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FULL PAPER

Lumbar plexus in patients with chronic inflammatory demyelinating polyradiculoneuropathy: evaluation with simultaneous T_2 mapping and neurography method with SHINKEI

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Objective: To evaluate the usefulness of simultaneous T_2 mapping and neurography with nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation enhancement imaging (SHINKEI) in the lumbar plexus to distinguish patients with chronic inflammatory demyelinating polyneuropathy (CIDP) from healthy controls.

Methods: Our institutional review boards approved this retrospective study, and written informed consent was waived. 10 patients with CIDP from 2015 to 2017 were studied along with 5 healthy controls on a 3 T scanner. The T_2 relaxation time and the size of the dorsal root ganglia and nerves of the lumbar plexus at L3-S1 were measured. Statistical analyses were performed with the Mann-Whitney U test and a receiver operating characteristics analysis.

Results: The T_2 relaxation times of the dorsal root ganglia and the nerves of the lumbar plexus were longer in the

CIDP patients (133.34 ± 41.36 and 130.40 ± 47.78 ms) compared to the healthy controls (114.69 ± 24.90 and 83.72 ± 17.51 ms, $p = 0.0265$ and $p < 0.0001$, respectively). The sizes of the nerves were larger in the CIDP patients (6.19 ± 2.28 mm) compared to the controls (4.54 ± 0.86 mm, $p < 0.0001$). However, there was no significant difference between the sizes of the ganglia in the CIDP patients and the controls. The receiver operating characteristics analysis revealed that the T_2 relaxation time of the nerves was best at distinguishing the CIDP patients from the controls ($A_z = 0.848$).

Conclusion: Patients with CIDP could be distinguished from healthy controls using simultaneous T_2 mapping and neurography with SHINKEI in the lumbar plexus.

Advances in knowledge: Patients with CIDP could be distinguished from healthy controls using simultaneous T_2 mapping and neurography with SHINKEI in the lumbar plexus.

INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a demyelinating disease of the peripheral nerves including both sensory and motor nerves.^{1–12} It sometimes involves the cauda equina³ and the lumbar plexus,^{4,6,7,12,13} which are more commonly involved than the brachial plexus.^{11,14}

MR neurography has been reported as clinically valuable to evaluate pathological conditions of the peripheral nerves.^{6–8,11,13–24} Usually, T_2 weighted images with fat suppression and T_1 weighted images are used to

evaluate diseases of the brachial plexus.¹⁶ Utility of diffusion-weighted imaging is also proposed in the lumbar plexus.¹³ The other study used diffusion-weighted whole-body imaging with background body signal suppression which revealed diffuse swelling of the lumbosacral plexus in a patient with CIDP.²⁵

With use of improved motion-sensitized driven equilibrium (iMSDE) and spectral attenuated inversion recovery sequence, three-dimensional nerve-sheath signal increased with inked rest-tissue rapid acquisition of relaxation imaging (SHINKEI)¹⁹ has been recently proposed which

sufficiently suppresses the signals of blood vessels, muscles, and fat tissue.^{6,7,11,19,21,22} With SHINKEI, we can obtain neurography with high spatial resolution and can evaluate the dorsal root ganglia and spinal nerves of the lumbar plexus; however, studies including a quantitative T_2 evaluation of this technique are lacking. Recently Yoneyama *et al* proposed an advanced sequence²² which can obtain MR neurography and T_2 mapping data simultaneously. However, to the best of our knowledge, there has been no report using this technique to evaluate patients with CIDP in the lumbar plexus. Therefore, we conducted the present study to evaluate the usefulness of simultaneous T_2 mapping and neurography with SHINKEI in the lumbar plexus to distinguish patients with CIDP from healthy controls.

MATERIALS AND

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

Patients

Simultaneous T_2 mapping and neurography with SHINKEI have been part of our routine MR protocol for imaging of the lumbar plexus. We analyzed the MRI data of 58 consecutive patients with CIDP who were identified between October 2015 and October 2017. The diagnosis of CIDP was made when they met the definite diagnostic criteria of the European Federation of Neurological Societies/Peripheral Nerve Society.¹ The exclusion criteria were as follows: (a) patients with diagnoses other than CIDP ($n = 31$), and (b) patients without specific diagnoses ($n = 17$). A total of 10 patients with CIDP (seven males, three females; age range 11–68 years; median age 47.5 years) were identified on the basis of these criteria. All but one patient had undergone previous treatment with intravenous immunoglobulin ($n = 9$), steroids ($n = 5$), plasmapheresis ($n = 4$), or another immunosuppressant ($n = 3$). The patient demographics are summarized in Table 1. Five healthy controls (four males, one female; age range 27–45 years; median age 41 years) were also studied. These cases were collected from our database who had been imaged without neurological abnormality.

Imaging technique

All of the patients and healthy controls underwent MRI with a 3.0 T system (Ingenia; Philips, Best, Netherlands) with a dStream TotalSpine coil. Simultaneous T_2 mapping and neurography with SHINKEI were obtained in the coronal plane as part of the lumbar plexus MR neurography protocol. The details of SHINKEI have been described elsewhere.^{6,7,11,19,21} Briefly, it is a turbo spin echo with a diffusion-weighted prepulse called "iMSDE" to suppress signals from vessels and muscles and a spectral adiabatic inversion recovery (SPAIR) 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 T_2 weighting.¹⁹ SHINKEI parameters included TR/TE = 2500/51 ms, FOV = 290×290 mm, ETL = 90, acquisition matrix = 256×245, reconstructed voxel size = 0.60×0.60 × 2.0 mm³, $b = 10$ s/mm², iMSDE duration = 36 and 72 ms, acquisition time = 5 min 45 s. These images were obtained in a coronal plane. MR neurography was obtained with both iMSDE preparation times as follows:

$$S_{\text{neurography}} = S_{\text{preptime-1}} + S_{\text{preptime}}$$

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

T_2 map was calculated as follows:

$$S_{\text{preptime}} = S_0 * e^{(\text{preptime}/T_2)}$$

A T_2 map was calculated by pixel-by-pixel fitting of the magnitude image intensities from both prep-times to a mono-exponential relaxation model.²⁶

Analysis

All MR images were saved as DICOM and viewed using Osirix software ver. 6.5.2 (Pixmeo, Geneva, Switzerland). Region of

Table 1 Patient demographics

No.	Age	Sex	Previous treatment	CSF protein	Dominant extremities	Dominant symptoms	Duration (year)
1	34	F	IVIG, PP, IS	68	L	M > S	0.5
2	52	M	IVIG	59	U	M	5
3	31	M	IVIG, steroids, PP, IS	40	U = L	M > S	20
4	64	M	IVIG, steroids, IS	30	L	M > S	9
5	43	M	IVIG	92	L	S > M	0.5
6	80	M	None	96	L	M > S	9
7	11	F	IVIG, steroids, PP	110	L	M > S	1
8	57	F	IVIG, IS	36	U = L	M	3
9	35	M	IVIG, steroids, PP, IS	228	L	S > M	4
10	53	M	IVIG, steroids	300	L	M > S	6

IVIG, intravenous immunoglobulin; PP, plasmapheresis; IS, immunosuppressant; L, lower; U, Upper; M, motor; S, sensory.

Figure 1. The T_2 relaxation times of the dorsal root ganglia (a) and nerves (b) are significantly longer in the patients with CIDP (133.34 ± 41.36 and 130.40 ± 47.78 ms) compared to the healthy controls (114.69 ± 24.90 and 83.72 ± 17.51 ms, $p = 0.0265$ and $p < 0.0001$, respectively). CIDP, chronic inflammatory demyelinating polyneuropathy.

interests (ROIs) were placed at the dorsal root ganglia and nerves of the lumbar plexus at L3–S1 by a neuroradiologist (AH, 20 year-experience in neuroradiology). The T_2 relaxation times and the sizes of the dorsal root ganglia and spinal nerves were measured. Circular ROIs not to include the outside tissue on neurography were placed within the bilateral dorsal root ganglia or nerves at L3–S1 and copied to T_2 map to measure signal intensity. Linear ROIs were placed in the same structures on neurography to measure the sizes. A reviewer was blind to the diagnosis at the time of measurement. Statistical analyses were performed by the same author using JMP software (v. 13.0.0; SAS Institute, Cary, NC) and MedCalc Software (v. 17.9.7; MedCalc, Mariakerke, Belgium). Techniques used included the Mann–Whitney U test, and the receiver operating characteristics analysis.²⁷ p -values < 0.05 were considered significant. The Bonferroni correction was used for multiple comparisons.

RESULTS

The T_2 relaxation times of the dorsal root ganglia and the nerves of the lumbar plexus were significantly longer in the patients with CIDP (133.34 ± 41.36 and 130.40 ± 47.78 ms) than in the healthy controls (114.69 ± 24.90 and 83.72 ± 17.51 ms, $p = 0.0265$ and $p < 0.0001$, respectively; Figure 1). The sizes of the nerves were significantly larger in the CIDP patients (6.19 ± 2.28 mm) than in the controls (4.54 ± 0.86 mm, $p < 0.0001$; Figure 2). The dorsal root ganglia were also larger in the CIDP patients (6.62 ± 1.74 mm) compared to the controls (6.29 ± 0.84 mm), however, but the differences were not significant ($p = 0.4325$).

Figure 2. The sizes of the dorsal root ganglia (a) are larger in the CIDP patients (6.62 ± 1.74 mm) than in the controls (6.29 ± 0.84 mm), but not significantly so ($p = 0.4325$). The sizes of the nerves (b) are significantly larger in the CIDP group (6.19 ± 2.28 mm) compared to the controls (4.54 ± 0.86 mm, $p < 0.0001$). CIDP, chronic inflammatory demyelinating polyneuropathy.

The receiver operating characteristics analysis revealed the best diagnostic performance using T_2 relaxation time of the nerves (are under the curve = 0.848). The sensitivity, the specificity, and the AUC were 0.55, 0.73 and 0.63 for the T_2 relaxation time, and 0.31, 0.93, and 0.54 for the size of the ganglia, respectively (Figure 3). These were 0.71, 0.85, and 0.85 for the T_2 relaxation time, and 0.71, 0.75, and 0.77 for the size of the nerves, respectively. The AUC was larger for the T_2 relaxation time of the nerves. There were significant differences between the AUC of the T_2 relaxation time of the nerves and the AUC of the T_2 relaxation times ($p < 0.0001$) and the sizes ($p < 0.0001$) of the dorsal root ganglia. However, there was no significant difference between the AUC of the T_2 relaxation time and that of the sizes

Figure 3. The ROC analysis reveals the best diagnostic performance with the T_2 relaxation time of the nerves (AUC = 0.848). AUC, area under the curve; ROC, receiver operating characteristics.

Figure 4. A 45-year-old male control subject. MR neurography (a) visualizes the dorsal root ganglia (*arrowheads*) and the spinal nerves of L5 (*arrows*). The T_2 relaxation times are 114.43 ± 19.11 ms in the dorsal root ganglia and 86.67 ± 12.12 ms in the nerves (b).

of the nerves ($p = 0.1436$). 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 [Figures 4 and 5](#).

DISCUSSION

Our findings demonstrated the utility of the new MR neurography technique that enables us to obtain high-resolution MR neurography and T_2 map simultaneously. Using our simultaneous T_2 mapping and neurography with SHINKEI, we observed that the T_2 relaxation times of the dorsal root ganglia and the nerves of the lumbar plexus were longer in the patients with CIDP than in healthy controls. Although the increased signal intensity on T_2 weighted images in CIDP patients had already been identified with an older method, we propose that our technique is beneficial because of the high spatial resolution and its T_2 quantification. Simultaneous T_2 mapping and neurography with SHINKEI are also unique in that it provides sufficient suppression of the signals of the fat, vessels and muscles using two types of prepulses (spectral adiabatic inversion recovery and iMSDE) in the lumbar plexus.

This is the first report describing a simultaneous quantitative evaluation of the T_2 values and the sizes of the lumbar plexus in patients with CIDP and healthy controls. Among the sizes and T_2 values of the dorsal root ganglia and nerves, we found that the T_2 relaxation time of the nerves in the lumbar plexus was the best to differentiate CIDP patients from healthy controls. This finding was in line with an earlier study evaluating the lumbar plexus using SHINKEI, which showed the contrast-to-noise ratio as the

Figure 5. A 35-year-old male with CIDP. MR neurography (a) visualizes the enlarged dorsal root ganglia (*arrowheads*) and the spinal nerves of L5 (*arrows*). The T_2 relaxation times are 208.37 ± 28.09 ms in the dorsal root ganglia and 216.92 ± 54.22 ms in the spinal nerves (b). CIDP, chronic inflammatory demyelinating polyneuropathy.

best diagnostic marker.⁷ In addition, our present findings are basically the same as those of our unpublished data indicating that the T_2 relaxation time of the roots of the brachial plexus showed the best diagnostic performance with simultaneous T_2 mapping and neurography with SHINKEI to discriminate CIDP patients from healthy controls.

Qualitative studies have shown increased signal intensity on T_2 weighted images in the brachial plexus or other nerve trunks in patients with CIDP.^{4,7} Several quantitative studies have evaluated T_2 values in the normal peripheral nerves in animals. Zhang et al reported that the T_2 relaxation times of the nerves in rats were approx. 50 ms,²⁰ and Shen et al reported that the T_2 relaxation times of the nerves in rabbits were approx. 40 ms.¹⁷ Karampinos et al evaluated L4 nerves in humans and reported that the T_2 relaxation times were significantly longer in the dorsal root ganglia (78.0 ± 11.9 ms) than in more distal regions (59.5 ± 7.4 ms).¹⁸ Sollmann et al also evaluated the L4, L5 and S1 nerves in healthy humans and reported that the T_2 relaxation times were 74.6 ± 4.7 ms for preganglionic, 84.7 ± 4.1 ms for ganglionic, and 65.4 ± 2.5 ms for post-ganglionic segments, respectively.²⁴ In the present study, we observed longer T_2 relaxation times of the ganglia (114.69 ± 24.90 ms) than those of the nerves in the controls (83.72 ± 17.51 ms), which agrees these studies.^{18,24} We think the presence of gray matter in the dorsal root ganglia could cause longer T_2 values than in nerves.

However, our observed values were longer than those in the previous studies.^{17,18,20,24} There might be several reasons for these findings. First, simultaneous T_2 mapping and neurography

with SHINKEI calculated the T_2 relaxation time by only two prep-times, which may cause unstable T_2 assessments. T_2 quantification with three or more points might be better, which needs longer acquisition time. In addition, because each prep-time is acquired separately, there might be contamination from adjacent tissues. The inversion recovery technique used to suppress fat signals in SHINKEI might alter the quantification of T_2 relaxation times with a contamination of T_1 effect. However, we note the clinical utility of our technique, which obtains high-resolution MR neurography and T_2 mapping in the lumbar plexus in approx. 6 min. Contrary to our results, 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 T_2 relaxation time.⁸ Further evaluations of our technique in the extremities are thus necessary.

We observed that the sizes of the nerves were significantly larger in the CIDP patients compared to the healthy controls. The same tendency was observed at the dorsal root ganglia, but the difference was not significant. The qualitative assessment of nerve hypertrophy by MR has also been reported.³ Symonds et al described nerve root enlargement on myelography in patients with CIDP.²⁸ Midoroni et al also reported that the nerve roots in the lumbar spine were enlarged, most significantly in the extraforaminal region.³ Matsuda et al stated that the nerve hypertrophy in CIDP was attributable mainly to the proliferation of Schwann cells caused by repeated demyelination and remyelination.²

The mucopolysaccharides in the endometrium as secondary products associated with nerve degeneration²⁹ and edema³⁰ may also contribute to nerve swelling in CIDP.³¹ Bradley et al reported that proximal onion bulb hypertrophic changes in the brachial plexus were associated with inflammatory cell infiltrates.³² These might explain the larger sizes of the nerves in individuals with CIDP compared to healthy controls. Duggins et al reported that patients with hypertrophy had a relapsing-remitting course and a significantly longer disease duration.⁴ Ishikawa et al also reported that the volume of the peripheral nerves was positively correlated

with disease duration.¹² In contrast, Midroni et al observed that there was no obvious relationship between symptom duration and the presence or extent of nerve root enlargement.³ Further research is necessary to test these findings.

With another quantitative MRI technique, Adachi et al evaluated the brachial and lumbar plexuses and reported that the diffusivity in CIDP patients with nerve swelling or abnormal signals showed higher diffusivity compared to CIDP patients without these findings and healthy controls.¹³ 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.⁵ We have not used diffusion-weighted or tensor imaging. We might need further study to compare our current technique with diffusion-weighted imaging.

There are some limitations to this study. We could not evaluate the entire length of the lumbar plexus because of its complex form. The lack of contrast material might be another limitation. We do not routinely use contrast in CIDP patients with clinically stable symptoms. The small number of patients included should be a limitation. We are continuing to gather patient's data. Nerve size in CIDP is known to vary with disease activity and treatment response on ultrasound.⁹ We are continuing to gather patient data.

In conclusion, with simultaneous T_2 mapping and neurography with SHINKEI we were able to obtain high-resolution MR neurography with T_2 mapping simultaneously in the lumbar plexus. Patients with CIDP could be distinguished from healthy controls using simultaneous T_2 mapping and neurography with SHINKEI in the lumbar plexus.

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