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Serial diffusion-weighted MR imaging findings in a patient with Epstein-Barr virus encephalitis

Sirs: Acute encephalitis is a serious neurological complication associated with Epstein-Barr virus (EBV) infection and its neuroimaging abnormalities have been infrequently reported [1]. We describe a patient with EBV encephalitis in whom serial diffusion-weighted MR imaging (DWI) showed reversible vasogenic edema in the bilateral basal ganglia along with a complete clinical recovery within a few weeks. To our knowledge, no study has used serial DWI and calculation of apparent diffusion coefficient (ADC) values in cases of EBV encephalitis with basal ganglia involvement.

Our patient was a previously healthy, 17-year-old male student who developed several seizures and subsequent mental deterioration after 3 days’ history of mild fever and throbbing headache. On admission, he was drowsy but did not show any focal neurological deficits. Vital signs were normal except for a mild fever (37.9 °C). Neither hepatosplenomegaly nor lymphadenopathy could be demonstrated. Initial brain CT was unrevealing and the first DWI showed symmetrical hyperintensities in the bilateral basal ganglia and left insular cortex with increased ADC values (Fig. 1A, B). Conventional MRI showed hyperintensities in the same regions on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images (Fig. 1C). No contrast enhancement was found and the vascular morphology was normal on MR angiography.

Laboratory studies were significant for marginal leukocytosis (lymphocyte 65 %) and elevated creatine kinase. The CSF contained 150 white cells/mm³ (60 % lymphocytes), elevated protein (80 mg/dL), and normal glucose. The serum and CSF concentrations of lactate and pyruvate were normal. Antibiotic treatment was started with ceftriaxon and ampicillin. The antiviral agent acyclovir was given as well. On day 2, he regained alertness but was confused and occasionally agitated. The CSF was found to be positive for EBV DNA, as detected by polymerase chain reaction (PCR). Serologic studies revealed positive IgM titers for both the EBV viral capsid antigen (> 1:12) and EBV nuclear antigen (> 1:12), confirming the diagnosis of acute EBV encephalitis. Serologic or CSF studies for Japanese encephalitis, Creutzfeldt-Jakob disease, Wilson disease, Huntington disease, and acute demyelinating encephalomyelitis (ADEM). All of these disorders were excluded in our patient on clinical grounds and based on the results of detailed serologic and CSF tests.

A prognosis of EBV encephalitis is generally favorable; however, severe cases may be lethal or result in permanent disability [1, 5]. Reliable indices for predicting the prognosis in individual cases have not yet been established. Sener [8] described DWI findings in herpes simplex encephalitis and found that increased ADC values, representative of vasogenic edema, were
associated with a favorable outcome. In contrast, reduced ADC values, suggesting cytotoxic edema, may indicate tissue necrosis and a poor outcome. Similarly, Axer et al. [9] reported two cases of ADEM involving the brainstem and found that vasogenic edema were associated with an excellent prognosis with reversibility of the lesions, whereas cytotoxic edema led to persistent disability with irreversible tissue damage.

Interestingly, the first DWI in our patient showed hyperintensities due to the T2-shine-through effect (the substantial contribution of T2 hyperintensities observed on DWI images) and increased ADC values, which is suggestive of vasogenic rather than cytotoxic edema. The follow-up DWI with FLAIR images revealed a substantial reduction in the extent of the lesions to normal and normalized ADC values along with the marked clinical recovery. Whether MRI signal abnormalities are linked to EBV immune reaction or caused by direct EBV invasion into the central nervous system remains to be determined. However, serial DWI findings of reversible vasogenic edema as well as a complete recovery following appropriate treatment may suggest the role of demyelinating inflammation or vasculitis in the pathogenetic mechanism of the basal ganglia lesion in EBV encephalitis.

This case report suggests that the brain lesions affected by EBV infection show reversible vasogenic edema, providing evidence for the reversibility of the lesions and a favorable outcome in patients with EBV encephalitis.

References