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Case Report

Herpes simplex encephalitis resulting in neurologic sequelae: A case report

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ABSTRACT

Herpes simplex virus (HSV) is the most common cause of sporadic viral encephalitis. Fever, impaired consciousness, and focal neurological findings are typical clinical features of HSV encephalitis. Herpes simplex virus encephalitis often progresses with focal edema, necrosis, and hemorrhage in the temporal and frontal lobes. Sudden onset fever and symptoms suggestive of temporal lobe involvement are typical clinical features of HSV encephalitis. The mortality rate is high in untreated cases. Permanent neurological sequelae can be seen even in cases treated appropriately and in a timely manner. The gold standard in definitive diagnosis is the detection of HSV-deoxyribonucleic acid in the cerebrospinal fluid by polymerase chain reaction. Cranial magnetic resonance imaging is the best imaging modality for the diagnosis of HSV encephalitis. In this case report, we represent a 62-year-old male patient with HSV encephalitis who developed pneumonia and acute deep-vein thrombosis during treatment.

Keywords: Acute deep vein thrombosis, acyclovir, encephalitis, herpes, pneumonia.

The most prevalent agent of viral encephalitis is the herpes simplex virus (HSV).[1-3] Encephalitis is usually caused by the reactivation of a latent virus.[1,4] It can occur at any age, in every season, and in both sexes.[3,5,6] It is characterized by numerous neurological findings such as epileptic seizures, personality changes, and confusion after a few days of headache and fever. The detection of HSV-deoxyribonucleic acid (DNA) in cerebrospinal fluid (CSF) by polymerase chain reaction (PCR) is the gold standard for diagnosis.[2] Treatment with intravenous (IV) acyclovir at a dose of 30 mg/kg/day for 21 days is recommended. In cases where an adequate clinical response is not obtained, it has been reported that extending the duration of treatment is advantageous.[7] Furthermore, early treatment is critical in preventing mortality and morbidity.[2-8]

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CASE REPORT

A 62-year-old male patient was admitted to the hospital with complaints of nausea, vomiting, fever, absent-mindedness, slurred speech, and impaired balance. It was learned that he had been suffering from nausea, vomiting, and loss of appetite for five days, as well as a high fever, meaningless speech, balance impairment, diabetes, and hypothyroidism for the last two days. Physical examination revealed that the patient's general condition was poor, that he was uncooperative, sleepy, and agitated, and that his nuchal rigidity was suspiciously positive. The body temperature was 39°C, and the blood pressure (BP) was 122/55 mmHg. He was taken to intensive care and intubated. Cranial computed tomography (CT) revealed no acute pathology or edema (Figure 1). The patient underwent a lumbar puncture. The CSF was clear, with 20 leukocytes/mm^3, 50 erythrocytes/mm^3, 61.3 mg/dL protein, and 94 mg/dL glucose (concomitant blood glucose 149). The hemogram revealed a leukocyte count of 12.600/mm^3, and cranial magnetic resonance imaging (MRI) revealed involvement in the temporal and frontal regions (Figure 2). The patient was given IV acyclovir 3×10 mg/kg and IV ceftriaxone 2×2 gr. The

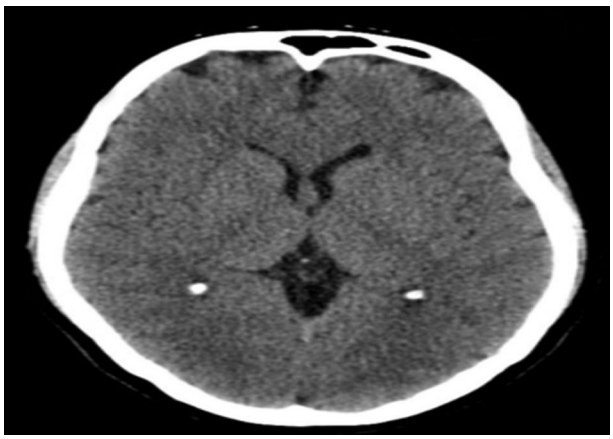

Figure 1. The cranial computer tomography within normal limits.

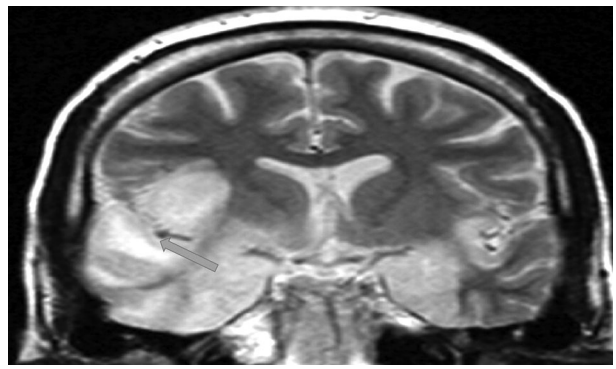

Figure 2. Magnetic resonance imaging: T2-weighted images and after contrast, the area demonstrating non-homogenous intensification, mainly marginal, involving the temporal and parietal lobes and the base of the frontal lobe.

patient was extubated on the fifth day after having stable vital signs and was transferred to the ward on the eighth day. There was no growth in blood and CSF cultures. The CSF HSV-PCR was positive on the eighth day. Electroencephalography (EEG) revealed discharges consistent with encephalitis. Antiepileptic medication was started. On the ninth day of treatment, the body temperature was 39°C. When pneumonic infiltration was detected on posterior anterior (PA) chest graphy, ceftriaxone was discontinued and IV meropenem 3×1 gr was started. On the sixth day of hospitalization, the patient experienced edema, elevated temperature, and tenderness in the right leg. Lower extremity venous Doppler ultrasonography revealed an acute deep vein thrombosis. Thrombectomy is performed by cardiovascular surgery. For 22 days, acyclovir was administered. The patient displayed disorientation and a loss of intellectual function.

DISCUSSION

Herpes simplex virus encephalitis is an infection that causes focal edema, necrosis, and hemorrhage in the temporal and frontal lobes.[9] The sudden onset of fever and symptoms suggestive of temporal lobe involvement is common clinical characteristics of HSV encephalitis. Clinical manifestations include altered mental status, motor weakness, disorientation, aphasia, hallucinations, behavioral disorders, and focal or generalized seizures.[7,9] For example, distinguishing HSV encephalitis from other viral encephalitis or noninfectious diseases is quite challenging. Varicella zoster virus, another herpes virus, can occasionally cause shingles or encephalitis in non-immunocompromised adult patients.[10,11]

Mortality is high in untreated cases. Intravenous acyclovir 30 mg/kg/day is administered in treatment. Even with treatment, however, neurological sequelae are prevalent. Therefore, IV acyclovir is advised until the diagnosis of HSV encephalitis is confirmed or an alternative diagnosis is established. In HSV encephalitis, CSF findings are nonspecific. Mild to moderate lymphocyte-dominated pleocytosis, as well as an increase in erythrocytes and proteins, may be observed. The CSF/serum glucose ratio is usually normal. Polymerase chain reaction assay can identify HSV-DNA during the acute phase and up to 1-2 weeks of treatment.[7] According to a study by Tebas et al.,[12] the sensitivity and specificity of PCR were 96% and 98%, respectively. In the CSF taken on the first day for our patient, HSV-PCR was positive.

In this case with typical clinical and MRI findings, CSF samples were collected within the first week of the beginning of symptoms and sent to the laboratory for PCR detection of the HSV-DNA genome, which revealed a positive result. In cases where herpes encephalitis is suspected (acute or subacute altered consciousness, focal involvement on neurologic examination, temporal lobe involvement on cranial MRI, lymphocytic pleocytosis in CSF, etc.), acyclovir treatment should be started while waiting for the PCR results since the mortality rate is high in untreated cases.

Acyclovir treatment should be continued up until the PCR test is negative, albeit if clinical and radiologic signs are not entirely consistent with herpes encephalitis.[12]

The diagnosis of encephalitis requires a quick neuroradiologic examination. In the detection of HSV encephalitis, MRI is more effective than CT. In 17 cases of encephalitis, Domingues et al.[13] demonstrated that the concordance between MRI and PCR was significantly more significant than CT and EEG. In the early stages, edema is observed in the temporal lobe.[7,13] In our case, the cranial CT performed in the emergency department was found to be normal. Following that, an MRI revealed involvement in the temporal and frontal lobes. Electroencephalography showed periodic lateralized epileptiform discharges consistent with the literature.

In conclusion, CSF should be obtained for PCR analysis for HSV-DNA, and acyclovir treatment should begin right once in patients who have herpes encephalitis based on clinical findings, CSF results, and cranial MRI. The diagnosis is greatly aided by the use of MRI in combination with PCR. It should be remembered that early treatment significantly lowers mortality and neurologic sequelae.

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Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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