosacral plexus were measured.

Results. The CSA of nerve roots of CIDP, Charcot-Marie-Tooth disease type-1 and polyneuropathy, organomegaly, endocrinopathy, M protein, and skin changes syndrome patients were all shown extensive by MRN. The sensitivity of MRN in diagnosing CIDP was 81.25% (26/32), the specificity was 68.18% (15/22), the positive predictive value was 78.79% (26/33), the negative predictive value was 71.43% (15/21), the accuracy was 75.93% (40/54), the misdiagnosis rate was 24.07% (13/54), and the kappa value was 0.498. Receiver operating characteristic analysis showed higher diagnostic accuracy for CIDP with the CSA of the lumbosacral plexus (area under the curve [AUC] = 0.762) and that of the brachial plexus (AUC = 0.762), and the combined of both examinations did not improve the diagnostic efficacy compared with either (AUC = 0.769).

Conclusion. The nerve roots of CIDP, Charcot-Marie-Tooth disease type-1, and polyneuropathy, organomegaly, endocrinopathy, M protein, and skin changes syndrome were difficult to distinguish by MRN. Atypical CIDP patients had less nerve root injury compared with typical CIDP patients. MRN of either the brachial plexus or the lumbosacral plexus had a high diagnostic accuracy for CIDP, and it is not necessary to perform both parts of the examination.

Key words: brachial plexus, chronic inflammatory demyelinating polyradiculoneuropathy, lumbosacral plexus, magnetic resonance neurography, nerve root cross-sectional area.

Level of Evidence: 2

Spine 2020;45:1506–1512

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated disease that targets the myelin sheaths of peripheral nerves and causes a symmetrical motor-sensory disturbance. Usually, lumbosacral plexus injury is more common than brachial plexus injury.[1–3] Neuropathological biopsy samples have shown the demyelination of medullated nerve fibers, the proliferation of Schwann cells, and the formation of onion skin-like structures.[4] Nerve biopsy is a traumatic examination that can cause irreversible nerve damage to the patient. Lumbar puncture is also an invasive examination with certain pain and risk. The operators of the electrophysiological testing are required to be highly skilled because they have a certain degree of subjective judgment for the results. Gadolinium-enhanced magnetic resonance imaging of spinal roots and a trial of immunotherapy with an objective assessment of endpoints may assist the diagnosis.[5] The diagnosis of CIDP relies on a combination of clinical, electrodiagnostic, and laboratory features such as cerebrospinal fluid analysis and nerve biopsy to exclude other diseases that look like CIDP.

From the [a]Department of Neurology, Renmin Hospital of Wuhan University, Wuhan, China; and [b]Department of Radiology, Union Hospital of Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Acknowledgment date: December 24, 2019. First revision date: March 31, 2020. Acceptance date: April 18, 2020.

CZ and ZL contributed as co-corresponding authors.

The manuscript submitted does not contain information about medical device(s)/drug(s).

No funds were received in support of this work.

No relevant financial activities outside the submitted work.

Address correspondence and reprint requests to Zuneng Lu, PhD, Department of Neurology, Renmin Hospital of Wuhan University, Wuhan 430060, Hubei Province, P.R. China; E-mail: lzn196480@126.com

DOI: 10.1097/BRS.0000000000003599

With the development of science and technology, the quantification of nerves under imaging has become possible.[6,7] In particular, magnetic resonance neurography (MRN) has rapidly promoted the development of peripheral nerve imaging and can be used not only to survey the diameter and cross-sectional area (CSA) of nerves but also to determine the volume.[8,9] Three-dimensional sampling perfection with application-optimized contrasts using different flip angle evolutions (3D SPACE) is a more commonly used MRN sequence with high soft-tissue resolution and multiparameter imaging, and because of the advantage of comprehensive imaging, 3D SPACE has gradually become the leading method to assess damage to the plexuses.[10]

However, to the best of our knowledge, all studies that have been reported were about the diagnostic capacity of imaging techniques for CIDP rather than their differential diagnostic value because their control groups were healthy people and not patients who have other peripheral nerve diseases for which their clinical manifestations are similar to those of CIDP.[11–13] Therefore, the purpose of this study was to explore the value of 3D SPACE to distinguish CIDP from other peripheral neuropathies from both qualitative and quantitative aspects.

MATERIALS AND METHODS

Patients

Between May 2017 and May 2019, 32 consecutive patients with CIDP were recruited prospectively (24 males and 8 females; age range 18–74 yrs old; mean age 49.03 yrs, SD ± 14.02 yrs). For the diagnosis of definite CIDP, we used the diagnostic criteria proposed by the Joint Task Force of the European Federation of Neurological Societies and the Peripheral Nerve Society to classify them as typical CIDP and atypical CIDP.[5] The clinical presentations of typical CIDP were a chronically progressive or recurrent symmetric proximal and distal weakness, sensory dysfunction, and tendon reflexes absent or reduced of all extremities.[14] The atypical CIDP were regarded as the clinical variants of CIDP which were classified into five subtypes according to various clinical symptoms that included distal acquired demyelinating symmetric neuropathy, purely motor or sensory CIDP, Lewis-Sumner syndrome , and focal CIDP.[15,16] In addition, 22 non-CIDP patients with symptoms similar to CIDP and difficult to be discriminate were recruited and imaged as a control group (14 males and 8 females; age range 14–76 yrs old; mean age 40.65 yrs, SD ± 17.65 yrs),and which were divided into two types, diseases with thickened nerve roots and diseases with nonthickened nerve roots. Thickened nerve roots peripheral neuropathies included Charcot-Marie-Tooth disease type-1 (CMT-1) (n = 5) and polyneuropathy, organomegaly, endocrinopathy, M protein and skin changes (POEMS) syndrome (n = 3). Nonthickened nerve roots peripheral neuropathies included CMT-2 (n = 2), hereditary neuropathy with pressure palsies (n = 4), brachial plexus injury (n = 2), diabetic peripheral neuropathy (n = 1), multifocal motor neuropathy (n = 1), chronic axonal peripheral neuropathy (n = 1), vitamin B12 deficiency peripheral neuropathy (n = 1), and multiple system atrophy (n = 1). No patients with CIDP had a family history of inherited peripheral neuropathies. For clinical assessment, the Hughes grade was used as the disability grade scale, and its score ranged from 0 (no signs of disability) to 6 (death).[17]

Imaging Technique

All participants were prospectively examined with a 3.0 T MR scanner (Magnetom Trio, Siemens Healthcare, Erlangen, Germany) using the three-dimensional sampling perfection with application-optimized contrasts using different flip angle evolution (3D SPACE) sequences with a neck matrix coil, and three body matrix anterior coils were applied. Subjects were placed in the gantry in the supine position with their head in the neutral position and instructed to breathe calmly. The contrast agent (0.1 mL/kg, Gadovist; Bayer Pharma AG) was intravenously administered before the brachial plexus and lumbosacral plexus were enhanced for scanning. 3D SPACE parameters were as follows: TR/TE = 3000/270 ms, FOV = 448 × 448 mm^2, voxel size = 1.0 × 1.0 × 1.0 mm^3, slice thickness = 1.0 mm, slice gap = 0 mm, slice = 144. The acquisition time of the brachial plexus and lumbosacral plexus imaging was 20 minutes.

Maximum Indensity Projection images were reconstructed by built-in 3D postprocessing software (3D Syngo MR workspace; Siemens Healthcare, Erlangen, Germany). The bilateral CSA of the nerves at the C7-C8 and L4-S1 levels was measured on the coronal planes. All work was completed independently by two senior radiologists blinded to all of the patients' information, and each side's average CSA of the brachial and lumbosacral nerves roots was calculated separately.

Written informed consent was obtained from all patients before participation. The Clinical Research Ethics Committee approved this prospective study of Renmin Hospital of Wuhan University (2017K-045), and all the procedures were performed following the relevant guidelines/regulations in the Declaration of Helsinki.

Statistical Analysis

Statistical analysis was performed in SPSS 21.0 (SPSS, Chicago, IL). All values were shown as the mean ± standard deviation unless stated otherwise. Nonparametric tests (Kruskal–Wallis test) were used for continuous variables and chi-squared tests were used for categorical variables. Receiver operating characteristic (ROC) curves were used to assess diagnostic accuracy, and the respective area under the curve (AUC) values are reported. Two-sided *P* values were calculated for all analyses. $P < 0.05$ was considered significant.

RESULTS

Clinical Characteristics

The clinical data for patients with CIDP and the control group are summarized in Table 1. CMT-1 is the most common type of CMT, and its predominant damage is nerve demyelination. The nerve roots of CMT-1 and POEMS syndrome were

TABLE 1. Clinical Features of Different Groups

Variables	CIDP (n = 32)	Thickened Nerve Roots Diseases (n = 8)		Nonthickened Nerve Roots Diseases (n = 14)	*P* Value
		CMT-1 (n = 5)	POEMS Syndrome (n = 3)		
Male/female	24/8	4/1	2/1	8/6	0.659
Age (yrs)	49.03 ± 14.02	22.40 ± 9.32	46.33 ± 6.66	43.85 ± 19.69	0.016*
Age at onset (yrs)	43.58 ± 14.96	22.67 ± 5.77	44.00 ± 5.29	36.54 ± 21.81	0.033*
Disease duration (yrs)	3.645 ± 2.94	2.50 ± 0.87	2.33 ± 1.53	7.35 ± 7.93	0.270
Hughes grade	2.13 ± 0.85	2.00 ± 1.41	2.00 ± 1.00	1.77 ± 0.60	0.270
Average CSA					
Brachial plexus	36.30 ± 25.44	28.53 ± 11.77	39.99 ± 9.63	18.26 ± 4.83	<0.001†; 0.001§
Lumbosacral plexus	69.82 ± 38.15	69.08 ± 43.34	61.02 ± 26.76	28.48 ± 8.92	<0.001†; 0.004‡

**CIDP versus CMT-1.*

†CIDP versus nonthickened nerve roots diseases.

‡CMT-1 versus nonthickened nerve roots diseases.

§POEMS versus nonthickened nerve roots diseases.

CIDP indicates chronic inflammatory demyelinating polyradiculoneuropathy; CMT-1, Charcot Marie Tooth type 1; CSA, cross-sectional area; POEMS, polyneuropathy, organomegaly, endocrinopathy, M protein and skin changes.

obviously enlarged and difficult to distinguish from CIDP by MRN, so they were listed and analyzed separately. No significant differences were noted with gender, disease duration, or Hughes grades among the different groups.

MRN of the Brachial Plexus and Lumbosacral Plexus

No significant difference among CIDP, CMT-1, and POEMS syndrome was found in clinical and CSA data, except the age at evaluation and onset for CMT-1 patients were younger than those of CIDP patients ($P = 0.016$ and $P = 0.033$). CIDP, CMT-1, and POEMS syndrome all showed nerve root thickening and inflammatory changes on MRN, which were difficult to distinguish among each other but easy to distinguish from the nonthickened nerve root disease. In our study, five patients with CMT-1 were recognized as having CIDP, and the misdiagnosis rate for CMT-1 was up to 100%. Two of three POEMS syndrome were recognized as CIDP by MRN and the misdiagnosis rate for POEMS syndrome was 66.7%. Only one POEMS syndrome patient was not recognized as CIDP because MRN showed the bone cortical destruction invaded the surrounding soft tissues, which indicated a high possibility for neoplastic lesions (Figure 1A–D). Compared with nonthickened nerve roots peripheral neuropathies, patients with CIDP and CMT-1 had a larger CSA of brachial plexus nerve roots ($P < 0.001$ and 0.001), and patients with CIDP and POEMS syndrome had a larger CSA of lumbosacral plexus nerve roots ($P < 0.001$ and $P = 0.004$) (Figure 2).

Comparison of Detection Rates

The comparison between MRN diagnosis results and clinical diagnosis results is shown in Table 1. Expert

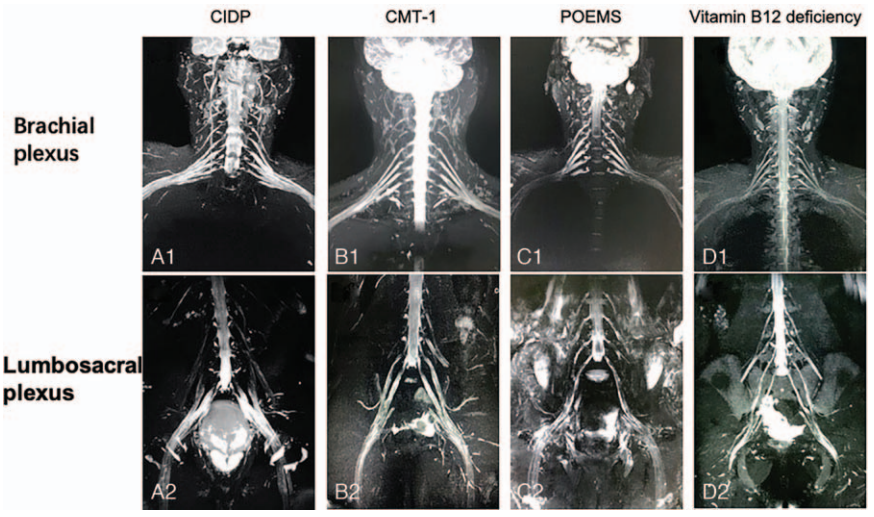

Figure 1. Representative MRN images of MIP reconstruction of the brachial plexus and lumbosacral plexus of CIDP, CMT-1, POEMS syndrome, and HNPP patients. A, A 34-year-old CIDP patient with thickened nerve roots, uneven signal, and irregular borders. B, An 18-year-old CMT-1 patient with evenly thickened nerve roots, symmetrically uniform signals, and clear edges. C, A 52-year-old POEMS syndrome patient with symmetrically thickened nerve roots, uneven signals, and a clear border. D, A 37-year-old vitamin B12 deficiency peripheral neuropathy patient with the brachial plexus and lumbosacral plexus roots are thinner, the edges are smooth, and the morphology and signal are not abnormal. CIDP indicates chronic inflammatory demyelinating polyradiculoneuropathy; CMT-1, Charcot Marie Tooth disease type 1; nr-CSA, cross-sectional area of nerves roots; POEMS, polyneuropathy, organomegaly, endocrinopathy, M protein and skin changes; MRN, magnetic resonance neurography.

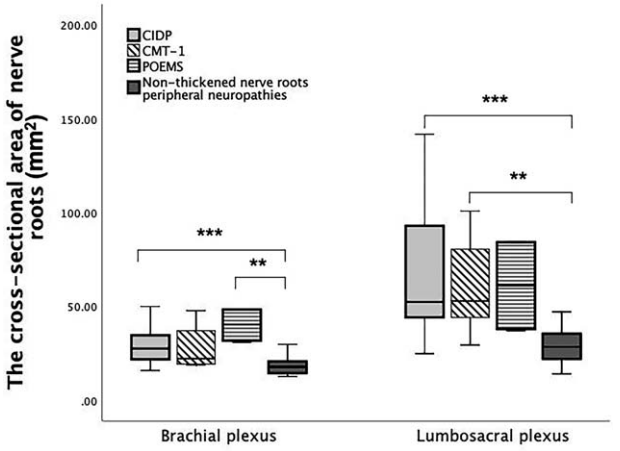

Figure 2. The CSA of brachial plexus and lumbosacral plexus in different groups. CIDP and CMT-1 patients had a larger CSA of brachial plexus nerve roots compared with nonthickened nerve roots peripheral neuropathies ($P = 0.000$ and 0.001). CIDP and POEMS syndrome patients had a larger CSA of lumbosacral plexus nerve roots compared with nonthickened nerve roots peripheral neuropathies ($P = 0.000$ and $P = 0.004$). CIDP indicates chronic inflammatory demyelinating polyradiculoneuropathy; CMT-1, Charcot Marie Tooth disease type 1; CSA, cross-sectional area; POEMS, polyneuropathy, organomegaly, endocrinopathy, M protein and skin changes.

neuroradiologists were blinded to the clinical information results so as to judge whether a patient had CIDP only through the imaging changes in patients' MRN results. Of the 54 patients, 33 were diagnosed with CIDP by MRN, including 26 CIDP patients confirmed by clinicians, 5 CMT-1 patients, and 2 POEMS syndrome patients. Of the 21 patients who were not diagnosed with CIDP by MRN, 6 were confirmed atypical CIDP by clinicians, and 15 other patients had various peripheral neuropathies (Table 2).

The sensitivity of MRN in diagnosing CIDP was 81.25% (26/32), the specificity was 68.18% (15/22), the positive predictive value was 78.79% (26/33), the negative predictive value was 71.43% (15/21), the accuracy was 75.93% (41/54), the misdiagnosis rate was 24.07% (13/54), and the kappa value was 0.498.

ROC Analysis of the Brachial Plexus and Lumbosacral Plexus

Among the 32 patients, 5 (15.6%) patients started with upper limb symptoms, 16 (50%) patients started with lower limb symptoms, and 11 (34.4%) patients started with both extremities simultaneously. In four of the five patients with CIDP who developed numbness and pain in both upper limbs, the lumbosacral nerve roots were also thickened or inflamed. To evaluate whether the combined diagnosis of brachial with lumbosacral plexus by MRN is more effective than that of brachial or lumbosacral plexus alone in patients with CIDP.

In this study, MRN of either the brachial plexus or lumbosacral plexus alone was an auxiliary diagnosis method for CIDP, each with an AUC of 0.762 (95% CI, 0.645–0.878 and 0.653–0.871, respectively) in ROC curve analysis. MRN of the brachial plexus and lumbosacral plexus combined did not significantly increase the diagnostic value for CIDP compared with MRN of either of them alone, with an AUC of 0.769 (95% CI, 0.661–0.877) (Figure 3).

TABLE 2. Comparison Between MRN Diagnosis Results and Clinical Diagnosis Results

MRN	Clinical Diagnosis		Total
	Positive	Negative	
Positive	26	7	33
Negative	6	15	21
Total	32	22	54

MRN indicates magnetic resonance neurography.

DISCUSSION

Few studies have evaluated the differential diagnostic value of MRN for CIDP. Few studies have evaluated the differential diagnostic value of MRN for CIDP. In our study, the symptoms of patients in the control group were similar to those of patients with CIDP, which showed symmetrical or asymmetrical motor-sensory disturbance, reflexes weakened, chronic disease course, and the electrophysiology showed multiple nerves damage, so it is challenging to differentiate these patients in clinical practice. The controls might suffer from hereditary peripheral neuropathy, such as CMT or HNPP, or other diseases that cause peripheral nerve damage, such as diabetes, POEMS syndrome, or nerve injury. These patients required MRN to aid in their diagnosis.[18,19]

The results of our data show that the nerve roots in CIDP, CMT-1, and POEMS syndrome patients were all enlarged on MRN, and the CSA among them showed no significant difference. These three diseases were difficult to identify, and the misdiagnosis rate was high; therefore, the imaging results needed to be combined with clinical assessments. Compared with CMT-1 patients, our data showed that the onset ages of CIDP patients were greater. CMT-1 and CIDP are both demyelinating motor-sensory neuropathies, but the former is an acquired disease, and the latter is congenital. Studies on distinguishing CIDP and CMT-1 have reported that CIDP shows greater asymmetry in motor nerve conduction velocity and compound muscle action potential amplitudes than CMT-1, and the CSA of the peripheral nerves of CMT-1 patients was larger than that of CIDP patients on nerve ultrasonography.[20,21] Despite this, it is difficult to distinguish these diseases because their clinical symptoms, electromyography results, and imaging results are all similar. In most cases, the diagnosis of CMT-1 was confirmed by peripheral myelin protein-22 gene analysis.[22] Polyneuropathy is often an initial manifestation of POEMS syndrome, and this disorder is frequently misdiagnosed as CIDP because of its low incidence and similar symptoms to CIDP. In our study, the clinical data, disability scores, and CSA on MRN of POEMS syndrome and CIDP all showed

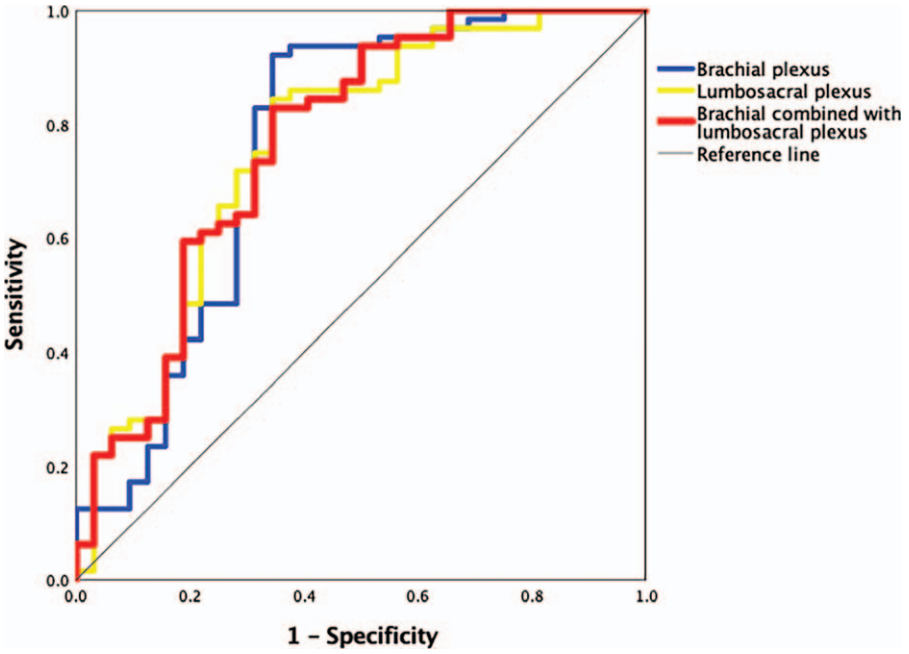

Figure 3. ROC analysis for the brachial plexus, the lumbosacral plexus, and the brachial plexus and lumbosacral plexus combined in the diagnostic value of MRN in CIDP. MRN of either the brachial plexus or the lumbosacral plexus could effectively diagnose CIDP, with an AUC of 0.762 (95% CI, 0.645–0.878 and 0.653–0.871), respectively. MRN of the brachial plexus and lumbosacral plexus combined did not significantly increase the diagnostic value for CIDP compared with MRN of either of them alone, with an AUC of 0.769 (95% CI, 0.661–0.877). AUC indicates area under the curve; CI, confidence interval; ROC, receiver operating characteristic; MRN, magnetic resonance neurography; CIDP, chronic inflammatory demyelinating polyradiculoneuropathy.

no significant differences. Still, the MRN of one patient showed the damage to the bone cortex, the unclear boundaries, and the nearby muscle groups were invaded, which was recognized POEMS syndrome. Studies have reported that POEMS syndrome has a greater axonal loss and greater slowing of the intermediate nerve segments than CIDP, and nerve biopsy can be helpful in distinguishing them.[23–25] The clinical characteristic comparison between CIDP and other peripheral neuropathies showed no differences except the CSA of the brachial plexus and lumbosacral plexus of CIDP patients was significantly increased compared with that of the other patients. Therefore, we could use MRN as a method to identify CIDP and other peripheral neuropathies, but CMT-1 and POEMS still need to be recognized by genetic testing and other clinical examinations and laboratory analyses.

Our study is the first to discuss the noninvasive discriminative diagnostic value of MRN for CIDP from qualitative and quantitative aspects. Many studies have found that imaging examinations could observe the nerve root enlargement and signal intensity changes of patients with disease compared with those of healthy people.[2] These studies compared their CSA and analyzed the correlation among imaging data, electromyography, and clinical features, but they demonstrated only their ability to detect rather than to differentially diagnose.[26–28] In this study, there were 54 patients, including 32 with CIDP and 22 with other peripheral neuropathies. There were five CMT-1 and two POEMS syndrome patients misdiagnosed with CIDP, and six CIDP patients could not be recognized by MRN who had atypical CIDP (kappa = 0.498). These results indicate that MRN had a moderate level of consistency with clinical diagnosis results. The typical CIDP patients were all recognized by MRN, and it may be that the brachial plexus and lumbosacral plexus lesions in atypical CIDP patients are less severe than those in typical CIDP patients.

In the quantitative study of MRN for the diagnosis of CIDP, we measured the CSA of the brachial plexus and lumbosacral plexus nerve roots of all patients and used an ROC curve to evaluate the value of MRN in independently diagnosing the CIDP patients. The AUC of the CSA of the brachial plexus and lumbosacral plexus nerve roots are both 0.762, and our study confirmed their diagnostic value for CIDP patients, but we also wanted to know whether it is necessary to measure both sites because either site has the same diagnostic value, and the cost of MRN testing is expensive. When we evaluated the MRN of the brachial plexus and lumbosacral plexus combined, the AUC for diagnostic efficacy reached 0.769, which suggests that double-site examination does not significantly improve the diagnostic efficacy of MRN in CIDP compared with single-site testing.

The main limitation of our study was the small sample size. In particular, we compared CMT-1 and POEMS separately with other peripheral neuropathies, which could bring about bias. However, to reduce the bias as much as possible, we measured bilateral sides data of the CSA of the nerves at the C7-C8 and L4-S1 levels. Another limitation for this study was that our control group composed of not a single disease but different diseases with different numbers, so the results about kappa value and AUC would be changed if types and numbers of patients changed. In this case, we want to emphasize that the result about differential diagnosis efficacy just for the data of this study without extension. The main findings were that CMT-1 and POEMS were difficult to be distinguished with CIDP by MRN but

nonthickened nerve roots peripheral neuropathies were easier relatively.

In conclusion, as the first study to discuss the diagnostic value of MRN for CIDP from qualitative and quantitative aspects, this study finds that CIDP, CMT-1, and POEMS are similar in clinical features, disability score, and the CSA of nerve roots, and they were difficult to be discriminate by MRN. The MRN can be used to distinguish thickened nerve roots peripheral neuropathies and nonthickened nerve roots peripheral neuropathies. The CSA of the brachial plexus and lumbosacral plexus nerve roots of CIDP patients is significantly larger than that of patients with other peripheral neuropathies, excluding CMT-1 and POEMS. The brachial plexus and lumbosacral plexus lesions in atypical CIDP patients are less severe than those in typical CIDP patients, and typical CIDP patients are more likely to be identified by MRN. The diagnosis of CIDP by MRN is moderately consistent with the clinical diagnostic results. MRN of either the brachial plexus or the lumbosacral plexus has a high diagnostic accuracy for CIDP, and MRN of the brachial plexus and the lumbosacral plexus combined does not significantly improve diagnostic accuracy; therefore, it is not necessary to perform an MRN examination of both plexuses.

➢ Key Points

- ❑ This was the first study to discuss the diagnostic value of MRN for CIDP from qualitative and quantitative aspects.
- ❑ This study finds that CIDP, CMT-1, and POEMS are similar in clinical features, disability score, and the CSA of nerve roots, and they were difficult to be discriminate by MRN.
- ❑ MRN of either the brachial plexus or the lumbosacral plexus has a high diagnostic accuracy for CIDP, and MRN of the brachial plexus and the lumbosacral plexus combined examination does not significantly improve diagnostic accuracy; therefore, it is not necessary to perform an MRN examination of both plexuses.

References

1. Hiwatashi A, Togao O, Yamashita K, et al. Lumbar plexus in patients with chronic inflammatory demyelinating polyneuropathy: evaluation with 3D nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation enhancement imaging (3D SHINKEI). *Eur J Radiol* 2017;93:95–9.
2. Tazawa K, Matsuda M, Yoshida T, et al. Spinal nerve root hypertrophy on MRI: clinical significance in the diagnosis of chronic inflammatory demyelinating polyradiculoneuropathy. *Intern Med* 2008;47:2019–24.
3. Adachi Y, Sato N, Okamoto T, et al. Brachial and lumbar plexuses in chronic inflammatory demyelinating polyradiculoneuropathy: MRI assessment including apparent diffusion coefficient. *Neuroradiology* 2011;53:3–11.
4. Mathey EK, Park SB, Hughes RAC, et al. Chronic inflammatory demyelinating polyradiculoneuropathy: from pathology to phenotype. *J Neurol Neurosurg Psychiatry* 2015;86:973–85.
5. Joint Task Force of the EFNS and the PNS. European Federation of Neurological Societies/Peripheral Nerve Society Guideline on management of chronic inflammatory demyelinating polyradiculoneuropathy: report of a joint task force of the European Federation of Neurological Societies and the Peripheral Nerve Society—First Revision. *J Peripher Nerv Syst* 2010;15:1–9.
6. Kronlage M, Pitarokoili K, Schwarz D, et al. Diffusion tensor imaging in chronic inflammatory demyelinating polyneuropathy: diagnostic accuracy and correlation with electrophysiology. *Invest Radiol* 2017;52:701–7.
7. Niu J, Li Y, Liu T, et al. Serial nerve ultrasound and motor nerve conduction studies in chronic inflammatory demyelinating polyradiculoneuropathy. *Muscle Nerve* 2019;60:254–62.
8. Pitarokoili K, Schlamann M, Kerasnoudis A, et al. Comparison of clinical, electrophysiological, sonographic and MRI features in CIDP. *J Neurol Sci* 2015;357:198–203.
9. Ishikawa T, Asakura K, Mizutani Y, et al. MR neurography for the evaluation of CIDP. *Muscle Nerve* 2017;55:483–9.
10. Zhang X, Li M, Guan J, et al. Evaluation of the sacral nerve plexus in pelvic endometriosis by three-dimensional MR neurography. *J Magn Reson Imaging* 2017;45:1225–31.
11. Gilmore KJ, Doherty TJ, Kimpinski K, et al. Reductions in muscle quality and quantity in chronic inflammatory demyelinating polyneuropathy patients assessed by magnetic resonance imaging. *Muscle Nerve* 2018;58:396–401.
12. Jang JH, Cho CS, Yang KS, et al. Pattern analysis of nerve enlargement using ultrasonography in chronic inflammatory demyelinating polyneuropathy. *Clin Neurophysiol* 2014;125:1893–9.
13. Hiwatashi A, Togao O, Yamashita K, et al. Simultaneous MR neurography and apparent T2 mapping in brachial plexus: evaluation of patients with chronic inflammatory demyelinating polyradiculoneuropathy. *Magn Reson Imaging* 2019;55:112–7.
14. Gorson KC, Ropper AH. Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP): a review of clinical syndromes and treatment approaches in clinical practice. *J Clin Neuromuscul Dis* 2003;4:174–89.
15. Nobile-Orazio E. Chronic inflammatory demyelinating polyradiculoneuropathy and variants: where we are and where we should go. *J Peripher Nerv Syst* 2014;19:2–13.
16. Kuwabara S, Isose S, Mori M, et al. Different electrophysiological profiles and treatment response in 'typical' and 'atypical' chronic inflammatory demyelinating polyneuropathy. *J Neurol Neurosurg Psychiatry* 2015;86:1054–9.
17. Hughes RA, Newsom-Davis JM, Perkin GD, et al. Controlled trial prednisolone in acute polyneuropathy. *Lancet* 1978;2:750–3.
18. Hiwatashi A, Togao O, Yamashita K, et al. Evaluation of chronic inflammatory demyelinating polyneuropathy: 3D nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation enhancement imaging (3D SHINKEI). *Eur Radiol* 2017;27:447–53.
19. Kronlage M, Bäumer P, Pitarokoili K, et al. Large coverage MR neurography in CIDP: diagnostic accuracy and electrophysiological correlation. *J Neurol* 2017;264:1434–43.
20. Kang JH, Kim HJ, Lee ER. Electrophysiological evaluation of chronic inflammatory demyelinating polyneuropathy and Charcot-Marie-Tooth type 1: dispersion and correlation analysis. *J Phys Ther Sci* 2013;25:1265–8.
21. Sugimoto T, Ochi K, Hosomi N, et al. Ultrasonographic nerve enlargement of the median and ulnar nerves and the cervical nerve roots in patients with demyelinating Charcot-Marie-Tooth disease: distinction from patients with chronic inflammatory demyelinating polyneuropathy. *J Neurol* 2013;260:2580–7.
22. Sevilla T. The genetics of type 1 Charcot-Marie-Tooth disease, the hereditary focal neuropathies and the hereditary distal motor neuropathies. *Rev Neurol* 2000;30:71–9.

23. Guo X, Qin X, Zhang Y, et al. Electrophysiological features of POEMS syndrome and chronic inflammatory demyelinating polyneuropathy. *J Clin Neurosci* 2014;21:587–90.
24. Mauermann ML, Sorenson EJ, Dispenzieri A, et al. Uniform demyelination and more severe axonal loss distinguish POEMS syndrome from CIDP. *J Neurol Neurosurg Psychiatry* 2012;83:480–6.
25. Piccione EA, Engelstad J, Dyck PJ, et al. Nerve pathologic features differentiate POEMS syndrome from CIDP. *Acta Neuropathol Commun* 2016;4:116.
26. Shibuya K, Sugiyama A, Ito S, et al. Reconstruction magnetic resonance neurography in chronic inflammatory demyelinating polyneuropathy. *Ann Neurol* 2015;77:333–7.
27. Matsuoka N, Kohriyama T, Ochi K, et al. Detection of cervical nerve root hypertrophy by ultrasonography in chronic inflammatory demyelinating polyradiculoneuropathy. *J Neurol Sci* 2004;219:15–21.
28. Schady W, Goulding PJ, Lecky BR, et al. Massive nerve root enlargement in chronic inflammatory demyelinating polyneuropathy. *J Neurol Neurosurg Psychiatry* 1996;61:636–40.