Leptomeningeal Enhancement in Acute Cerebellitis Associated with Epstein-Barr Virus

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A 58-year-old healthy woman suffered from headache and high fever above 38°C. At three days after symptom onset, she noticed speech disturbance. On the following day, she was admitted to our hospital because of gait disturbance and exacerbation of the speech disturbance. On admission, her body temperature was 37.5°C. Neurological examinations disclosed ataxic dysarthria, limb ataxia and wide-based gait. Meningism was not apparent. Titers of Epstein-Barr virus (EBV) virus capsid antigen (VCA) IgG were 6.3 (normal range <1.0), VCA IgM 0.0 (normal range <1.0), Epstein-Barr nuclear antigen (EBNA) 8.0 (normal range <1.0), early antigen (EA) 0.2 (normal range <1.0) by enzyme-linked immunosorbent assay (ELISA) method. They indicated a previous infection of EBV. Cerebrospinal fluid (CSF) was watery clear and a cell count was 797/mm³ (a mononuclear cell 584, a polynuclear cell 213), a total protein 108 mg/dl, a glucose 45 mg/dl (a blood sugar 111 mg/dl). Furthermore, EBV-DNA was positive by polymerase chain reaction (PCR) whereas herpes simplex virus (HSV) and varicella zoster virus (VZV)-DNA were negative. T2-weighted brain MRI showed no abnormalities (Picture 1A). However, gadolinium-enhanced T1-weighted brain MRI demonstrated a leptomeningeal enhancement of the cerebellum (Picture 1B). She was diagnosed as having acute cerebellitis. The symptoms gradually improved after admission. Examinations of CSF 17 days after onset showed that the cell
count was 79/mm³, a total protein 25 mg/dl. T2-weighted image 21 days after onset did not show abnormalities including atrophy (Picture 1C), and enhanced MRI revealed that the degree of leptomeningeal enhancement was decreased (Picture 1D). She was discharged at 33 days after onset without neurological deficits. While the follow-up examination of CSF 72 days after onset showed that the EBV-DNA remained positive, that at 163 days after onset did not detect the EBV-DNA. Although the reactivation of EBV was not confirmed serologically, we considered the cerebellitis was caused by intrathecal reactivation of EBV. In a literature search of the English language literature, there has been only one case report demonstrating an abnormal finding of enhanced MRI in EBV-associated cerebellitis. The finding is an enhancement of the cerebellar hemisphere (1). To our knowledge, this is the first English language report which demonstrates leptomeningeal enhancement in acute cerebellitis associated with EBV. An enhanced MRI may be useful to confirm lesions in EBV-associated acute cerebellitis.

Reference