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VANISHING SPINAL CORD AFTER VARICELLA-ZOSTER VIRUS MYELITIS

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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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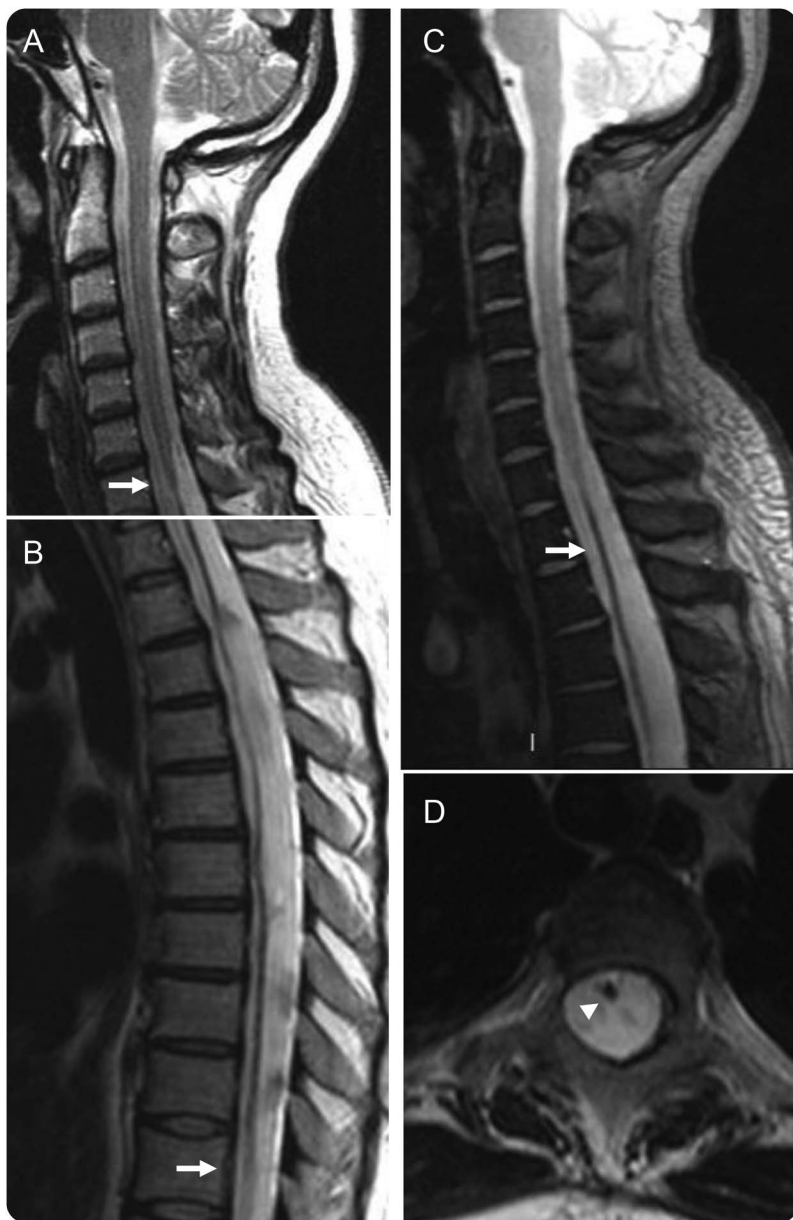
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Figure 1 Spinal cord MRI features at onset



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.

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Vanishing spinal cord after varicella-zoster virus myelitis

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