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Asymmetric brachial plexus MRI abnormalities in Lewis-Sumner syndrome

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AUTHOR AND AFFILIATION

Salma Tabati[1], Leila Tamaoui[2,3], Nazha Birouk[2,3], Nada Adjou[1], Hamza Retal[1,2], Meriem Fikri[1,2], Firdaous Touarsa[1,2]

[1] Department of Neuroradiology, Specialty Hospital of Rabat, Rabat, Morocco
[2] Faculty of Medicine and Pharmacy, Mohammed V University in Rabat, Rabat, Morocco
[3] Department of Clinical Neurophysiology, Specialty Hospital of Rabat, Rabat, Morocco

Corresponding author: Salma Tabati

ABSTRACT

Lewis-Sumner syndrome is an uncommon asymmetric inflammatory demyelinating neuropathy. A 20-year-old patient presented with multifocal upper-limb paraesthesias, finger-extensor weakness and generalised areflexia. Electrophysiology showed proximal motor conduction blocks despite normal distal sensory studies. MRI demonstrated bilateral asymmetric C5-T1 root and brachial plexus enlargement, STIR hyperintensity and enhancement, with intercostal nerve involvement. Intravenous immunoglobulin was initiated. Contribution: MRI supported the diagnosis and revealed clinically occult disease extension.

KEYWORDS

Lewis-Sumner syndrome; multifocal CIDP; brachial plexus; magnetic resonance neurography; nerve root hypertrophy; conduction block; STIR-SPACE; multifocal motor neuropathy

MAIN ARTICLE

INTRODUCTION

Lewis-Sumner syndrome (LSS), also termed multifocal acquired demyelinating sensory and motor neuropathy, is an asymmetric immune-mediated neuropathy within the multifocal chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) spectrum. It usually affects the upper limbs and combines multifocal weakness, sensory symptoms, conduction block and reduced reflexes.[1-4] Magnetic resonance imaging (MRI) is supportive rather than diagnostic, but may demonstrate enlargement, T2/STIR hyperintensity and enhancement of cervical roots and the brachial plexus.[5-9] This report highlights extensive, asymmetric MRI abnormalities and the diagnostic pitfall created by normal conventional sensory nerve conduction studies.

ETHICAL CONSIDERATIONS

Institutional review board approval was waived for this single case report.

The case was managed in accordance with the Declaration of Helsinki.

Written informed consent for publication of the clinical information and images was obtained from the patient.

PATIENT PRESENTATION

A previously healthy 20-year-old patient presented with a 2-month history of progressive paraesthesias in both upper limbs. Symptoms involved the right ulnar territory and the left ulnar, radial and median territories, with difficulty performing fine hand movements. Examination demonstrated bilateral finger-extensor weakness and absent deep tendon reflexes in all four limbs. No relevant neurological, systemic, traumatic, infectious or neoplastic history was reported. The asymmetric involvement of several nerve territories and generalised areflexia suggested an acquired inflammatory demyelinating neuropathy.

Motor nerve conduction studies showed preserved distal median and ulnar compound muscle action potential amplitudes, but multifocal proximal conduction blocks, abnormal temporal dispersion and prolonged minimal F-wave latencies (Table 1; Figure 4). Conventional median, ulnar, sural and superficial peroneal sensory responses were within laboratory reference ranges. Somatosensory evoked potentials after median and ulnar stimulation

showed delayed proximal responses, supporting proximal sensory pathway involvement that was not detected by distal sensory recordings.[10]

Contrast-enhanced brachial plexus MRI demonstrated bilateral multifocal thickening of the C5-T1 roots, most pronounced at right C6 and left C7. The involved roots were hyperintense on fluid-sensitive sequences, including three-dimensional STIR-SPACE, and enhanced after gadolinium (Figures 1 and 3). Abnormalities extended through the trunks, divisions and cords. Distal involvement predominated in the right ulnar nerve and in the left ulnar, radial and median nerves, concordant with the clinical distribution. Asymmetric enlargement also involved the left second and right fourth intercostal nerves, indicating disease beyond the clinically dominant territories. T1- and T2-weighted imaging showed no proximal muscle oedema, atrophy or fatty replacement (Figure 2).

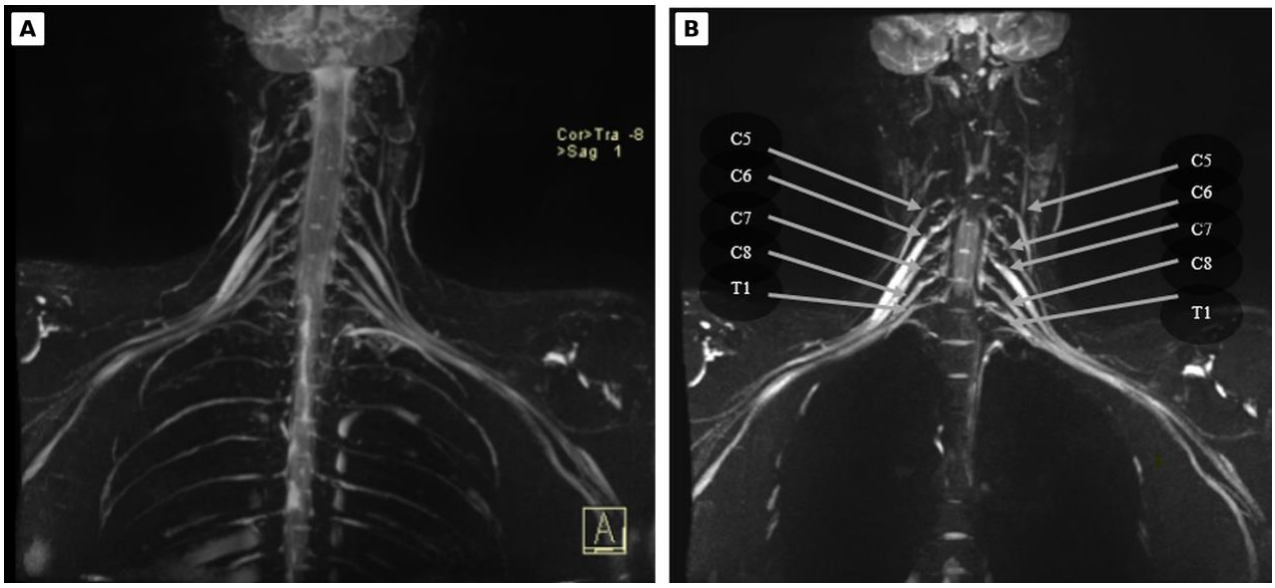

Figure 1. Coronal three-dimensional STIR-SPACE MR neurography of the brachial plexuses.

(A) Maximum-intensity or multiplanar reformatted overview demonstrates bilateral neural enlargement and hyperintensity.

(B) Annotated image identifies the C5-T1 roots; abnormalities were asymmetric, with predominant enlargement of the right C6 and left C7 roots.

Source: Authors' own clinical images; 2026.

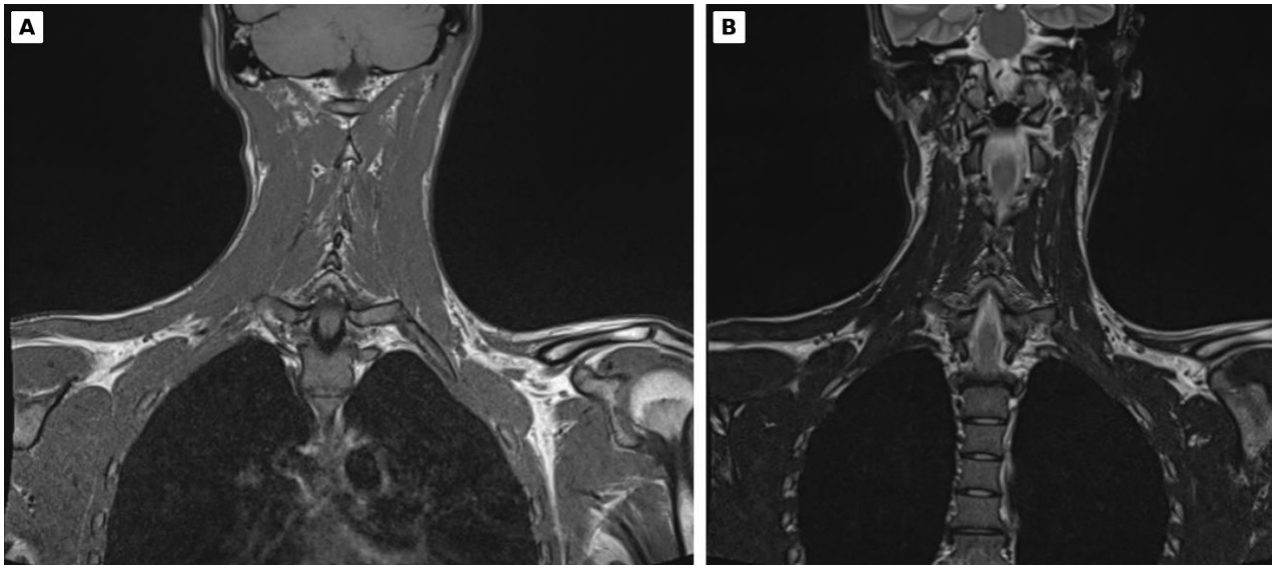

Figure 2. Coronal MRI assessing muscle denervation.

(A) T1-weighted and (B) T2-weighted images show no oedema, volume loss or fatty replacement in the examined shoulder-girdle and proximal upper-limb musculature.

Source: Authors' own clinical images; 2026.

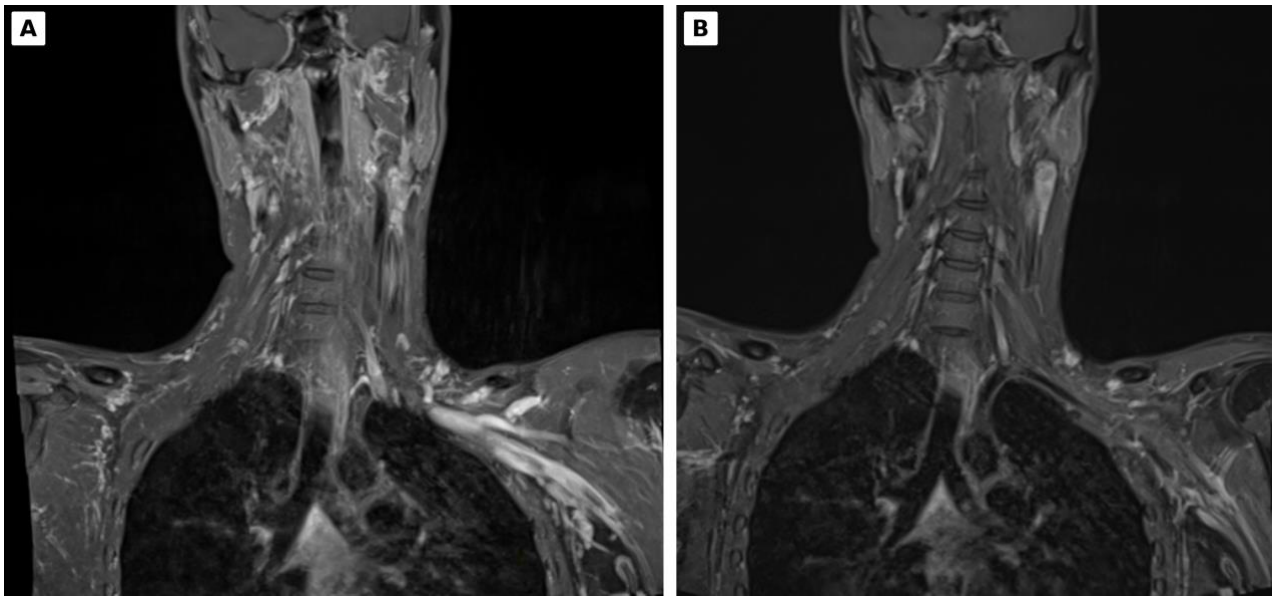

Figure 3. Coronal post-gadolinium T1-weighted fat-suppressed images demonstrate enhancement of the bilateral cervical nerve roots and brachial plexus components, with asymmetric severity.

Source: Authors' own clinical images; 2026.

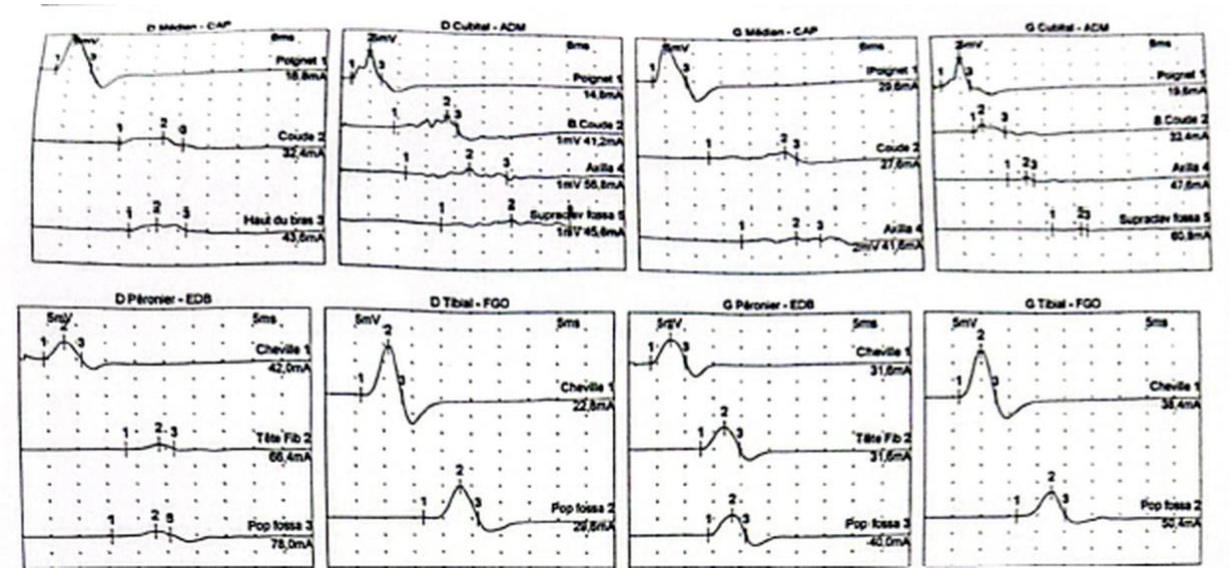

Figure 4. Motor nerve conduction traces demonstrate multifocal conduction block and abnormal temporal dispersion in the median and ulnar nerves across proximal segments.

Source: Authors' own electrophysiological recordings; 2026.

Table 1: Summary of electrophysiological findings

Study	Distal response	Proximal finding	Interpretation
Median motor, right/left	Wrist CMAP 14.0/12.8 mV	Marked proximal amplitude reduction, temporal dispersion; F-wave latency 41/78 ms	Multifocal proximal demyelination with limited distal axonal loss
Ulnar motor, right/left	Wrist CMAP 9.8/9.7 mV	Marked proximal amplitude reduction, temporal dispersion; F-wave latency 54/91 ms	Multifocal proximal demyelination
Median, ulnar, sural and superficial peroneal sensory studies	Amplitudes, latencies and velocities within laboratory reference ranges	No significant side-to-side asymmetry	Normal distal sensory recordings

Study	Distal response	Proximal finding	Interpretation
Somatosensory evoked potentials	Distal sensory responses preserved	Delayed proximal responses after median and ulnar stimulation	Supportive of proximal sensory pathway involvement

CMAP, compound muscle action potential. Confirm the exact stimulation segments because the source manuscript alternates between elbow-level and non-entrapment proximal sites.

MANAGEMENT AND OUTCOME

The patient received intravenous immunoglobulin at an induction dose of 2 g/kg over 5 consecutive days, followed by maintenance treatment of 1 g/kg every 3 weeks. Physiotherapy and occupational therapy focused on upper-limb strengthening and fine motor function.

During clinical follow-up, the patient showed a favourable clinical response, with clear improvement in the presenting neurological symptoms. No repeat electrophysiological examination or follow-up MRI was performed.

DISCUSSION

The combined clinical, electrophysiological and MRI pattern favoured LSS. The principal diagnostic challenge was the normality of conventional sensory conduction studies, which may suggest multifocal motor neuropathy (MMN) when asymmetric distal weakness and motor conduction blocks coexist. However, the patient's sensory complaints, multiterritorial sensory distribution and abnormal proximal somatosensory evoked potentials supported sensory involvement and therefore LSS rather than a purely motor neuropathy.[10,11]

MRI can support the diagnosis when disease is proximal, electrodiagnostic criteria are incomplete or an infiltrative or compressive plexopathy must be excluded.[5-9] Three-dimensional STIR-SPACE is useful because of its spatial resolution, homogeneous fat suppression and multiplanar capability, allowing continuous assessment from the roots to terminal branches. In this patient, MRI depicted bilateral but markedly asymmetric root and plexus abnormalities, multifocal distal extension and intercostal nerve involvement. The latter may represent clinically silent disease and could be overlooked on a narrowly focused plexus examination.

These findings are not specific. Typical CIDP is usually more symmetric; MMN lacks objective sensory involvement; neuralgic amyotrophy commonly presents with acute severe

pain and denervation oedema; vasculitic neuropathy is predominantly axonal; hereditary neuropathy with liability to pressure palsies favours recurrent pressure-related lesions at entrapment sites; and neoplastic or compressive plexopathy requires a mass or structural cause (Table 2). Nerve enlargement and enhancement should therefore be interpreted with the clinical time course and electrophysiology. Qualitative plexus MRI is also susceptible to physiological asymmetry, vascular mimics, motion, incomplete fat suppression and coil-related signal variation.[7-9]

The absence of muscular denervation despite extensive neural abnormalities suggested limited axonal injury at the time of imaging, consistent with preserved distal motor amplitudes. Nevertheless, the lack of objective follow-up data in the source manuscript limits assessment of treatment response and radiological reversibility.

Table 2: Principal differential diagnoses

Differential diagnosis	Overlapping features	Features favouring Lewis-Sumner syndrome
Multifocal motor neuropathy	Asymmetric weakness and multifocal motor conduction blocks	Sensory symptoms, multiterritorial sensory involvement and abnormal proximal sensory evoked potentials
Typical CIDP	Areflexia, acquired demyelination and root/plexus enlargement	Markedly multifocal and asymmetric distribution
Neuralgic amyotrophy	Patchy asymmetric brachial plexus abnormalities	No acute severe pain and no muscular denervation on MRI
Vasculitic neuropathy	Multifocal asymmetric sensorimotor deficits	Electrophysiology showed demyelinating conduction block rather than an axonal pattern
Hereditary neuropathy with liability to pressure palsies	Young age and focal conduction abnormalities	No recurrent pressure-induced palsies or restriction to entrapment sites

Differential diagnosis	Overlapping features	Features favouring Lewis-Sumner syndrome
Neoplastic or compressive plexopathy	Nerve enlargement and enhancement	No mass, osseous lesion or compressive abnormality

CONCLUSION

LSS should be considered in an asymmetric multifocal neuropathy combining weakness, sensory symptoms, conduction blocks and areflexia. Brachial plexus MRI may reveal bilateral but asymmetric root and plexus enlargement, STIR hyperintensity and enhancement, while identifying clinically occult involvement such as intercostal nerve abnormalities. MRI is supportive and must be correlated with electrophysiology, particularly when distal sensory studies are normal.

FIGURE LEGENDS

Figure 1. Coronal three-dimensional STIR-SPACE MR neurography of the brachial plexuses. (A) Maximum-intensity or multiplanar reformatted overview demonstrates bilateral neural enlargement and hyperintensity. (B) Annotated image identifies the C5-T1 roots; abnormalities were asymmetric, with predominant enlargement of the right C6 and left C7 roots. Source: Authors' own clinical images; year of acquisition to be confirmed.

Figure 2. Coronal MRI assessing muscle denervation. (A) T1-weighted and (B) T2-weighted images show no oedema, volume loss or fatty replacement in the examined shoulder-girdle and proximal upper-limb musculature. Source: Authors' own clinical images; year of acquisition to be confirmed.

Figure 3. Coronal post-gadolinium T1-weighted fat-suppressed images demonstrate enhancement of the bilateral cervical nerve roots and brachial plexus components, with asymmetric severity. Source: Authors' own clinical images; year of acquisition to be confirmed.

Figure 4. Motor nerve conduction traces demonstrate multifocal conduction block and abnormal temporal dispersion in the median and ulnar nerves across proximal segments. Source: Authors' own electrophysiological recordings; year of acquisition to be confirmed.

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Competing interests

The authors declare that they have no financial or personal relationships that may have inappropriately influenced them in writing this article.

CRediT authorship contribution

Author contributions blinded for review; complete the CRediT roles in the full manuscript and official aosis form.

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Data availability statement

The data supporting the findings of this case report are available from the corresponding author on reasonable request, subject to patient confidentiality.

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