Prolonged Herpes Simplex Virus Encephalitis in a Patient with Mental Retardation and Epilepsy

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Abstract

Herpes simplex virus (HSV) is a leading cause of severe encephalitis which is related with high mortality and morbidity. This report describes a successfully treated HSV encephalitis (HSVE) with prolonged course in a patient with mental retardation and chronic convulsion. Diagnosis was made by using cerebrospinal fluid (CSF) examination, cranial MRI signs and CSF polymerase chain reaction for HSV. The patient was treated with intravenous acyclovir (ACV) for three weeks which gave a favorable outcome.

Introduction

Herpes simplex virus encephalitis (HSVE), is one of the most severe infections of the central nervous system. HSVE patients who do not receive antiviral treatment have an extremely high mortality rate (70%) (1). The administration of acyclovir (ACV) improved mortality and morbidity for patients with HSVE but, the poor outcome rates still remain high, at 30-50% although highly effective therapy with ACV (2). Some patients with HSVE follow a prolonged course even with appropriate ACV treatment (3).

This report describes a successfully treated HSV encephalitis (HSVE) with prolonged course in a patient with mental retardation and chronic convulsion.

Case Report

A 16-year-old obese female with mental retardation and epilepsy presented with a seven-day history of fever (38°C), convulsion and altered sensorium. On admission she was drowsy, with neck rigidity and meningeal irritation sign. C-reactive protein was 35 mg/L and erythrocyte sedimentation rate was 50 mm/h. White blood cell count and other biochemical tests were normal. An urgent cranial computed tomography was performed and hypodens lesions in right temporal lobe were detected. Cranial MRI with gadolinium showed hypodens lesions and increased signal intensity in temporal, frontal, and talamic lobes. Left lateral ventricle was partially obliterated due to the mass effect of these lesions but there was no shift. Lumbar puncture revealed that 150/mm³ leukocytes (80% neutrophils), a protein concentration of 142 mg/dl, and a glucose concentration of 43 mg/dl (simultaneous blood glucose was 96 mg/dl).

ACV (3x750 mg a day) and ceftriaxone (2x2 gr a day) were started on the first day of hospitalization. She was receiving a combination therapy including valproate, carbamazepine, levetiracetam, lamotrigine and biperidene before admission. The anticonvulsant therapy was modified by neurologists. CSF was sterile and Gram, Ehrlich-Ziehl-Nielsen and India ink stains were negative. Electroencephalography showed epileptic activities, spike and wave discharges in the bilaterally frontotemporal region and real-time polymerase chain reaction (PCR) of HSV-1 in cerebrospinal fluid (CSF) was positive. Therefore, she was diagnosed as HSVE. On day 11 after admission, her clinical picture improved but, lesions placed in temporal lobe and insula progressed and new lesions and cortical contrast fixation were detected in cranial MRI. After four days, the clinical picture of the patient was deteriorated. Her seizures were aggravated, drowsiness and abducens nerve paralysis occurred. There was regressive changes in cranial MRI findings except for perilesioner enhancement of edema. Lumbar puncture performed and 19/mm³ leukocyte, 1/mm³, erythrocyte were detected. CSF protein level was 88 mg/dl and glucose was 43 mg/dl (simultaneously blood glucose was 90 mg/dl). ACV therapy was continued for 22 days. After her clinical and neurological state improved again, she followed up seven days without therapy in hospital. No more problem occurred in this and outpatient period during six months.

Discussion

HSV remains an important cause of meningitis and encephalitis in adults. In spite of high rates of mortality and morbidity in HSVE, ACV treatment has been proven to reduce mortality to approximately 20% (4). In patients with high clinical suspicion of HSVE, CSF analysis including molecular tests yields highest diagnostic value. PCR has become the standard diagnostic test, even a “gold standard” (5). HSV
antibodies or HSV DNA have been detected after the first week from the onset of neurological symptoms (6). In our case, HSV DNA was detected on the second day of hospitalization by real-time PCR. In addition to clinical and CSF findings, both cranial CT and MRI supports the HSVE diagnosis in this case and so, early treatment was begun. But atypical CT findings may be confusable in HSVE. Differentiating HSVE from other intracranial pathologies including perilesioner edema or mass effect may be difficult in some patients (7). In this case, the diagnosis confirmed on the second day of hospitalization by real-time PCR but, on day 14 her clinical situation was progressed and diffuse perilesioner edema was detected in cranial MRI. Therefore, prolonged course of HSVE was suggested and ACV treatment was decided to complete to three weeks. Taira et al. (3) reported eight of 23 HSVE patients as prolonged course without improvement within two weeks. They found significant differences between non-prolonged and prolonged group such as low Glasgow coma scale (GCS) score, poor clinical outcome and higher rates of CT lesions. Low GCS score and higher rate of CT lesions were identified as predictors of prolonged HSVE in multivariate analysis in their study (3). Conventional predictors of poor outcome in HSVE also have been reported as age over 30 years, symptom duration more than four days before ACV treatment, low GCS score ($\leq 6$), presence of abnormal lesions in neuroimaging and $\geq 100$ copies/ml of HSV DNA in initial CSF (8). Some patients with HSVE follow a prolonged course in spite of appropriate therapy with ACV like our case. Prolongation of HSVE was considered to result from insufficient HSV inhibition or secondary encephalitis. Insufficient HSV inhibition may be introduced by low ACV dose and ineffective therapy due to drug resistance. Corticosteroids have been considered beneficial in experimental HSVEs by reducing chemokines and the extent of chronic inflammation and neuronal damage (9). Taira et al (3) found that most of patients given corticosteroids did not have a prolonged course.

In conclusion, early detection and prompt treatment with ACV is essential in HSVE due to high mortality rates. In some patients with poor prognostic factors, the disease may progress with a prolonged course and delayed neurological progression. Prolonged ACV treatment and monitoring closely may reduce morbidity or complications.

References

Illustrations

Illustration 1

Figure 1: Cranial CT shows hypodens lesions in right temporal lobe and mass effect on left lateral ventricle on admission.

Illustration 2

Figure 2: Cranial MRI with gadolinium demonstrated hypodens lesions and increased signal intensity in temporal, frontal and talamic lobes.
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