Nominal dysphasia and euphoria caused by EBV encephalitis

Kursat Bora Carman,¹ Ayten Yakut,¹ Arzu Ekici,¹ Sedat Isikay²

¹Department of Pediatric Neurology, Eskisehir Osmangazi University Hospital, Eskisehir, Turkey
²Department of Pediatric, Gaziantep University, Gaziantep, Turkey

Correspondence to
 Dr Kursat Bora Carman,
 kbcarm@gmail.com

SUMMARY

Encephalitis is an uncommon neurological complication of Epstein-Barr virus (EBV) infection and usually presents with confusion, decreased level of consciousness, fever, epileptic seizure, emotional instability and chorea. We present a patient with EBV encephalitis, characterised by nominal dysphasia, euphoria and personality changes.

BACKGROUND

Epstein-Barr virus (EBV), cause of infectious mononucleosis (IM) which is a benign, self-limiting disease, is a member of the human herpes virus family.¹ However, about 20% of patients with primary EBV infection have various complications.² Central nervous system (CNS) involvement occurs in about 1–5% of the patients with IM. Neurological complications of EBV infection are meningitis, encephalitis, cranial nerve palsies, cerebellitis, myelitis, metamorphopsia (Alice in Wonderland syndrome). The manifestations of EBV encephalitis may precede, occur concomitantly with, or follow the symptoms of IM with various intervals, occasionally in the absence of clinical features of IM.^{3–4} EBV encephalitis most often presents with confusion, decreased level of consciousness, fever, epileptic seizure, emotional instability and chorea.^{1–3} We present a patient with EBV encephalitis, characterised by nominal dysphasia, euphoria and personality changes.

CASE PRESENTATION

A 15-year-old previously healthy girl was admitted to our hospital with 2 weeks history of fever (38°C), headache, vomiting, difficulty in naming objects, forgetfulness and euphoria.

A physical and neurological examination revealed a head circumference of 54 cm, a height of 157 cm and a weight of 37 kg. No skin eruption, lymphadenopathy, or hepatosplenomegaly was noted. She was disorientated, dysarthric and she had difficulty in naming objects but her reading and writing abilities were normal. She could not remember the names of her family members. She was euphoric and laughing inappropriately. Her cranial nerves were intact. Fundus examination was normal. No meningeal and focal neurological signs were present. Her muscle tone was normal with normoactive deep tendon reflexes. No pathologic reflexes were found.

INVESTIGATIONS

Laboratory investigations including biochemistry, complete blood count, peripheral smear were normal. Hepatitis markers were negative. Analysis of a cerebrospinal fluid (CSF) showed a protein

level of 29 mg/dl, a glucose level of 106 mg/dl and any white blood cells. Herpes simplex virus (HSV) PCR test was negative.

Serum anti-EBV antibodies are as follows: viral capsid antigen (VCA) IgM (–), VCA IgG(+), EBV nuclear antigen (EBNA) IgM (+), EBNA IgG(+), early antigen (EA) IgM(+), EA IgG(–), p22 IgM (+).

Brain T2-weighted MRI showed hyper intensities on left temporal lobe, insular cortex and uncus (figure 1).

DIFFERENTIAL DIAGNOSIS

HSV encephalitis, other viral encephalitis, acute stroke.

TREATMENT

Acyclovir treatment was given for 2 weeks. Three days later her symptoms were improved except mild euphoria. She was discharged from hospital on the 21st day with nominal dysphasia and euphoria.

OUTCOME AND FOLLOW-UP

During 7 months follow-up period her neurological examination was turned completely normal. She was able to speak fluently. She achieved full recovery. Control brain MRI demonstrated encephalomalacic areas on previously affected regions (figure 2).

DISCUSSION

The neurologic manifestations of EBV infection are meningitis, myelitis, Guillain-Barre syndrome, Metamorphopsia (Alice in Wonderland syndrome) and acute cerebellar ataxia and encephalitis is estimated to occur at a rate of 0.05 cases per year in a population 1 000 000.^{5–6}

The pathogenesis of EBV associated neurological disorders is unclear. Some authors suggest that direct virus invasion of CNS is responsible. Support for this hypothesis comes from observations in which neurological improvement was accompanied by the disappearance of EBV DNA from CSF. Moreover, a brain biopsy study identified EBV DNA in brain sample of EBV associated encephalitis. An alternative theory is that neurological syndromes associated with EBV are immunologically mediated being caused by infiltration of cytotoxic CD8 cells into the leptomeninges or neural tissues.¹

Patients with EBV associated encephalitis typically have fever, headache stiff neck vomiting, altered mental status and frequently accompanied by focal abnormalities, such as hemiparesis and abnormal movements such as chorea. Seizures are present in almost half of patients with EBV.² Our patient presented atypical symptoms such as

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Figure 1 Brain MRI study on admission. Coronal T2-weighted image showing hyper intensities within left temporal lobe, insular cortex and uncus.

dysarthria, euphoria, laughing inappropriately and nominal dysphasia.

Dysphasia is a language characterised by the inability to name people and objects that are correctly perceived. It is associated with lesions of the particularly temporal lobe. Translation of thoughts and images into words is impaired. Pure anomic aphasia is likely to arise from either an inferior temporal lesion or a temporo-parietal lesion.⁷ Our patient has had left temporal

Figure 2 Control brain MRI study 7 months later coronal T2 image showing encephalomalacia on previously hyper intensities within left temporal lobe, insular cortex and uncus.

lesion on brain MRI on admission. The MRI scan of patients with EBV encephalitis might be normal up to 40% of cases.

The cerebral hemisphere, basal ganglia, cerebellum, brain-stem, thalamus are the regions most frequently involved in EBV encephalitis. However the temporal lobe is also main target of HSV encephalitis. Therefore EBV encephalitis may mimic HSV encephalitis.² Ku *et al*⁸ reported a herpes simplex encephalitis patient presented with selective language aphasia. In our patient we ruled out HSV encephalitis because HSV PCR test was negative. In our patient, the diagnosis of EBV encephalitis was based on clinical features, MRI findings and serological tests indicated reactivation type EBV infection. PCR for EBV in CSF which has a limited value in diagnosis was not done. Hausler *et al*⁹ reported 10 patients with neurological complication and detectable circulating EBV DNA, but only one was positive for EBV DNA in CSF. Another research done with 21 patients showed that positive EBV DNA in CSF in 11 of 21 patients diagnosed as having EBV encephalitis.³

The prognosis of EBV encephalitis varies from complete recovery to death. Kasim *et al*⁴ reported that 17% of patients had neurological sequelae such as epilepsy, mental impairment and optic atrophy and 10% patients died. After 1 year follow-up clinical valuation revealed that our patient's neurological examination was normal including speech fluency and comprehension. She demonstrated good recovery. However control MRI demonstrated that encephalomalacic changes were developed in the left temporal lobe, insula and uncus.

Learning point

In conclusion, in patients with nominal dysphasia, behavioural changes and temporal lobe lesions demonstrated on brain MRI, Epstein-Barr virus (EBV) encephalitis should be considered in differential diagnosis and serological tests for EBV should be done.

Competing interests None.

Patient consent Obtained.

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