

Herpes Simplex Encephalitis Mimicking as Cerebral Infarction

S Pirasath, G Selvaratnam, J Pradeepan

Professorial Medical Unit Teaching Hospital, Jaffna, Sri Lanka

*Corresponding author: Pirasath S, Professorial Medical Unit Teaching Hospital, Jaffna, Sri Lanka, Tel: +94112961000; E-mail: selladurairasath81@gmail.com

Rec Date: Apr 22, 2016, Acc Date: Oct 12, 2016, Pub Date: Oct 20, 2016

Copyright: © 2016 Pirasath S, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited

Abstract

Herpes simplex virus (HSV) encephalitis is the fulminant necrotizing viral encephalitis associated with high mortality and morbidity. Here we report a case of HSV encephalitis mimicking cerebral infarction who recovered with intensive intravenous acyclovir therapy.

Key words:

Herpes simplex encephalitis; Cerebral infarction; Acyclovir

Introduction

HSV encephalitis is caused by HSV-1 (90%) and HSV-2 (10%) in adults. Despite intensive intravenous acyclovir therapy, it has high morbidity and mortality. Polymerase chain reaction (PCR) for HSV in the cerebrospinal fluid (CSF) has a definitive role in diagnosis. Early computed tomography (CT) of brain may reveal changes which mimics cerebral infarction and mislead the diagnosis. Here we report a case with HSV encephalitis mimicking cerebral infarction in CT imaging who recovered with intensive intravenous acyclovir therapy.

Case Report

A 53 year old women presented with a history of fever with headache for 10 days, confusion and one episode of seizure on the day of admission. She denied weakness. She had hypertension, hypothyroidism and hyperlipidemia which were well under control with medication (Losatan 25mg twice daily, Thyroxine 50mg mane, Atrovastatin 10mg nocte). On examination, she was disorientated to place, time and person with receptive aphasia. Further neurological examination revealed neck stiffness with normal symmetrical tendon reflexes and negative babinski sign. Her pulse rate was 84 beats per minute, regular and blood pressure was 130/80 mmHg.



Figure 1: The noncontrast computed tomography imaging scan of brain showed multiple hypodense areas in superior- inferior tentorial region, left temporo- parietal region and cerebellum suggestive of multiple cerebral infarctions/septic embolizations (involved MCA and PCA territories).

Remaining systemic examination was unremarkable. An initial noncontrast CT scan of brain showed multiple hypodense areas in superior-inferior tentorial region, left temporo- parietal region and cerebellum sparing basal ganglia suggestive of multiple territories of cerebral infarction or septic emboli (Figure 1).

Her basic hematological and biochemical investigations including renal and liver profile were normal except mild elevation of inflammatory markers (ESR-50mm/1st hr, CRP-45mg/dL). The cerebrospinal fluid analysis revealed pleocytosis with predominant neutrophils initially, then predominant lymphocytosis, with mild elevation of protein and normal sugar level. The Electroencephalogram (EEG) showed the abnormalities of generalized slow waves with predominant theta and delta waves suggestive of local encephalitis. Repeated CT imaging was also demonstrated above hypodense areas in same regions; not consistent with one arterial or venous territory, favouring diagnosis of localized encephalitis specific for HSV. The HSV I IgM and IgG (20 fold titre) in blood and CSF by indirect immunofluorescence test (ELISA) confirmed herpes simplex virus infection. The workup to rule out atherosclerotic risk factors including vasculitic and thrombophilic screening were negative. She was treated with intravenous antibiotics and acyclovir with steroids cover for three weeks. Her condition was improved with mild memory impairment after course of treatment. On review in four weeks with CT imaging , she was able to go back to her personal job.

Discussion

HSV encephalitis is caused by HSV-1 (90%) and HSV-2 (10%) [1]. It enters into the sensory branch of lingual nerve via infecting nasopharyngeal cells. It ascends to trigeminal ganglion and remains latent for a longtime. Reactivation can result in fulminant haemorrhagic necrotizing encephalitis [2].

The clinical manifestation is nonspecific, but it typically presents with fever, headache, confusion and focal or generalized seizures [3]. This patient presented with these typical clinical symptoms and receptive aphasia. HSV has a high affinity for limbic systems with bilateral or asymmetrical involvement. The limbic system is an integrated structure of brain involved in memory function. This patient also presented with memory impairment and disorientation to place, time and person.

The CSF analysis usually demonstrates pleocytosis predominant lymphocytosis with elevated protein. However, CSF analysis showing pleocytosis with predominant neutrophils initially, then predominant lymphocytosis has been reported in literature [4]. The pleocytosis with predominant neutrophils initially, then predominant lymphocytosis and elevated protein in this patient made possible diagnosis of HSV encephalitis. The isolation of the HSV virus in the CSF is diagnostic value in HSV encephalitis [5]. The testing takes several days to perform. The serological tests (IgM and IgG) are sensitive to detect low amounts of antibodies from spinal fluid, compared with antibody levels in the serum (6). The serum IgG level is more important in diagnosis of HSV encephalitis than serum IgM level (6). The demonstration of IgG antibodies in blood and CSF was the diagnostic tool used to confirm HSV encephalitis in our patient.

The electroencephalography (EEG) shows the abnormalities in medial temporal lobes and inferolateral frontal lobes with bilateral, but typically asymmetrical involvement [7]. It is a sensitive and nonspecific indicator of cerebral involvement that may give clues to the diagnosis and progression of encephalitis (8). EEG may show generalized slow waves with predominant theta and delta waves was also supported diagnosis of encephalitis in our patient.

HSV encephalitis may mimic middle cerebral artery (MCA) infarction demonstrating hypodense areas in imaging studies. However, the basal ganglia are typically spared in HSV encephalitis that will help to distinguish it from a MCA infarction [9]. Gliomas may be misdiagnosed as HSV encephalitis initially, but MRI images of focal, multifocal or diffuse hypo-intensity on T1 images and hyper-intensity on T2 images will be helpful to distinguish it from HSV encephalitis [10].

The combination of the clinical scenario, CSF analysis, EEG and CT imaging findings highly suggested HSV encephalitis and permitted commencement of treatment with intravenous acyclovir. Furthermore, presence of IgG antibodies in serum and CSF confirmed the diagnosis of HSV encephalitis.

Early initiation of treatment determines the outcome of HSV encephalitis and reduces the mortality and subsequent neurological deficit. Overall mortality is over 70%. Prompt treatment with intravenous acyclovir reduces substantially both mortality from 70% to 20% and neurological deficit (11) among survivors. Only 2.5 % of affected patients have ever fully recovered [11], and our patient was fortunate to be among one of them.

References

1. Bulakbasi N, Kocaoglu M (2008) Central nervous system infections of herpesvirus family. *Neuroimaging Clin. N. Am* 18: 53-84.
2. Tyler KL (2004) Herpes simplex virus infections of the central nervous system: encephalitis and meningitis, including Mollaret's. *Herpes* 11: 57A-64A.
3. Whitley RJ, Tilles J, Linneman C (1982) Herpes simplex encephalitis: Clinical assessment. *JAMA* 247: 317-320.
4. Koskiniemi M, Vaheri A, Taskinen E (1984) Cerebrospinal fluid alteration in HSV encephalitis. *Rev Infect Dis* 6: 608-618.
5. Solbrig MV, Hasso AN, Jay CA (2008). CNS viruses--diagnostic approach. *Neuroimaging Clin. N. Am* 18: 1-18.
6. Koskiniemi M., Rantalaiho T., Piiparinen H (2001) Infections of the central nervous system of suspected viral origin: a collaborative study from Finland. *J. Neurovirol* 7: 400-408.
7. Whitley RJ (2006) "Herpes simplex encephalitis: adolescents and adults". *Antiviral Res* 71: 141-148.
8. Westmoreland B.F (1999) The EEG in cerebral inflammatory processes. In: Niedermeyer E., Lopes Da Silva F. Eds, *Electroencephalography*, 4th edition, Williams & Wilkins, Baltimore: 302-316.
9. Tai-Seung Nam, Kang-Ho Choi (2011) Glioblastoma mimicking herpes simplex encephalitis. *J. Korean Neurosurg* 50: 119-122.
10. Banavtala J (2011) Herpes Simplex Encephalitis. *The Lancet Infectious Diseases* 11: 80-81.
11. Leonard JR, Moran CJ, Cross DT (2000) MR imaging of herpes simplex type 1 encephalitis in infants and young children: a separate pattern of findings. *AJR Am J Roentgenol* 174: 1651-1655.