

Temporal Lobe Hemorrhage as a Complication of HSV Encephalitis, a Case Report

Ashes Rijal¹, Sharmila Chaudhary¹, SANGAM SHAH², Asmita Itani¹, Anil Suryabanshi¹, and Sangharsha Thapa³

¹Tribhuvan University Institute of Medicine

²Maharajgunj Medical Campus, Institute of Medicine

³University of Minnesota Medical School Twin Cities

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Abstract

Herpes simplex encephalitis (HSE) is the most common type of sporadic encephalitis that inflicts high rates of morbidity and mortality. Differentiating a progressing encephalitis syndrome from a lobar hemorrhage as a complication presents a challenge and requires great vigilance and insight on part of the treating physician.

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Author:

Ashes Rijal^{1,2}, Sharmila Chaudhary¹, Sangam Shah¹, Asmita Itani¹, Anil Suryabanshi¹, Sangharsha Thapa³

Author's affiliation:

1. Tribhuvan University, Institute of Medicine, Maharajgunj, Kathmandu 44600, Nepal
2. Nepal Health Frontiers, Tokha-5, Kathmandu
3. Jacobs School of Biomedical Sciences, University of Buffalo, Department of Neurology, Buffalo, USA

Abstract

Herpes simplex encephalitis (HSE) is the most common type of sporadic encephalitis that inflicts high rates of morbidity and mortality. Differentiating a progressing encephalitis syndrome from a lobar hemorrhage as a complication presents a challenge and requires great vigilance and insight on part of the treating physician.

Keywords:

Herpes simplex encephalitis, Lobar hemorrhage, Encephalitis

Key clinical message:

This case underlined the importance of having high suspicion for lobar hemorrhage as a complication of HSV encephalitis and shone light upon the added complexity it poses on management on an already deadly disease.

Introduction:

Herpes Simplex Virus (HSV) encephalitis remains one of the most common causes of fatal sporadic viral encephalitis(1, 2). The causative agent in beyond the neonatal period is usually HSV-1 but HSV-2 and HSV-1 both can cause HSV encephalitis (HSE) in neonatal period. The mortality rate is high in non-treated patients compared to treated ones. The mortality rate of HSE has been reduced to 28% from 70% with development of anti viral therapy.(3). HSV- encephalitis accounts approximately 10-20 percent of the 20,000 annual viral encephalitis in US(4). Incidence of HSV encephalitis varies worldwide with incidence reported around 0.7 to 13.8 per 100,000 for all ages. (0.7-12.6 /100000 for adults and 10.5-13.8/100000 for children)(2, 5). Patients with HSV-1 encephalitis usually presents with altered mentation and consciousness for more than 24 hours, fever, new onset of seizures or focal neurological deficits. Focal neurological deficits may include any focal cranial nerve deficits, paraparesis, dysphasia, aphasia or ataxia(6). The diagnosis of herpes simplex encephalitis is confirmed by positive polymerase reactions (PCR) results for HSV DNA in the cerebrospinal fluid(CSF)(7). HSV encephalitis causes perivascular edema and hemorrhagic necrosis of the brain parenchyma, frequently in orbitofrontal and temporal region. Magnetic resonance imaging (MRI) performed in the acute course of the disease helps to get best visualisation of cerebral edema and hemorrhagic necrosis(8). However, the occurrence of a frank intracerebral hematoma in the setting of HSE is rare. We report a case of delayed temporal lobe hemorrhage managed conservatively in an immunocompetent patient with HSE.

Case report:

A 37-year-old male presented to our hospital with persistent severe headache and fever. He was apparently well 8 days back when he had malaise and moderate headache. Following this, the same day he had fever of recorded temp of 104F with chills and rigor associated with generalized myalgia but reported no vomiting, photophobia or loss of consciousness. The next day he had multiple episodes of vomiting followed by an episode of loss of consciousness associated with tonic spasm of body and followed by clonic jerky movements of limbs for about one minute. It was associated with frothing from the mouth, lateral tongue bite and deviation of angle of mouth to the left side. There was no bowel bladder incontinence. It was followed by period of post ictal confusion for about half an hour where he couldn't speak coherently but was able to move all four limbs. He was swiftly taken to nearby hospital and managed as seizure disorder. During the six days stay at the hospital he did not have another episode of loss of consciousness but fever and headache persisted which were still moderate in intensity. Likewise, after the first three days of hospital stay he complained of increased somnolence and was disorientation to time and place. He developed irrelevant talking and mutism at other times. Thus, dissatisfied with the level of care and deteriorating status of patient, the family members brought him to our hospital after being discharged on patient request. On presentation, he had a fever of 102F. On CNS examination his naming, short term, and long term memory were impaired. His sensory and motor examination were intact. There were no cerebellar or meningeal signs. Planters were bilateral downgoing. His chest and cardiovascular examination were within normal limits. Thus with these background clinical features a provisional diagnosis of viral encephalitis was made and acyclovir started.

Investigations were sent, including blood investigation, cerebrospinal fluid (CSF) analysis. Here total white blood cells (WBC)counts were raised to about 19680 cells/ cu mm³ and blood sodium was 127mEq/L rest were within normal limits. On CSF examination, his blood count was raised to 200 cells/cu mm with

monomorphs of 40% and polymorphs of 60%, sugar level was normal (60), and protein was raised (159); RBCs were not mentioned. Microbiological exam of CSF showed no growth on culture and no bacterial cells on gram stain. MRI brain showed features suggestive of herpes encephalitis involving right temporal lobe and cingulate gyrus without any signs of hemorrhage as depicted in figure 1. These findings were supportive of our provisional diagnosis of viral encephalitis. Thus, CSF Polymerase chain reaction (PCR) was sent which came positive for HSV 1 DNA. Thus a final diagnosis of HSV 1 encephalitis was made.

However, on day six he began to develop severe headache and begun to be disoriented to time place and person. Consequently, a head Computed Tomography (CT) scan of head showed hematoma on right temporal lobe, as seen on the figure 2. A coagulation profile was done at that time showed no abnormality. Therefore, he was shifted to medical ICU for close monitoring of his hemodynamic and neurological status. The headache persisted for three more days then gradually subsided. To address the raised ICP head elevation was done, mannitol and hypertonic saline were used but steroids were not. No surgical interventions were needed. He was well oriented to time place and person by the end of sixth day of ICU stay. His naming, short term and long term memory were still impaired otherwise his neurological exam was the same. Following eight days of ICU stay he was shifted to general ward. On discharge, his naming, short term and long term memory function had not still recovered. Motor and sensory examinations were intact throughout. He had completed 21 days of acyclovir therapy. He was discharged after 27 days of hospital stay on levateracetam for seizure prophylaxis and was asked to come for follow up in 15 days. His assessment 15 days later, he still had problems with naming, short term and long term memories but was gradually improving. Rest of the neurological examinations were normal.

Discussion:

Herpes simplex virus is a neuro-tropic virus that enters the body through the mucosal surface. It travels along the axons through retrograde transport and lies dormant in the sensory ganglia. The exact mechanism of transport to CNS is not described but it is postulated that it is a similar mechanism of retrograde transport along the sensory nerves, given its predilection to mesiotemporal lobe and orbitofrontal cortex(9). Once in the CNS, the manifestations of encephalitis prevail. CNS complications include seizures (38.4%), status epilepticus (5.5%), acute respiratory failure (20.1%), ischemic stroke (5.6%) and intracranial hemorrhage (2.7%)(10). Thus, intracranial hemorrhage being rare but fatal consequence in many instance(10). Untreated, the mortality rate in herpes encephalitis due to its complication can reach to 70% and most of the survivors can have serious neurological deficits like neuropsychiatric or neurobehavior issues. HSE encephalitis needs a prompt treatment with IV acyclovir for the reduction of mortality and morbidity from HSV encephalitis and its complication.

The exact mechanism of hemorrhage has not been found but few theories have been described to show the relationship of HSV encephalitis and lobar hemorrhages. Vasculitis or transient hypertension caused by increased ICP plays a major role in having lobar hemorrhage. The hypothesis include the rupture of the small vessels due to these above causes(11). Magnetic resonance imaging (MRI) of the brain is the preferred imaging study; T2 images may be more helpful than T1 images(12).

Our patient's presentation was consistent with HSV encephalitis but later he developed brain hematoma. Otherwise, the clinical features of brain hemorrhage were not distinctive and overlapped significantly with those of HSV encephalitis. It is thus difficult to suspect this complication on clinical grounds alone. The lack of clinical improvement or the worsening of initial symptoms, particularly during first to second week of admission, should lead to this suspicion and be followed by a neuroimaging study. If the evidences discard the possibility of structural complication, a lack of improvement with the treatment of acyclovir may raise concern of acyclovir resistance or treatment adverse effect. This scenario may warrant the start or switch the therapy to foscarnet(13).

During management of this complication, supportive care and intensive monitoring was sufficient, and surgi-

cal intervention was not required. Mannitol was used for management of raised ICP but corticosteroids were not used, the role of which remains still debatable in this area(14). A multicenter randomized controlled trial was done to study the role of corticosteroid in treatment of HSV encephalitis but was prematurely terminated due to small no of study participants(15). Thus, higher level of evidence to support the use of corticosteroid is lacking. Intensive care physicians involvement in the care team seems imminent as up to 32% require ICU stay and 17% require mechanical ventilation(16). Similarly, need for surgical intervention may be as high as 50% following intracerebral hemorrhage as reported by Sainz et. al(17).The neurological outcome in the non-operated group was similar, although in this case it could be argued that they were less severely affected. Thus, the role of neurosurgery in these patients is still unclear.

Conclusion:

In conclusion, herpes simplex encephalitis presents as a potentially lethal disease if it was not for acyclovir therapy. Even still deceased burden remains high. Lobar hemorrhage as a complication further compounds the complexity of management and warrants higher level of care and demands vigilance in part of the management team to detect any neurological deterioration which at times may masquerade just as a progressing encephalitis syndrome.

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Conflict of Interest:

Authors' have no conflict of interest to declare

Authors' Contribution:

AR wrote the original manuscript, reviewed and edited the manuscript. SC, SS, AI, AS and ST reviewed and edited the manuscript.

Data availability statement:

All the required information is available in the manuscript itself.

Ethical consent statement:

Written informed consent was obtained from the patient for publication of case report.

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