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Original contribution

Simultaneous MR neurography and apparent T2 mapping in brachial plexus: Evaluation of patients with chronic inflammatory demyelinating polyradiculoneuropathy

Akio Hiwatashi[a,*], Osamu Togao[b], Koji Yamashita[b], Kazufumi Kikuchi[b], Daichi Momosaka[b], Hiroshi Nakatake[b], Ryo Yamasaki[c], Hidenori Ogata[c], Masami Yoneyama[d], Jun-ichi Kira[c], Hiroshi Honda[b]

[a] Departments of Molecular Imaging & Diagnosis, Graduate School of Medical Sciences, Kyushu University, Japan
[b] Clinical Radiology, Graduate School of Medical Sciences, Kyushu University, Japan
[c] Neurology, Graduate School of Medical Sciences, Kyushu University, Japan
[d] MR Clinical Science, Philips Japan, Tokyo, Japan

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ABSTRACT

Purpose: MR neurography is known to be useful to evaluate nerve pathology. The purpose of this study was to evaluate the usefulness of simultaneous apparent T2 mapping and neurography with nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation enhancement imaging (SHINKEI) to distinguish patients with chronic inflammatory demyelinating polyneuropathy (CIDP) from healthy subjects.

Materials and methods: This retrospective study included 13 patients with CIDP and five healthy subjects from 2015 to 2017. The T2 relaxation time and the size of the cervical ganglia and roots of the brachial plexus were measured. Statistical analyses were performed with the Mann-Whitney *U* test and receiver operating characteristics (ROC) analysis.

Results: The T2 relaxation times of the ganglia and roots were longer in patients with CIDP (119.31 ± 35.53 msec and 111.15 ± 33.82 msec) than in healthy subjects (101.42 ± 26.42 msec and 85.29 ± 13.22 msec, $P = 0.0007$ and $P < 0.0001$, respectively). The sizes of the ganglia and the roots were larger in patients with CIDP (6.25 ± 1.56 mm and 4.37 ± 1.71 mm) than in healthy subjects (5.59 ± 1.08 mm and 3.50 ± 0.62 mm, $P = 0.0114$ and $P = 0.0014$, respectively). ROC analysis revealed that T2 relaxation time of the roots was best at distinguishing CIDP patients from healthy subjects (the area under the curve = 0.748).

Conclusion: Patients with CIDP could be distinguished from healthy subjects using simultaneous apparent T2 mapping and neurography with SHINKEI.

1. Introduction

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a demyelinating disease of the peripheral nerves presenting with both sensory and motor symptoms [1–12]. It could be presenting as a monophasic, relapsing or progressive manner with insidious or stuttering progression or episodic exacerbations.

Previously various imaging modalities have been proposed to diagnose or monitor the patients' symptoms including plain X-ray [4], myelogram [2], Magnetic resonance (MR) imaging [3,4], and ultrasound [5]. MR neurography [6,7,9–21] is a useful MR technique with which to visualize nerves and evaluate them. It has been applied in patients with tumors, trauma, neuritis and compression of the brachial plexus. For MR neurography, a combination of fat-suppressed T2- and T1-weighted imaging sequences is used to evaluate diseases of the brachial plexus [13]. In addition, Takahara et al. developed MR neurography with diffusion-weighted imaging to visualize peripheral nerves [13].

Abbreviations: AUC, The area under the receiver operating characteristics curve; CIDP, Chronic inflammatory demyelinating polyradiculoneuropathy; iMSDE, improved motion sensitized driven equilibrium; MR, Magnetic resonance; ROC, Receiver operating characteristics; ROI, Region-of-interest; SHINKEI, Nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation imaging

* Corresponding author at: Department of Molecular Imaging & Diagnosis, Graduate School of Medical Sciences, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka 812-8582, Japan.
E-mail address: hiwatasi@radiol.med.kyushu-u.ac.jp (A. Hiwatashi).

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Three-dimensional nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation imaging (SHINKEI) [15] is a kind of new MR neurography method that suppresses the signals of blood vessels, muscles and fat tissue using improved motion-sensitized driven equilibrium (iMSDE) and spectral attenuated inversion recovery [9–11,15,18,19]. With SHINKEI, we can obtain neurography with high spatial resolution and can evaluate the dorsal root ganglia and roots of the brachial plexus, however, studies employing quantitative evaluation of this technique are lacking. Recently, Yoneyama et al. proposed an advanced sequence [19] which could obtain MR neurography and quantitative evaluation of T2 relaxation times simultaneously. However, to the best of our knowledge, there has been no report using SHINKEI with simultaneous apparent T2 mapping to evaluate patients with CIDP. Therefore, the purpose of this study was to evaluate the usefulness of simultaneous apparent T2 mapping and neurography with SHINKEI to distinguish patients with CIDP from healthy subjects.

2. Materials and methods

This retrospective study was approved by our institutional review boards, and written informed consent was waived.

2.1. Patients

Simultaneous apparent T2 mapping and neurography with SHINKEI has been part of our routine MR protocol for imaging of the brachial plexus. We analyzed the MR imaging data of 13 consecutive patients with CIDP who were identified between October 2015 and October 2017. Diagnosis of CIDP was made when they met the definite diagnostic criteria of the European Federation of Neurological Societies/Peripheral Nerve Society. [1] Exclusion criteria were as follows: (a) patients with diagnoses other than CIDP (n = 50), (b) patients without specific diagnoses (n = 18), and (c) patients with CIDP underwent repeated MR imaging (n = 2). A total of 13 patients (10 males, three females; age range, 11–80 years; median age, 52 years) were identified on the basis of these criteria. We evaluated initial MR imaging in two patients who had repeated examinations during this period. All but two patients underwent previous treatment with intravenous immunoglobulin (n = 11), steroids (n = 7), plasmapheresis (n = 4), and other immunosuppressants (n = 6). Patient demographics are described in Table 1. In addition, five healthy male subjects (five males; age range, 25–43 years; median age, 30 year) without neurological abnormality were included from our database in the same period without neurological abnormality.

Table 1
Patient demographics.

No.	Age	Sex	Previous treatment	CSF protein	Dominant symptoms	Duration (year)
1	34	F	IVIG, PP, IS	68	M > S	0.5
2	52	M	None	48	M > S	2
3	35	M	IVIG, Steroids, PP, IS	228	S > M	4
4	52	M	IVIG	59	M	5
5	61	M	IVIG, Steroids, PP, IS	52	M > S	5
6	22	M	IVIG, Steroids	300	M > S	5
7	42	F	IVIG	70	M > S	7
8	69	M	IVIG, Steroids, IS	40	S	8
9	80	M	None	96	M > S	9
10	65	M	IVIG, Steroids, IS	30	M > S	9
11	57	M	IVIG, Steroids, IS	57	S	12
12	11	F	IVIG, Steroids, PP	110	M > S	15
13	53	M	IVIG	78	M > S	15

Abbreviations: IVIG, intravenous immunoglobulin; PP, plasmapheresis; IS, immunosuppressant; M, motor; S, sensory.

2.2. Imaging technique

All patients underwent MR imaging with a 3.0-T system (Ingenia; Philips, Best, the Netherlands) with a dStream TotalSpine coil. Simultaneous T2 mapping and neurography with SHINKEI was obtained in the coronal plane as part of the brachial plexus MR neurography protocol (Fig. 1). The details of SHINKEI have been described elsewhere [9–11,15,18]. Briefly, it is a turbo spin echo with a diffusion-weighted prepulse called iMSDE to suppress signals from vessels and muscles and a short tau inversion recovery fat-suppression prepulse. These prepulse operations are followed by a readout procedure with tissue-specific variable refocusing flip-angle rapid acquisition with a relaxation enhancement sequence to acquire contrast-efficient T2-weighting [15]. To synthesize MR neurography and estimate apparent T2 relaxation time simultaneously, we applied two different iMSDE prep-times (sequence1 and 2) interleavely (by TR) in one single acquisition. SHINKEI parameters included TR/TE = 2600/61 msec, FOV = 220 × 307 mm, ETL = 100, acquisition matrix = 192 × 265, reconstructed voxel size = 0.60 × 0.60 × 1.15 mm^3, b = 10 s/mm^2, iMSDE prep-time = 36 msec and 72 msec, acquisition time = 6 min 9 s. These images were obtained in a coronal plane. MR neurography was obtained with both iMSDE prep-time as follows;

$$S_{neurography} = S_{preptime-1} + S_{preptime-2}$$

where $S_{neurography}$ indicates the signal intensity of the neurography, and $S_{preptime\text{-}1}$, and $S_{preptime\text{-}2}$ indicate those of the iMSDE durations of 36 and 72 msec, respectively.

T2 mapping was calculated as follows;

$$S_{preptime} = S_0 * e^{(-preptime/T2)}$$

A T2 map was calculated by pixel-by-pixel fitting of the magnitude image intensities from both prep-times to a mono-exponential relaxation model [22].

2.3. Analysis

All MR images were saved as DICOM and viewed using Osirix software version 6.5.2 (Pixmeo, Geneva, Switzerland). Regions of interests were placed at the cervical ganglia and roots of the brachial plexus bilaterally at C5-T1 by a neuroradiologist (A.H., 20 years' experience in neuroradiology). The T2 relaxation times and the sizes of the ganglia and roots were measured. Circular region-of-interests (ROIs) not to include the outside tissue on neurography were placed in the bilateral ganglia or roots at C5-T1 and copied to T2 map to measure T2 relaxation times. Linear ROIs were placed in the same structures on neurography to measure the sizes. Statistical analyses were performed by the same author using JMP software (version 13.0.0; SAS Institute, Cary, NC) and MedCalc Software (version 17.9.7; MedCalc, Mariakerke, Belgium). Techniques used included the Mann-Whitney *U* test, and receiver operating characteristics (ROC) curves [23]. *P*-values < 0.05 were considered significant. The Bonferroni correction was used for multiple comparisons.

3. Results

The T2 relaxation times of the ganglia and roots were longer in patients with CIDP (119.31 ± 35.53 msec and 111.15 ± 33.82 msec) than in healthy subjects (101.42 ± 26.42 msec and 85.29 ± 13.22 msec, P = 0.0007 and P < 0.0001, respectively; Fig. 2). The sizes of the ganglia and the roots were larger in patients with CIDP (6.25 ± 1.56 mm and 4.37 ± 1.71 mm) than in healthy subjects (5.59 ± 1.08 mm and 3.50 ± 0.62 mm, P = 0.0114 and P = 0.0014, respectively; Fig. 3). ROC analysis revealed the best diagnostic performance using T2 relaxation time of the roots. The sensitivity, the specificity and the value of the area under the ROC curve

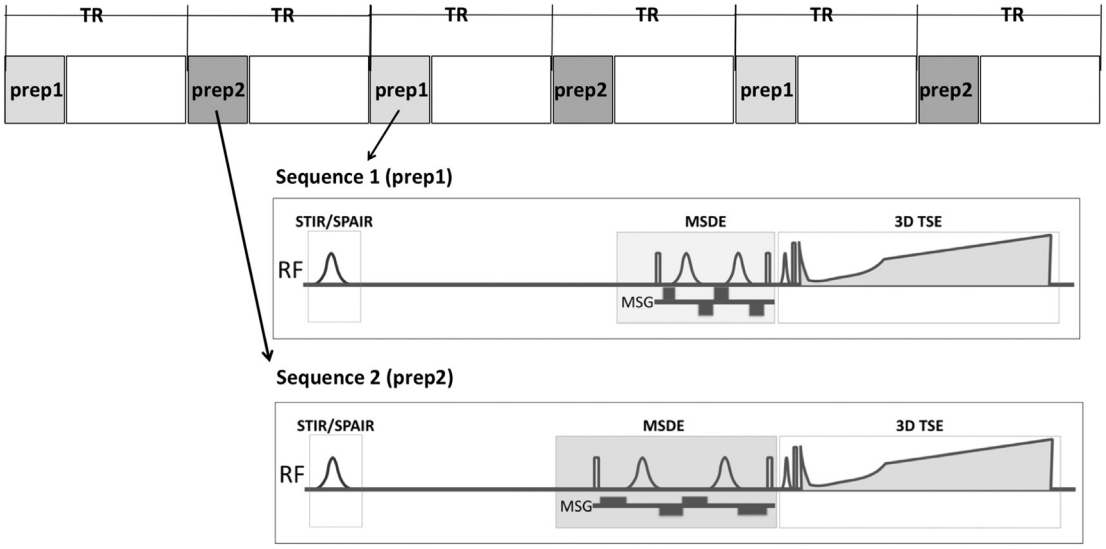

Fig. 1. Diagram of sequences used in this study. SHINKEI consists of iMSDE, inversion-recovery-based fat suppression and 3D T2-weighted TSE sequence. Simultaneous MR neurography and apparent T2 mapping with SHINKEI applies interleavely (by TR) two different prep-times (sequence1 and 2) in one acquisition for synthesis of MR neurography and apparent T2 estimation.

(AUC) were 0.58, 0.74 and 0.66 for the T2 relaxation time, and 0.33, 0.94, and 0.62 for the size of the ganglia, respectively (Fig. 4). These were 0.64, 0.84 and 0.75 for the T2 relaxation time, and 0.36, 1.00, and 0.65 for the size of the roots, respectively. The AUC was the largest with the T2 relaxation time of the roots, however, there were no statistically significant differences among these comparisons ($P > 0.008$; Bonferroni correction). When we used combined values of the size and the T2 relaxation time of the ganglia, the sensitivity, the specificity and the AUC were 0.48, 0.84 and 0.68. These of the roots were 0.65, 0.82, and 0.77, respectively. If we used combined values of the size and the T2 relaxation time of the ganglia and the roots, the sensitivity, the specificity and the AUC were 0.53, 0.96 and 0.78. There was no case with large tarlov cysts or perineural cysts which affected measurement. Representative images depicting a healthy control subject and a patient with CIDP are presented in Figs. 5 and 6.

4. Discussion

In this study, we have demonstrated a new MR neurography technique which enables us to obtain high-resolution MR neurography with simultaneous apparent T2 mapping based on two different T2 preparation times of iMSDE pre-pulse. We observed that the T2 relaxation times of the ganglia and roots of the brachial plexus were longer in patients with CIDP than in healthy controls. Among their sizes and T2 values, we found that T2 relaxation times at the roots of the brachial plexus may be the best to differentiate CIDP patients from healthy controls. Previous qualitative studies have shown increased signal intensity on T2-weighted images in the brachial plexus or nerve trunks in patients with CIDP [4–6,10,11,24]. A previous study with SHINKEI showed that the signal-to-noise ratio, contrast ratio and diameter of the cervical ganglia and roots of the brachial plexus in patients with CIDP were larger than those in healthy subjects [10]. In that study, the AUC was the larger for the SNR of the roots [10]. These findings agreed with those in our study. In this study, we found that the T2 relaxation times at the roots of the brachial plexus as the best to separate CIDP patients from healthy controls, however, its clinical utility might be limited because of low AUC value (0.75). Combination of these parameters could improve the AUC value, but it was slight (0.78).

There have been several previous studies evaluating T2 relaxation times for peripheral nerves of healthy animals. T2 relaxation times of

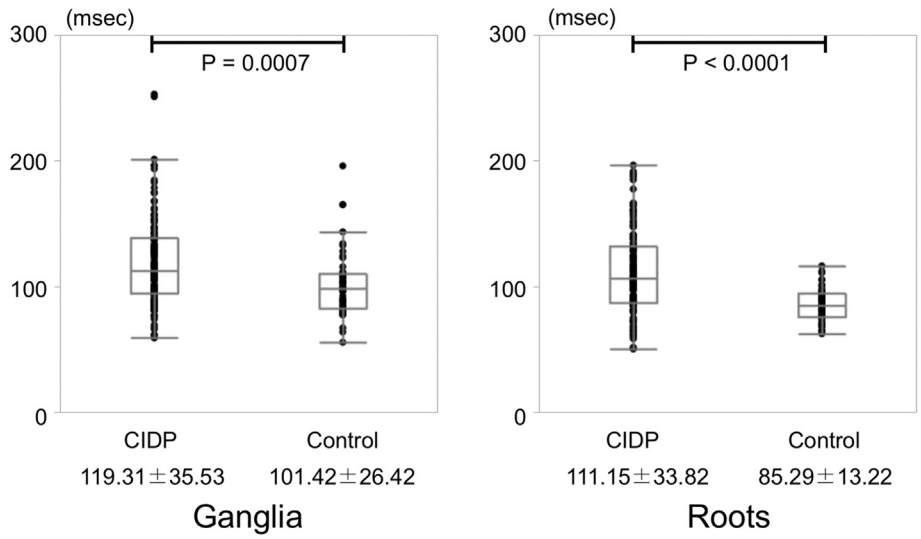

Fig. 2. T2 relaxation times for patients with CIPD and healthy controls in ganglia and roots.

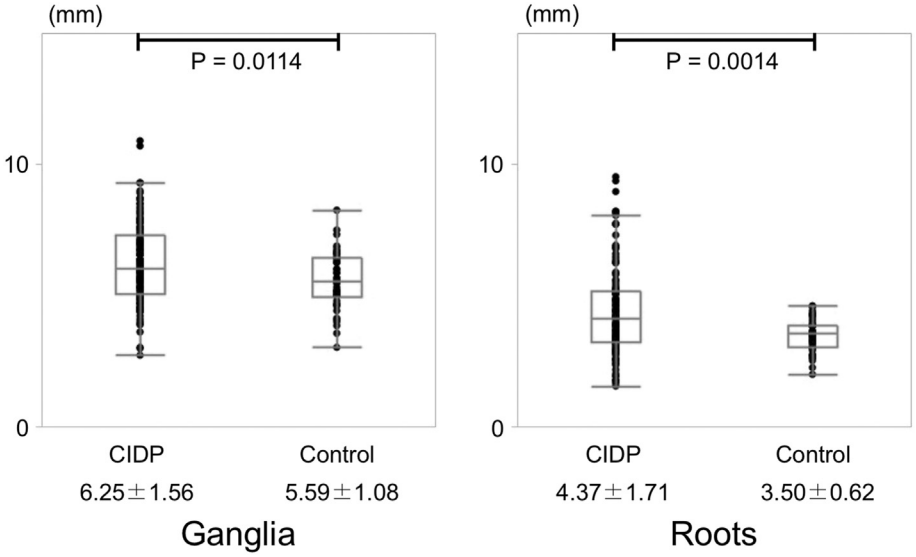

Fig. 3. Sizes for patients with CIPD and healthy controls in ganglia and roots.

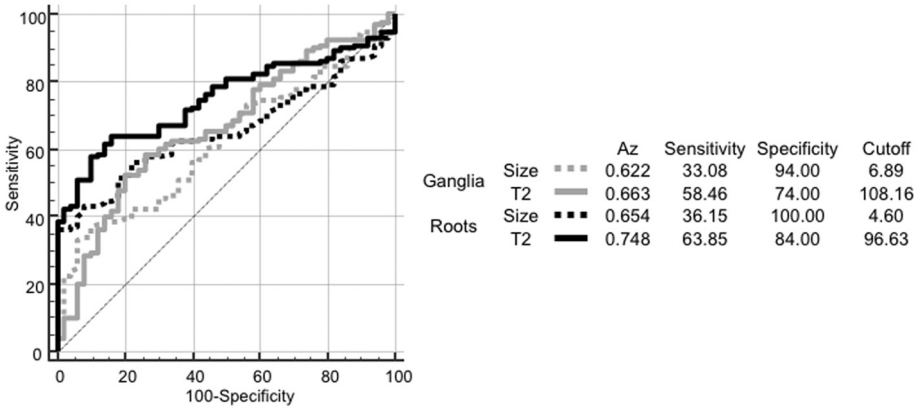

Fig. 4. ROC analysis which reveals the best diagnostic performance using T2 relaxation time of the roots.

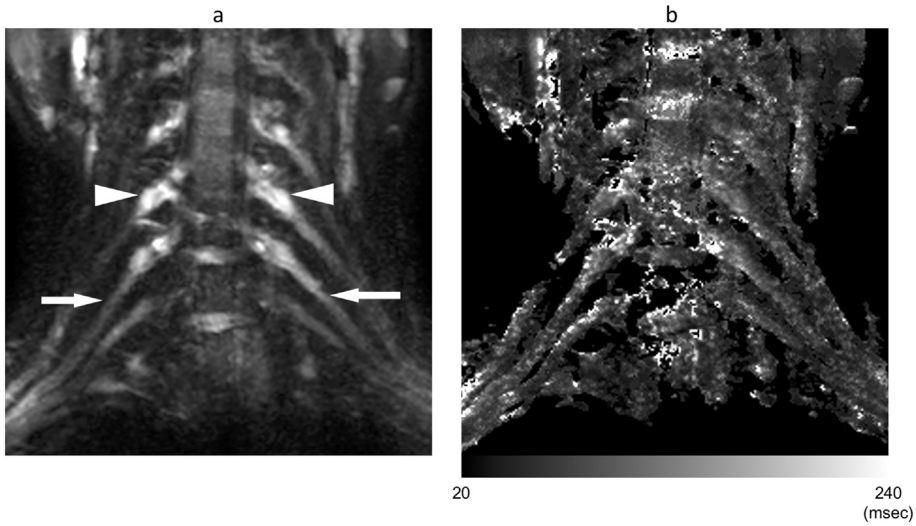

Fig. 5. A 39-year-old male healthy control. MR neurography (a) visualized the ganglia (arrowheads) and roots of the brachial plexus (arrows). T2 relaxation times were 80.40 ± 9.16 msec in the ganglia and 78.69 ± 12.41 msec in the roots (b).

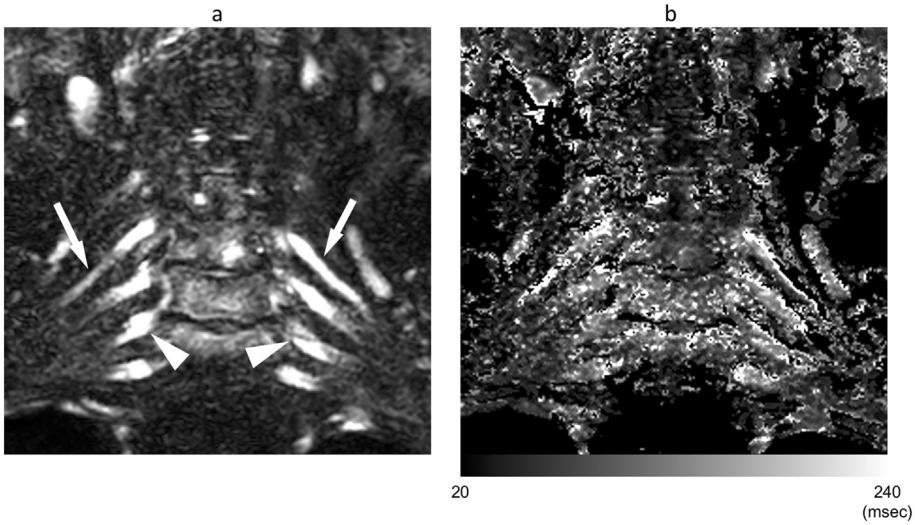

Fig. 6. A 52-year-old male with CIDP. MR neurography (a) visualized the enlarged ganglia (arrowheads) and roots of the brachial plexus (arrows). T2 relaxation times were 110.46 ± 22.67 msec in the ganglia and 101.31 ± 15.45 msec in the roots (b).

the nerves were reported by Zhang et al. as around 50 msec in rats [17] and by Shen et al. as around 40 msec in rabbits [14]. Karampinos et al. evaluated L4 nerves in humans and reported that the T2 relaxation times were statistically significantly longer in the dorsal root ganglia (78.0 ± 11.9 msec) than in more distal regions (59.5 ± 7.4 msec) [16]. In this study, we observed longer T2 relaxation times of the ganglia (111.15 ± 33.82 msec) than of the roots of the brachial plexus (85.29 ± 13.22 msec), which agrees with the previous study. The presence of gray matter in the ganglia may cause longer T2 values in ganglia than in roots of the brachial plexus. However, our observed values were longer than those in previous studies [14,16,17]. We postulate the following reasons for the discrepancy. First, simultaneous apparent T2 mapping and neurography with SHINKEI calculated the T2 relaxation time by only two prep-times, which may cause unstable T2 assessments. T2 quantification errors by T2 prep can occur in the presence of large B1 and B0 inhomogeneities. Particularly at 3T [25]. Weidlich et al. [26] demonstrated that the T2 values are overestimated in the simple exponential two parameter fit when B0 offsets are present because of the influence of T1 relaxation during the T2 preparation module. Since T1 is much larger than T2, the mixed T1 and T2 relaxation effects result in an overestimation of T2. Furthermore, the inversion recovery technique to suppress fat signals in SHINKEI might also alter the quantification of T2 relaxation times by contamination with the T1 effect. However, the clinical utility of our technique, which could obtain high-resolution MR neurography and apparent T2 mapping in the brachial plexus in about 6 min, cannot be denied. T2 relaxation time has been traditionally measured using multi-slice multi-echo spin echo sequences, but it is incompatible with three dimensional acquisitions which might be a disadvantage for MR neurography. Kronlage et al. evaluated the sciatic, tibial, median, ulnar, and radial nerves and reported that the MR signal increase in patients with CIDP was due to an increase in proton-spin-density, and not due to the increase in T2 relaxation time [12]. This was not in line with our results. We need further evaluation with our technique in the extremities in the future.

With other quantitative MR imaging, Adachi et al. evaluated the brachial and lumbar plexuses and reported that the diffusivity in CIDP patients with nerve swelling or abnormal signals was higher than those in CIDP patients without these findings and normal subjects [7]. Kakuda et al. evaluated the tibial nerve with diffusion tensor imaging and reported that the fractional anisotropy in CIDP patients was significantly lower than in healthy volunteers [8]. We have not used diffusion-weighted or tensor imaging. We need further evaluation of the clinical utility of these techniques and current results.

We observed that the sizes of the ganglia and the roots were larger in patients with CIDP than in healthy controls. Previously Symonds et al. reported nerve root enlargement on myelography in patients with CIDP [2]. Matsuoka et al. also reported enlarged cervical nerve roots on ultrasound [5]. Midoroni et al. reported that the nerve roots in the lumbar spine were enlarged, most significantly in the extraforaminal region [3]. Qualitative assessment of nerve hypertrophy also has been reported on MR [6]. These findings likely represent the pathological process of repeated demyelination and remyelination [4,24].

There are several limitations to this study. We quantified T2 relaxation time with two iMSDE duration times, because the other parameters were exactly the same in these two acquisitions. T2 quantification with three or more points might be better, which needs longer acquisition time and might cause image degradation due to motion. A simulation in which the accuracy of T2 estimation is plotted as function of T1 and T2 might be useful to verify our technique, however it might be difficult because of field inhomogeneity especially at 3T. We did not evaluate the entire length of the brachial plexus because of its complex form. We should establish the other analysis method such as automatical tracking of the nerves in future. Lack of contrast material might be another limitation. Because most of the patients included had stable symptoms, they did not want a contrast agent on follow-up MR examination. The small number of patients included and the lack of the evaluation of the other nerves might be other limitations. We are continuing to gather patients with more robust fat suppression techniques with SHINKEI. We did not compare out new technique with a classic SHINKEI showing only neurography because we did not observe a critical difference between our techniques clinically. Comparison with clinical assessment of specific nerves involved and other imaging findings might be preferable, however, it is also difficult to define specific nerves involved because of the various symptoms in patients included in this study.

In conclusion, with our new technique we could obtain high-resolution MR neurography with apparent T2 mapping simultaneously in the cervical ganglia and roots. Patients with CIDP could be distinguished from healthy subjects using simultaneous apparent T2 mapping and neurography with SHINKEI.

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MY is an employee of Philips Japan. He was not involved in data analysis in this study.

References

[1] PNS JTFotEat. European Federation of Neurological Societies/Peripheral Nerve Society Guideline on management of paraproteinemic demyelinating neuropathies. 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(3):185–95.

[2] Symonds CP, Blackwood W. Spinal cord compression in hypertrophic neuritis. Brain 1962;85(2):251–60.

[3] Midroni G, de Tilly LN, Gray B, Vajsar J. MRI of the cauda equina in CIDP: clinical correlations. J Neurol Sci 1999;170(1):36–44.

[4] Duggins AJ, McLeod JG, Pollard JD, Davies L, Yang F, Thompson EO, et al. Spinal root and plexus hypertrophy in chronic inflammatory demyelinating polyneuropathy. Brain 1999;122(Pt 7):1383–90.

[5] Matsuoka N, Kohriyama T, Ochi K, Nishitani M, Sueda Y, Mimori Y, et al. Detection of cervical nerve root hypertrophy by ultrasonography in chronic inflammatory demyelinating polyradiculoneuropathy. J Neurol Sci 2004;219(1–2):15–21.

[6] Tazawa K-I, Matsuda M, Yoshida T, Shimojima Y, Gono T, Morita H, et al. Spinal nerve root hypertrophy on MRI: clinical significance in the diagnosis of chronic inflammatory demyelinating polyradiculoneuropathy. Intern Med 2008;47(23):2019–24.

[7] Adachi Y, Sato N, Okamoto T, Sasaki M, Komaki H, Yamashita F, et al. Brachial and lumbar plexuses in chronic inflammatory demyelinating polyradiculoneuropathy: MRI assessment including apparent diffusion coefficient. Neuroradiology 2011;53(1):3–11.

[8] Kakuda T, Fukuda H, Tanitame K, Takasu M, Date S, Ochi K, et al. Diffusion tensor imaging of peripheral nerve in patients with chronic inflammatory demyelinating polyradiculoneuropathy: a feasibility study. Neuroradiology 2011;53(12):955–60.

[9] Ogata H, Yamasaki R, Hiwatashi A, Oka N, Kawamura N, Matsuse D, et al. Characterization of IgG4 anti-neurofascin 155 antibody-positive polyneuropathy. Ann Clin Transl Neurol 2015;2(10):960–71.

[10] Hiwatashi A, Togao O, Yamashita K, Kikuchi K, Ogata H, Yamasaki R, 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(2):447–53.

[11] Hiwatashi A, Togao O, Yamashita K, Kikuchi K, Kamei R, Momosaka D, 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.

[12] Kronlage M, Baumer P, Pitarokoili K, Schwarz D, Schwehr V, Godel T, et al. Large coverage MR neurography in CIDP: diagnostic accuracy and electrophysiological correlation. J Neurol 2017;264(7):1434–43.

[13] Takahara T, Hendrikse J, Yamashita T, Mali WP, Kwee TC, Imai Y, et al. Diffusion-weighted MR neurography of the brachial plexus: feasibility study. Radiology 2008;249(2):653–60.

[14] Shen J, Zhou CP, Zhong XM, Guo RM, Griffith JF, Cheng LN, et al. MR neurography: T1 and T2 measurements in acute peripheral nerve traction injury in rabbits. Radiology 2010;254(3):729–38.

[15] Yoneyama M, Takahara T, Kwee TC, Nakamura M, Tabuchi T. Rapid high resolution MR neurography with a diffusion-weighted pre-pulse. Magn Reson Med Sci 2013;12(2):111–9.

[16] Karampinos DC, Melkus G, Shepherd TM, Banerjee S, Saritas EU, Shankaranarayanan A, et al. Diffusion tensor imaging and T2 relaxometry of bilateral lumbar nerve roots: feasibility of in-plane imaging. NMR Biomed 2013;26(6):630–7.

[17] Zhang X, Zhang F, Lu L, Li H, Wen X, Shen J. MR imaging and T2 measurements in peripheral nerve repair with activation of toll-like receptor 4 of neurotmesis. Eur Radiol 2014;24(5):1145–52.

[18] Kasper JM, Wadhwa V, Scott KM, Rozen S, Xi Y, Chhabra A. SHINKEI—a novel 3D isotropic MR neurography technique: technical advantages over 3DIRTSE-based imaging. Eur Radiol 2015;25(6):1672–7.

[19] Yoneyama M, Togao O, Hiwatashi A, Ozawa Y, Obara M, Okuaki T, et al. SHINKEI quant: simultaneous acquisition of MR neurography and T2 mapping for quantitative evaluation of chronic inflammatory demyelinating polyneuropathy. Proc Int Soc Magn Reson Med 2016:1322.

[20] Chhabra A, Carrino JA, Farahani SJ, Thawait GK, Sumner CJ, Wadhwa V, et al. Whole-body MR neurography: prospective feasibility study in polyneuropathy and Charcot-Marie-Tooth disease. J Magn Reson Imaging 2016;44(6):1513–21.

[21] Schwarz D, Kele H, Kronlage M, Godel T, Hilgenfeld T, Bendszus M, et al. Diagnostic value of magnetic resonance neurography in cervical radiculopathy: plexus patterns and peripheral nerve lesions. Investig Radiol 2018;53(3):158–66.

[22] Brown R, Cheng Y-CN, Haacke E, Thompson M, Venkatesan R. Spin Density, T 1, and T 2 Quantification Methods in MR Imaging. Magnetic Resonance Imaging: Physical Principles and Sequence Design. Second Edition John Wiley & Sons, Inc; 2014.

[23] DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. Biometrics 1988;44(3):837–45.

[24] De Silva RN, Willison HJ, Doyle D, Weir AI, Hadley DM, Thomas AM. Nerve root hypertrophy in chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 1994;17(2):168–70.

[25] Wang G, El-Sharkawy AM, Edelstein WA, Schar M, Bottomley PA. Measuring T(2) and T(1), and imaging T(2) without spin echoes. J Magn Reson (San Diego, Calif:1997) 2012;214(1):273–80.

[26] Weidlich D, Schlaeger S, Kooijman H, Bornert P, Kirschke JS, Rummeny EJ, et al. T2 mapping with magnetization-prepared 3D TSE based on a modified BIR-4 T2 preparation. NMR Biomed 2017;30(11).