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IMAGES IN CLINICAL PRACTICE

Hypertrophic Lumbosacral Nerve Roots in a 19-Year-Old Woman with Chronic Inflammatory Demyelinating Polyneuropathy

Muna Abdul Ghani Zghair [1], Wassan Nori [2,*], Ahmed Atia [3]

[1]*Department of Radiology, College of Medicine, Mustansiriyah University, Baghdad, Iraq*
[2]*Department of Obstetrics and Gynecology, College of Medicine, Mustansiriyah University, Baghdad, Iraq*
[3]*Department of Anesthesia and Intensive Care, Faculty of Medical Technology, The University of Tripoli, Tripoli, Libya*

[*]*Corresponding author email: dr.wassan76@uomustansiriyah.edu.iq*

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A nineteen-year-old woman presented with a three-month history of progressive symmetrical limb weakness and sensory deficit. There was no history of trauma, systemic illness, or recent infection. Neurological evaluation confirmed global areflexia and gait impairment. Nerve conduction studies showed demyelinating polyneuropathy.

Magnetic resonance imaging was performed using a Philips 1.5-Tesla system with a lumbar spine coil, with multiple sequences including sagittal T2-weighted and T2-SPIR images to maximize diagnostic yield. The patient declined intravenous contrast administration; consequently, contrast-enhanced images were not obtained, which reduced the ability to confirm nerve root involvement. The total acquisition time was approximately ten minutes.

MRI examination revealed diffuse thickening of the lumbosacral nerve roots L2–S1 (Figure 1A,B) and scalloping of the posterior vertebral bodies (Figure 2A,B). The diagnosis of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) was the most likely cause. After commencing corticosteroid treatment, the patient showed a good response and improvement.

CIDP is an acquired immune-mediated neuropathy with a prevalence of approximately 1–9 per 100,000 and a higher male-to-female ratio (2:1), with a peak incidence in the fifth to sixth decades of life. However, it rarely involves children and young adults, creating diagnostic challenges, particularly when atypical radiological features are present. The differential diagnosis included acute Guillain-Barré syndrome, multifocal motor neuropathy, Charcot-Marie-Tooth disease, or

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diabetic neuropathies.

This case had several unusual features. The combination of young age and female sex is rare, whereas a good response to steroids has been reported in 70–80% of patients who improve with first-line immunomodulatory therapy. The imaging findings were also unusual; diffuse hypertrophic changes in the lumbosacral nerve roots are often mistaken for hereditary neuropathies such as Charcot–Marie–Tooth disease, which typically present with uniform, symmetric nerve root enlargement lacking inflammatory features. NCS/EMG revealed acquired demyelinating features with conduction block and temporal dispersion, favouring CIDP over hereditary or neoplastic mimics.

One limitation of this case is the absence of fat-suppressed sequences, as our hospital lacks a robust image-archiving backup system. In addition, the lack of contrast-enhanced images limits the ability to confirm nerve root involvement or detect subtle inflammatory changes, necessitating cautious interpretation of the imaging findings.

The prognosis of CIDP is generally favourable with sustained treatment; however, relapse has been reported in 30–50% of patients within five years.

This case underscores the importance of considering CIDP in adolescents and young adults presenting with chronic neuropathies, particularly when distinctive MRI findings are present. Early recognition, tailored immunotherapy, and patient education and rehabilitation remain essential for optimising functional outcomes.

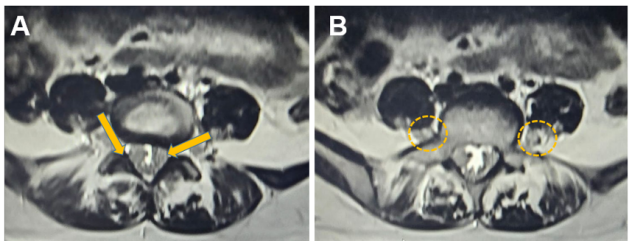

Figure 1. Axial T2-weighted images at L4–5 (A). Diffuse intradural thickening of the nerve roots causes marked circumferential narrowing of the dural sac, extending to the extramedullary component that expands the exit nerve roots. These combined features lead to spinal canal compression, as shown in (A), marked with yellow arrows. (B) Axial T2-weighted images at L5–S1. The canal is almost completely filled with hypertrophic nerve roots, with enlargement of the bilateral exit nerve roots (yellow circles). These findings highlight nerve-root hypertrophy, favouring CIDP over hereditary or neoplastic mimics. Axial images were included because they show segmental details of involvement and highlight the degree of canal compromise and nerve-root enlargement.

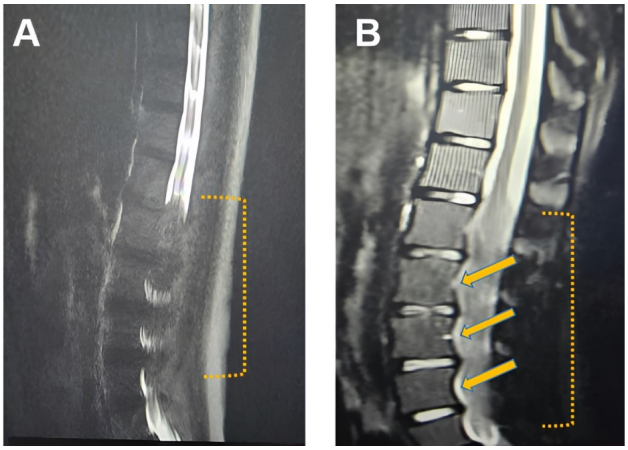

Figure 2. Parasagittal MR myelogram at L1–L5 (A) shows diffuse intradural thickening with hypertrophic, clumped nerve roots extending from L2 to S1, causing near-complete obliteration of the spinal canal. There is loss of the T2-hyperintense CSF signal, reflecting marked canal compression. In the lumbosacral region, the spinal canal is almost completely filled with hypertrophic nerve roots. The T2-weighted sagittal image (B) demonstrates posterior vertebral body scalloping (yellow arrows), indicating chronic remodelling and cortical bone resorption due to longstanding nerve-root enlargement. Sagittal images are included to depict the full craniocaudal extent of nerve-root hypertrophy from L1 to L5.