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Brief Communication

Utility of Sensory Nerve Conduction Study in Radiologically Positive Lumbosacral Plexopathy

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ABSTRACT: MRI is the most appropriate imaging method for visual evaluation of lumbosacral plexopathy (LSP) and a reference for comparing with nerve conduction study (NCS). Eight patients with clinical, electrophysiological, and lumbosacral plexus MRI findings suggestive of LSP were prospectively recruited. Saphenous nerve abnormalities were present in seven patients (88%), compared to three for the superficial fibular (38%), and three for the sural nerve (38%). MRI showed tumor, hematoma, abscess, contrast enhancement, or hyperintense signals on the T2-weighted sequences. The SN has the highest yield in MRI positive LSP and may be a vital adjunct for electrophysiological evaluation of LSP.

RÉSUMÉ : Utilité d'une étude de la conduction nerveuse sensorielle dans le cas d'une plexopathie lombosacrée détectée par IRM. L'imagerie par résonnance magnétique (IRM) est la méthode d'imagerie la plus appropriée pour l'évaluation visuelle de la plexopathie lombosacrée (PLS). Elle constitue aussi une référence en vue d'une comparaison avec les résultats d'une étude de la conduction nerveuse (ECN). Au total, ce sont 8 patients présentant des indices cliniques et électrophysiologiques d'une PLS, en plus d'indices évocateurs au niveau du plexus lombo-sacré détectés par IRM, qui ont été recrutés prospectivement. Des anomalies du nerf saphène (NS) étaient présentes chez 7 patients (88 %) alors que 3 d'entre eux ont montré des anomalies affectant le nerf fibulaire superficiel (38 %) et 3 autres patients des anomalies affectant le nerf sural (38 %). Des examens d'IRM ont par ailleurs montré une tumeur, un hématome, un abcès, un rehaussement de contraste ou des hypersignaux en ce qui concerne des séquences pondérées en T2. Les anomalies du NS ont eu le rendement le plus élevé en ce qui regarde la PLS détectée par IRM et peuvent représenter un complément indispensable pour l'évaluation électrophysiologique des cas de PLS.

Keywords: Lumbosacral plexopathy; Nerve conduction study; MRI; Diagnosis

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The lumbosacral plexus, comprising both lumbar (L2 to L4 roots) and sacral plexi (S1 to S4 roots), may be affected by multiple etiologies. Definitive localization of lumbosacral plexopathy (LSP) is often challenging and relies on combined clinical, radiological, and electrophysiological findings.[1]

Criteria to define the presence of LSP each had inherent pitfalls and required interpretation in relation to other findings. Absence of denervation of the paraspinal electromyography (EMG) and the presence of denervation of the glutei muscles would help localize the lesion to the LSP. Conversely, EMG findings of paraspinal fibrillations may also be present in patients with diabetes mellitus or previous lumbar surgery. Paraspinal fibrillations may be absent due to sampling error or early reinnervation. Hence, some investigators favor demonstrating absent or reduced sensory nerve action potential (SNAP) amplitudes of the sural or fibular nerves to suggest the presence of a postganglionic lesion outside the lumbosacral intraspinal canal. However, it is also known that variability in position of the dorsal root ganglion of lower lumbar sensory roots relative to the site of lesion, as well as conditions presenting as radiculoplexopathies, may be potential confounders. Up to 40% of L5 dorsal root ganglia may have an intraspinal location, as a potential confounder for the preservation of superficial fibular sensory responses in L5 radiculopathy.[2]

MRI is deemed the most appropriate imaging modality,[3] and a retrospective series comprising 137 patients with sciatica-like symptoms showed that MRI of the lumbosacral plexus had detected abnormalities in all.[4] It can thus be of value as a reference for comparing with other methods including nerve conduction study (NCS), which, to our knowledge, has not been investigated. We determine the yield of NCS of the saphenous (SN), superficial fibular (SFN), and sural nerves in relation to the presence of LSP visualized with MRI.

Each of the eight recruited patients over a 5-year period had 1) hip or back pain, unilateral leg weakness, and ipsilateral sensory abnormalities involving multiple dermatomes, 2) MRI evidence of LSP, 3) EMG evidence of denervation of glutei and lumbosacral plexus innervated muscles, 4) absence of denervation changes in the lumbar paraspinal EMG, and 5) no other findings to suggest

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Table 1: Summary of sensory nerve conduction study amplitudes and clinical features of patients

Patient	Age	Sex	L SN	R SN	L SFN	R SFN	L Sural	R Sural	Side	MRI findings; etiology
1	35	F	**1.93**	4.2	26.5	28.0	20.7	24.1	L	Cauda and plexus enhancement, post ankle fracture
2	69	F	4.1	5.5	17.6	15.4	21	20	R	Cauda and plexus enhancement; post fall
3	67	F	3.1	**0**	18.3	18.8	25.6	24.1	R	Hyperintense T2 plexus and muscle signal changes; idiopathic
4	74	M	2.8	**1.2**	3.4	**0**	5.2	5.2	R	Root (L3) and plexus tumor; schwanoma
5	48	M	5.1	**0**	4.5	**0**	5.7	**0**	R	Plexus enhancement; traumatic plexopathy
6	68	F	2.8	**1.6**	2.8	**1.59**	8.5	2.4	R	Hyperintense plexus T2 changes; diabetic plexopathy
7	33	M	**0**	2.1	8.4	8.9	15.8	11.1	L	Plexus hematoma; myeloid leukemia
8	28	M	**0.9**	2.1	9.1	8.6	8.8	4.3	L	Plexus microabcess; intravenous drug abuse

M: male; F: female; L: left; R: right; 0: absent; all amplitude values in μV; Bold values indicate abnormality; Side: side of initial complaint; Underlined values: abnormality side-to-side amplitude ratio; SN: saphenous nerve; SFN; superficial fibular nerve.
Normal SN, SFN, and sural nerve side to side amplitude ratios >0.5; SN, SPN, and sural nerve normal amplitude: >2 (local laboratory control values).

alternate diagnosis than LSP. Electrophysiological studies were performed at least 2 months after the onset of symptoms.

NCS of the SN[5] was performed with active adhesive recording electrodes placed anterior to the medial malleolus of the ankle, in the space between the malleolus and medial border of tibialis anterior tendon. A stimulating cathode was placed 10 cm proximal to the active recording electrode along the tibial bone medial border. The subject lay comfortably sideways with knees bent at 45°.

Ten averages were made to obtain an optimal SNAP, with stimulation intensity below 20 mA. All NCS were performed with a Dantec Keypoint (Natus, New York, USA) EMG machine. Onset latency and peak to peak SNAP amplitudes were measured; amplifier filter bands were 20 to 2 kHz, and surface skin temperature at or above 33° C. Limits of normality were 2 standard deviations (SD) below the mean value for amplitude or side-to-side amplitude ratio if normally distributed, or the 5th percentile if non-normally distributed. SFN and sural reference ranges were based on our laboratory control values.

Yields of NCS of the three sensory nerves were determined with MRI as the reference standard.

Comparison between 50 healthy controls and patients did not reveal any significant difference in terms of age (controls: 26 to 75, patients: 28 to 74, *t*-test, $p = 0.23$). For controls, mean amplitude of the SN (SD) was 3.55 (1.60) μV. The lower limit of normality at the 5th percentile was 2.1 μV as the data were not normally distributed. Mean latency (SD) was 2.49 (0.34) ms. Upper limit of normality at 2 SDs above the mean was 3.13 ms. Mean side-to-side amplitude ratio (SD) was 0.86 (0.2). Upper limit of normality at the 5th percentile was 0.7. Amplitude values of the SN in controls were comparable to previously published results.[5]

The patients comprised four men and four women (age: 28 to 74). Abnormalities of the SN were present in seven patients (88%), compared to three patients for the SPN (38%), and three patients for the sural nerve (38%). In three patients, only SN abnormalities were present in the NCS. None of the patients had abnormal SFN or sural NCS without abnormality of the SN NCS. Patients 6 and 8 showed abnormal side-to-side amplitude ratio of the sural nerves, in addition to absolute amplitude reduction of the SN (Table 1).

MRI showed space-occupying lesions (tumor, hematoma, abscess), contrast enhancement or hyperintense signals on the T2-weighted sequences. In two patients (Patients 1 and 2), cauda equina enhancement was demonstrated, suggesting radiculoplexopathy. In addition to the plexus, Patient 3 had T2 hyperintense signals in the glutei muscles.

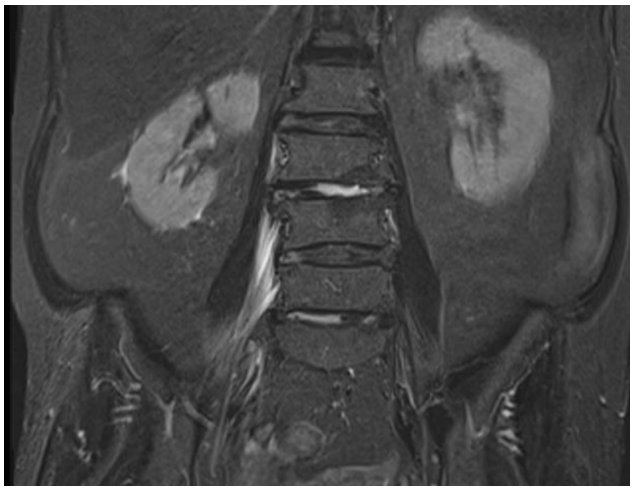

Figure 1: Abnormal T2 hyperintense signal of the right lumbosacral plexus predominantly at the L4 and L5 levels in a patient with diabetic LSP.

Figure 1 shows T2 hyperintense signal of the right lumbosacral plexus predominantly at the L4 and L5 levels in a patient with diabetic LSP.

Our findings suggest that the SN has a markedly higher yield in the electrophysiological evaluation of MRI positive LSP, compared to the SFN and sural nerves.

The SN (L3, L4) carry large fiber sensory afferents of variable proportions toward the femoral trunk. However, there may be contributions from adjacent segmental supply resulting in a larger dermatomal representation for the SN and sural nerves.[6] It may be difficult to correlate our patients' MRI with NCS findings in terms of dermatomal levels of involvement. Nonetheless, sensory NCS, particularly that of the SN, shows higher likelihood of abnormality when evaluating LSP regardless of etiology. While the exact reasons are unclear, it is possible that the SFN and sural NCS assess largely L5 levels and below,[6] which were less involved in our patient cohort.

Potential limitations exist, particularly regarding the yield of the SN NCS in radiculopathy which was not evaluated. However, a large study comprising 108 patients with lumbosacral radiculopathy from herniated disc found abnormalities of the superficial

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fibular nerve (12.1%) and sural nerve (2.4%), but none involving the SN, suggesting that the latter is not involved in the presence of root lesions.[7] These aspects can be explored further with the advances in MRI and MR neurography.[8]

In conclusion, our study supports the finding that NCS of the SN is a most useful adjunct for the electrophysiological evaluation of LSP.

Statement of authorship. YLL: writing, data collection.
YET: data collection, analysis.
RH: data collection, analysis.
PPCT: data collection, analysis.

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