



Images in medicine

Longitudinally extensive myelitis with complete spine cord involvement associated with systemic lupus erythematosus



Mielitis longitudinalmente extensa con afectación completa de la médula espinal asociada a lupus eritematoso sistémico

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A 34-year-old woman with a 16-year history of SLE (systemic lupus erythematosus) presented with rapidly progressive limb paresis and loss of sphincter control. She had a history of neurological, articular, and mucocutaneous SLE manifestations and was on azathioprine, chloroquine, and prednisolone. Examination revealed severe lower limb weakness (1/5 strength), absent deep tendon reflexes, and pleocytosis with elevated cerebrospinal fluid protein. MRI (magnetic resonance imaging) showed LETM (longitudinally extensive transverse myelitis) from the medulla oblongata to L1 (Fig. 1a). Anti-aquaporin-4 antibodies were negative, ruling out NMOSD (neuromyelitis optica spectrum disorder). Treatment included methylprednisolone pulses, plasmapheresis, and cyclophosphamide. The patient showed partial improvement, with complete recovery over 18 months. Follow-up MRI confirmed resolution of spinal lesions, supporting LETM secondary to SLE (Fig. 1b). LETM primarily affects the thoracic spinal cord, with MRI showing T2 hyperintense lesions spanning over three vertebral segments. Differential diagnoses include NMOSD (negative anti-aquaporin-4 antibodies) and multiple sclerosis (lack of dissemination in space and time). Approximately 80% of LETM cases in SLE have antiphospholipid antibodies, suggesting a role for vasculitis or thrombosis. This case highlights LETM as a severe neurological complication of SLE, necessitating prompt immunosuppressive therapy for optimal recovery.

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Figure 1. MRI contrasted sagittal T2 image showing: A: Cervical level with alteration in the signal intensity of the spinal cord from the bulbomedullary junction to the height of C5 (—→), and discrete expansion of the spinal cord. Thoracic level with alteration in the signal intensity of the spinal cord, with predominantly central hyperintensity in T2 (*) and extending from the C7-T1 level to the conus medullaris (+), without contrast enhancement areas. CONCLUSIONS: Alteration in signal intensity of the spinal cord, with central predominant heterogeneous hyperintensity. Lesion extends from the medullary junction to C5 (—→) and from C7-T1 to the conus medullaris (+). B: Image four years post-discharge, which appears within normal limits. There is no evidence of spinal cord injury when compared with previous studies.

Ethical considerations

The patient consented to the reporting of her case and signed written informed consent. This report was endorsed by the Institutional Ethics Committee.

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Conflicts of interest

All authors declare that they have no conflicts of interest.