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Neurol Neuroimmunol Neuroinflamm
2017;4:e364; doi: 10.1212/NXI.0000000000000364

VANISHING SPINAL CORD AFTER VARICELLA-ZOSTER VIRUS MYELITIS

OPEN

A 35-year-old woman presented with paraparesis, T7 sensory level, and urinary retention 5 days after developing chickenpox. Spinal cord MRI showed a longitudinally extensive myelitis (figure 1). Despite treatment with IV methylprednisolone (1 g/d × 5) and acyclovir (10 mg/kg/8 h × 3 weeks), the patient developed complete paraplegia, bilateral arm paresis, and a cervicothoracic sensory level. New MRI showed a cystic-like cervical lesion and patchy signs of subacute hemorrhage with gadolinium enhancement from C7 to conus (figure 1). The follow-up MRI 18 months later showed severe spinal cord atrophy below C7 with hemosiderin deposit (figure 2). The MRI findings, and the devastating evolution, mirror the pathologic features described in acute ascending necrotizing myelitis.[1]

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Author contributions: M.S., J.A., and A.S.: analysis/interpretation of the data and drafting and reviewing the manuscript. J.B., N.S.-V., J.S., Y.B., S.L., and F.G.: acquisition of data and revising the manuscript for intellectual content. All authors have approved final approval of the manuscript.

Study funding: No targeted funding reported.

Disclosure: M. Sepúlveda, J. Almeida, and J. Berenguer report no disclosures. N. Solá-Valls received research support from Instituto de Salud Carlos III, Spain and Fondo Europeo de Desarrollo Regional, Predoctoral grant for Health Research. J. Saura received travel funding and/or speaker honoraria from Pfizer. Y. Blanco reports no disclosures. S. Llufriu served on the scientific advisory board for Ixico and Novartis and received travel funding and speaker honoraria from Biogen, Teva, Novartis, and Genzyme. F. Graus is on the editorial board for Lancet*; holds a patent for use of IgLON5 as a diagnostic text; and receives publishing royalties from Euroimmun. A. Saiz received travel funding and/or speaker honoraria from and consulted for Bayer-Schering, Merck-Serono, Biogen Idec, Sanofi-Aventis, Teva, and Novartis. Go to Neurology.org/nn for full disclosure forms. The Article Processing Charge was funded by the Fundació Clinic per la Recerca Biomédica.*

Received March 29, 2017. Accepted in final form April 19, 2017.

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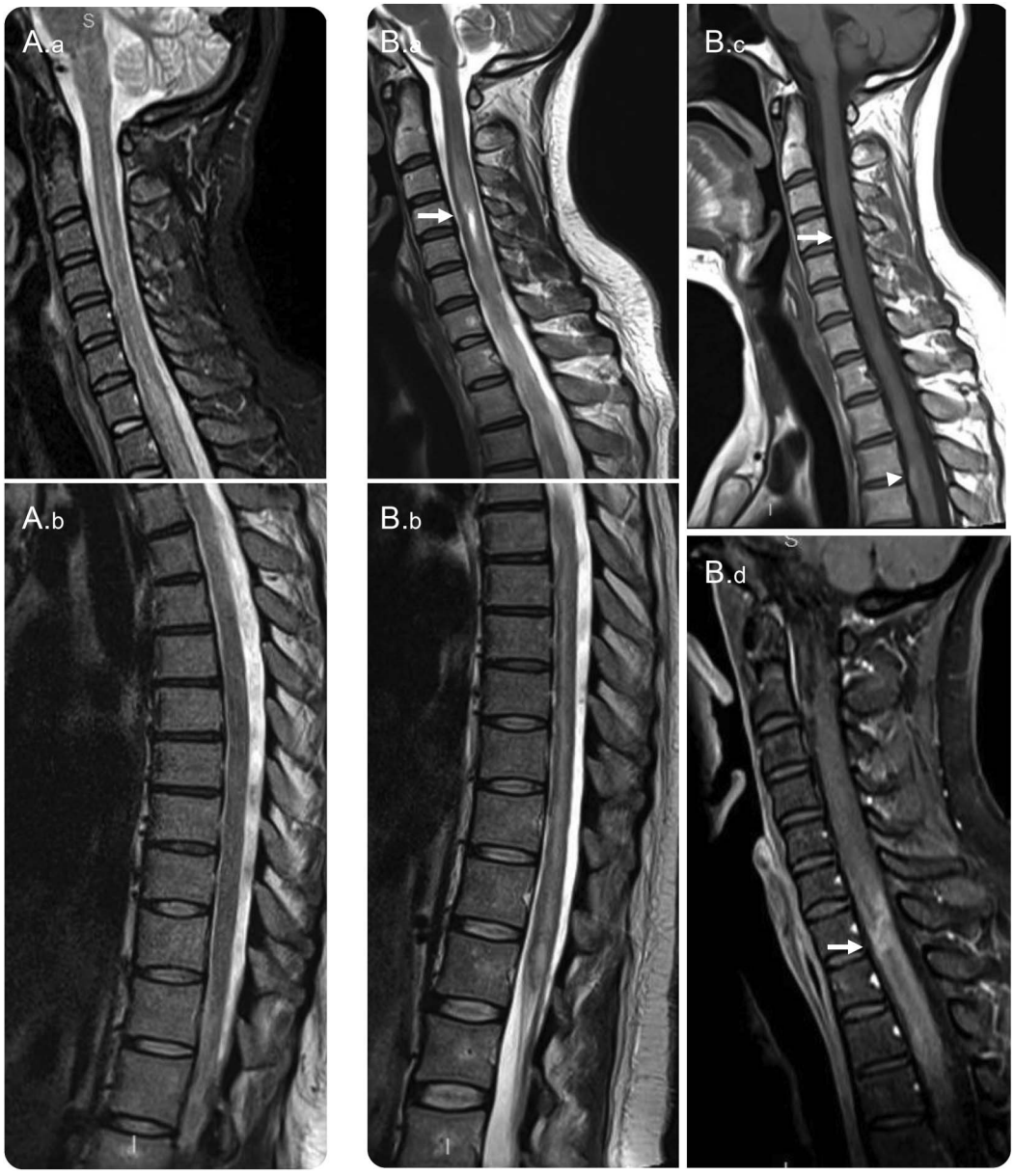
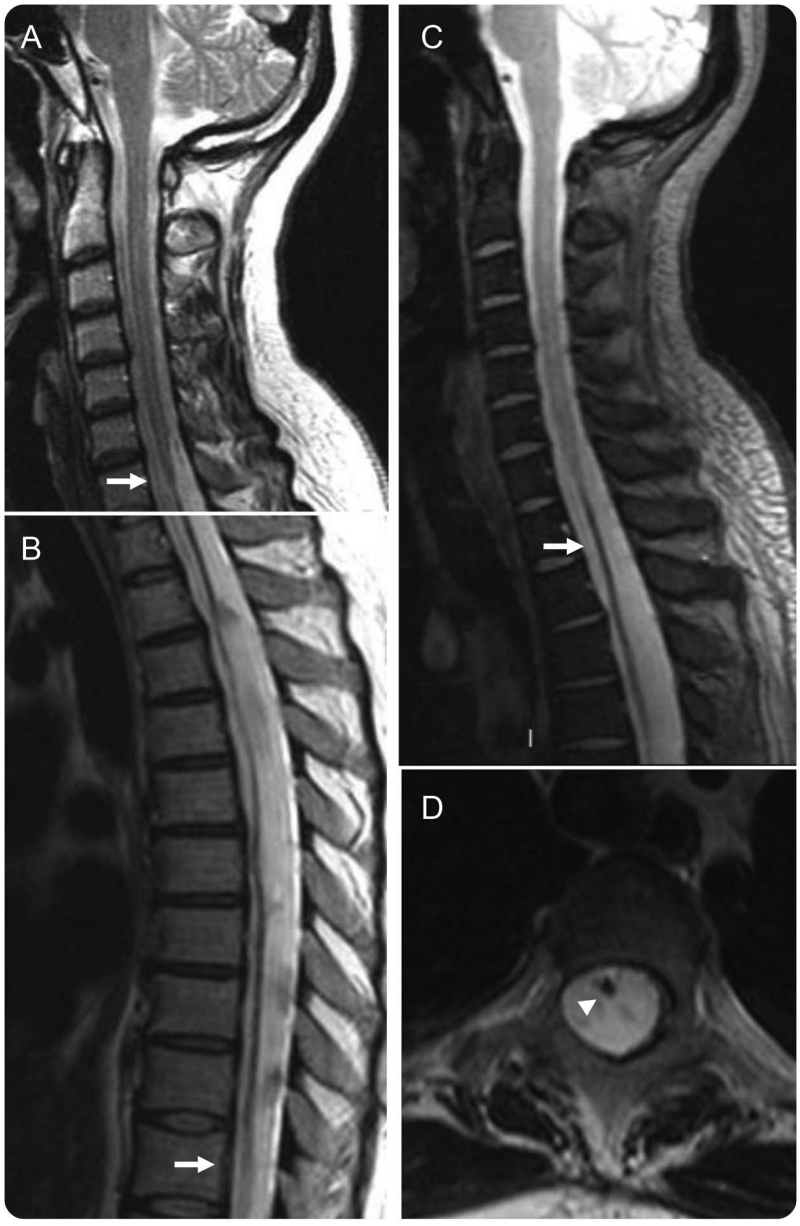
Figure 1 **Spinal cord MRI features at onset**



At onset, sagittal MRI shows an extensive cervicothoracic T2 hyperintensity (A.a and A.b). After 3 weeks of therapy, the extensive lesion persists (B.a and B.b) and shows a central cystic-like lesion at C4 level (B.a and B.c, arrows), suggestive signs of subacute hemorrhage (T1 hyperintensity; B.c, arrowhead), and gadolinium enhancement from C7 level (B.d, arrow).

Figure 2 **Severe spinal cord atrophy at long term**



Follow-up MRI 18 months later reveals a longitudinally severe spinal cord atrophy from C7 to conus (A and B, arrows). Note the filiform aspect of the spinal cord (axial T2-weighted image; D, arrowhead), and the hypointensity in T2 (B), and T2 gradient echo–weighted (C) images suggestive of hemosiderin deposit.

Vanishing spinal cord after varicella-zoster virus myelitis

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Neurol Neuroimmunol Neuroinflamm 2017;4;
DOI 10.1212/NXI.0000000000000364