


Encephalomyelitis After Varicella Zoster Reactivation in an Immunocompromised Patient

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Keywords

central nervous system diseases, encephalomyelitis, acute disseminated

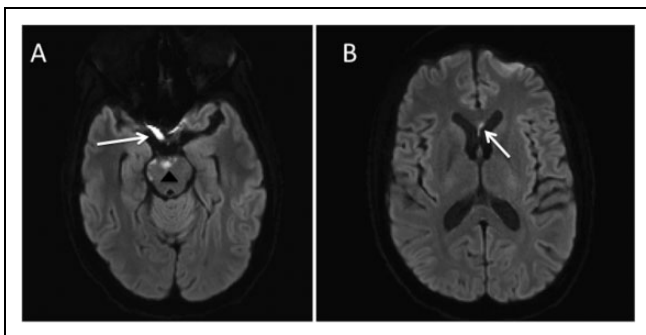


Figure 1. A, Axial diffusion-weighted magnetic resonance imaging (DW-MRI) demonstrates multifocal areas of restricted diffusion involving the pons (arrowhead) and right prechiasmatic optic nerve (arrow). B, There was additionally restricted diffusion in the corpus callosum (arrow).

A 33-year-old man with HIV/AIDS (CD4 50) with a recent diagnosis of disseminated mycobacterium avium complex presented 3 months later with headaches, new-onset seizures, right-sided blindness, and flaccid lower extremity paralysis. Lumbar puncture showed a lymphocytic pleocytosis and an elevated protein of 887.

Magnetic resonance imaging (MRI) of the brain demonstrated multifocal small-vessel territory ischemic infarcts (Figure 1). Magnetic resonance angiography demonstrated a mild beaded appearance of the intracranial vasculature most prominent in the right M1 branch of the middle cerebral artery. These findings are consistent with a mixed large-vessel and small-vessel vasculopathy. T2-weighted MRI of the thoracic spine demonstrated cord signal abnormalities (Figure 2), consistent with longitudinally extensive transverse myelitis. Central canal prominence (Figure 2B) may be related to alterations in cerebrospinal fluid flow dynamics. Cerebrospinal fluid polymerase chain reaction was confirmatory for varicella zoster virus. The patient was treated with parenteral and then oral acyclovir; however, his neurologic deficits

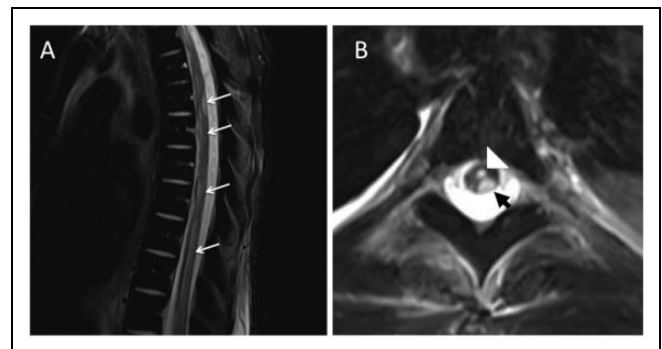


Figure 2. A, Sagittal T2-weighted magnetic resonance imaging (MRI) of the thoracic spine demonstrates a long segment of T2 hyperintense cord signal abnormality (white arrows). B, Axial T2-weighted MRIs of the thoracic spine (T5-T6 level) demonstrate T2 hyperintense cord signal abnormality, which predominantly involves the left dorsal column (short arrow). Prominence of the central canal is also seen (white arrowhead).

of lower extremity paralysis and right monocular vision loss still persisted.

Varicella zoster virus reactivation can result in myriad neurologic sequelae, including vasculopathy, ischemic optic neuropathy, and transverse myelitis.^{1,2} This patient has involvement of the optic nerve, which is rare. The pathophysiology is ischemic optic neuropathy likely due to involvement of the temporal artery, which can mimic giant cell arteritis.¹ Restricted diffusion can be used to help differentiate ischemic

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optic neuropathy from optic neuritis, which can present similarly and also occurs in the immunocompromised.^{1,3-6}

Declaration of Conflicting Interests

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