NEUROIMAGES

Bilateral extensive corticospinal tract lesions in MOG antibody–associated disease

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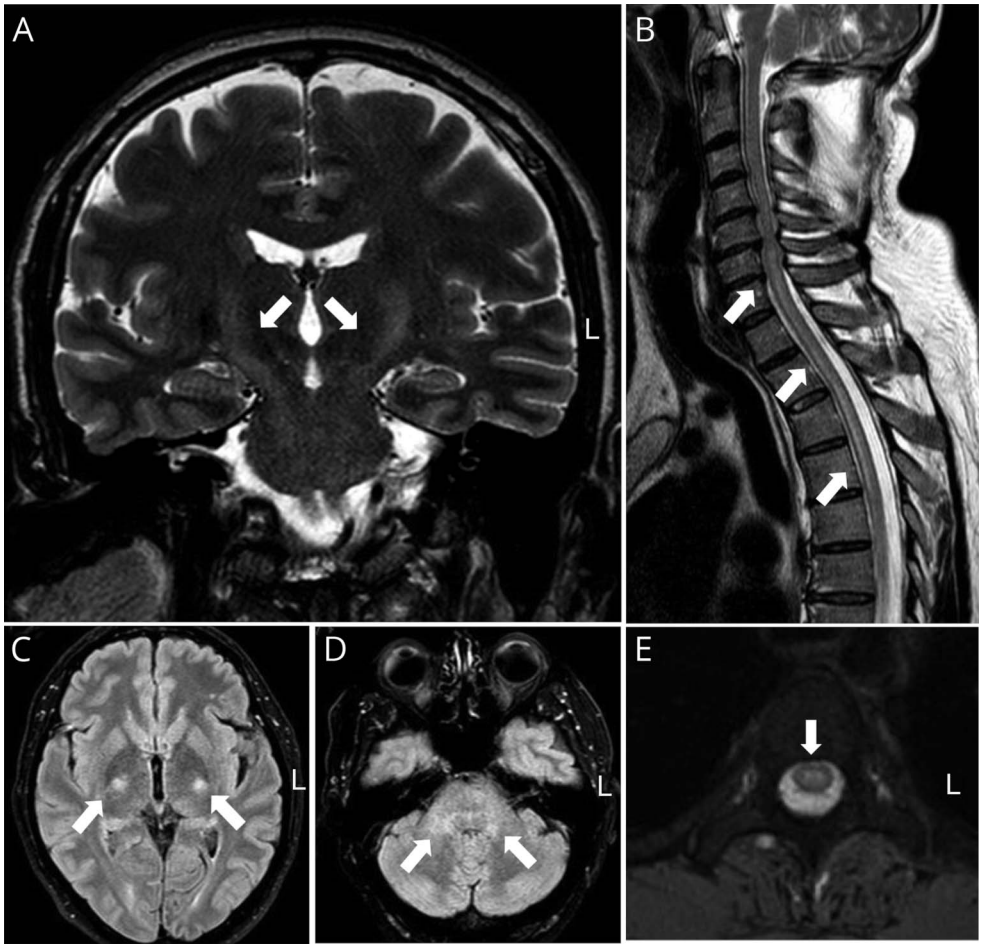
Figure 1 Brain and spinal cord MRI at presentation



T2 sequences show bilateral hyperintensity of corticospinal tracts, extending from the internal capsule to pons (A), and a longitudinally extendend hyperintese lesion of the spinal cord (B). Axial fluid-attenuated inversion recovery (FLAIR) sequences document bilateral lesions of corticospinal tracts (C) and middle cerebellar peduncles (D). Axial T2-fast field echo (T2-FFE) slice shows involvement of the central portion of the spinal cord (E, T3 level).

A healthy 53-year-old man developed acute flaccid paraplegia, spinal sensory level, and urinary retention. Examination revealed bilateral pronator drift. MRI demonstrated demyelinating non-enhancing lesions involving corticospinal tracts (CSTs), middle cerebellar peduncles, and spinal cord (figure 1). CSF analysis disclosed lymphocytic pleocytosis and mildly elevated protein.

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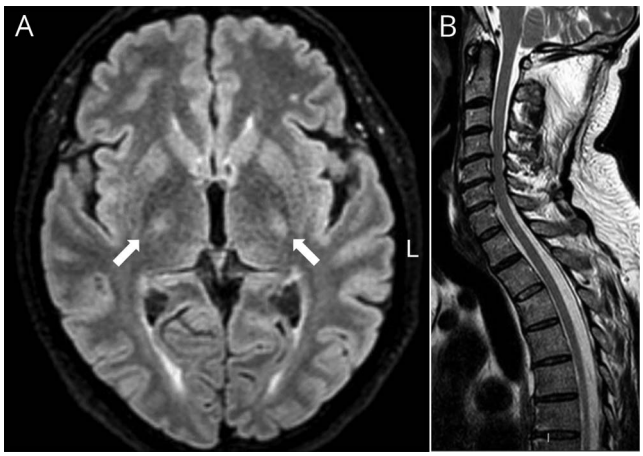
Figure 2 Brain and spinal cord MRI after treatment



Follow-up brain and spinal cord MRI after 24 days shows reduction of corticospinal tract hyperintensity in axial fluid-attenuated inversion recovery images (A); spinal cord MRI demonstrates resolution of cord lesion in sagittal T2 sequences (B).

Euroimmun fixed cell-based assay for serum anti–myelin oligodendrocyte glycoprotein (MOG) and anti–aquaporin-4 (AQP4) antibodies showed low positivity for MOG immunoglobulin G (IgG) (1:10). Treatment with high-dose glucocorticoids and IV immunoglobulin was effective (figure 2). Long CST lesions were found in anti–AQP4 IgG–seropositive neuromyelitis optica spectrum disorders.[1] Bilateral extensive lesions following CST are a novel finding in MOG-related CNS autoimmunity.[2]

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Disclosure

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Appendix Authors

Name	Location	Contribution
Vincenzo Mastrangelo, MD	Department of Biomedical and Neuromotor Sciences, University of Bologna, Italy	Designed and conceptualized study, analyzed the data, drafted the manuscript
Gian Maria Asioli, MD	Department of Biomedical and Neuromotor Sciences, University of Bologna, Italy	Acquisition of data, drafted the manuscript
Matteo Foschi, MD	Neurology Unit, S. Maria delle Croci Hospital–AUSL Romagna, Ambito Di Ravenna, Italy	Revised the manuscript for intellectual content
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