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# TEMPORAL LOBE ENCEPHALITIS: THE HALLMARK OF HSV IN ACUTE ENCEPHALITIS SYNDROME

**KEY WORDS:** Encephalitis, Herpes Simplex Virus Type 1, Comorbidity, Magnetic Resonance Imaging

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**ABSTRACT**

Acute encephalitis syndrome (AES) is a continuum of neurological disorders with infectious and non-infectious etiologies, most frequently herpes simplex encephalitis (HSE) involving bilateral temporal lobes. This is a presentation of a patient admitted with fever and altered sensorium, initially suspected of cerebrovascular accident with hypoglycemic brain injury. Laboratory findings revealed anemia, thrombocytopenia, electrolyte imbalance, and metabolic derangements, while magnetic resonance imaging demonstrated bilateral temporal and occipital lobe lesions. Cerebrospinal fluid polymerase chain reaction proved herpes simplex virus infection, and intravenous acyclovir with supportive therapy led to gradual clinical improvement and stabilization.

## INTRODUCTION

Acute encephalitis syndrome (AES) is defined by a sudden onset of fever, sensorium alteration, seizures or focal neurological impairment of the central nervous system and has included both infectious and non-infectious etiologies.[1] In India, the spectrum of AES has evolved over the decades, with Japanese encephalitis (JE) historically dominating outbreaks.[2] Despite this diversity, herpes simplex encephalitis (HSE) remains the most common sporadic focal viral encephalitis worldwide and continues to be a critical consideration in AES due to its treatable nature and high mortality if unrecognized.[3] The distinction between HSE and metabolic encephalopathy is based on the findings of cerebrospinal fluid (CSF) with pleocytosis and increased protein favoring infectious etiologies of the former and typical picture of the latter.[4]

Syndromic approach has been promoted in the Indian subcontinent where AES etiologies are heterogenous and of a regional nature so as to maximise diagnostic and treatment options. This case report presents the geriatric case of acute temporal lobe encephalitis with comorbidities that highlights the role of early identification and specific antiviral treatment in AES.

### Case Report Presentation

A 71-year-old woman with a history of type 2 diabetes mellitus and hypertension for past three years was referred to the hospital, 2 days post altered sensorium with fever and in unconscious state. Her random blood sugar was 60mg/dL at a district hospital at which she was referred. She was presented in a critical condition with blood pressure of 180/92 mmHg, heart rate 79/min, respiratory rate 22/min, temperature 101.6 causing due oxygen saturation being 95% on room air. Neurological assessment showed a Glasgow Coma Scale of E2V1M3, bilateral preservation of pupillary light reflexes, bilateral presence of up going plantar responses, which suggest severe cortical impairment and intact brainstem reflexes. Chest auscultation revealed bilateral crepitations which are in line with aspiration pneumonia. Based on this presentation, the tentative diagnosis was cerebrovascular accident with hypoglycemic brain injury and aspiration pneumonia in a diabetic hypertensive patient.

In the initial investigations, anemia (hemoglobin 9.3-10.8 g/dL), chronic thrombocytopenia (platelet count 0.54-1.25 lac/μL), and hypoalbuminemia (1.9-3.2 g/dL) were found. Electrolyte imbalances were hyponatremia (132 mmol/L) and hypocalcemia (0.98 mmol/L) and borderline hypokalemia (2.9 mmol/L). Arterial blood analysis revealed alkalemia (pH 7.46), low levels of $pCO_2$ (27.9 mmHg), hypoxemia ($pO_2$ 77 mmHg), and mild metabolic acidosis (base excess -4.0). Signal ECG showed a sinus rhythm with right bundle block. Microscopic analysis of urine showed mild pyuria.

MRI brain revealed widespread regions of foci of confined diffusion and T2/FLAIR hyperintensity of the bilateral temporal and occipital lobes with sparing the basal ganglia highly typical of herpes simplex virus (HSV) encephalitis. No mass effect or hemorrhage. (Figure 1) PCR in Cerebrospinal fluid of HSV 1/2 DNA was positive, which proved the diagnosis. Accordingly, final diagnosis was herpes simplex encephalitis in the setting of acute encephalitis syndrome.

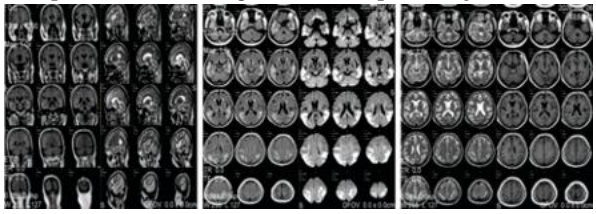

**Figure 1. Magnetic Resonance Imaging (MRI) of the Brain Revealed Asymmetric Involvement of the Bilateral Medial Temporal Lobes, with Predominant Involvement on One Side. The Affected Regions Included the Hippocampus, Amygdala, and Parahippocampal Gyrus, with Extension into the Insula and Inferior Frontal Cortex. On T2-weighted and FLAIR Sequences, there were Hyperintense Signal Changes in the Involved Cortical and Subcortical Regions. Corresponding Areas Appeared Hypointense on T1-weighted Images. Diffusion-weighted Imaging (DWI) Demonstrated Areas of Restricted Diffusion, Consistent with Cytotoxic Edema.**

An empirical therapy was started using injections of acyclovir 10 to 15mg/kg (TID), dexamethasone 16mg (BD), intravenous ceftriaxone 1 g every 12 hours, piperacillin tazobactam, levetiracetam, thiamine, ondansetron, paracetamol, ramipril, and nicardipine daily. Supportive care included oxygen supplementation, intravenous fluids, potassium chloride 20 mEq in 500 mL NS infusion, strict glucose monitoring, and propped-up positioning.

The patient was in coma and had an overall GCS score that varied (E2V2M3 on the first day, then E4V2M2) during the

course in the hospital. The family was informed that there was a guarded prognosis. In the following days, she exhibited progressive normalization of hematological values, as well as partial recovery of the nervous system. She was discharged in stable condition.

## DISCUSSION

HSE is a rapid onset of neurological disorders that either can appear acutely or subacutely and cause focal or diffuse neurological injuries. The commonest causative agent of the sporadic life-threatening encephalitis is HSV 1, and there is no seasonal variation.[5] Disease burden is high in children and elderly, with nearly one-third of cases occurring in younger populations and half in those over 50 years.[6] Recognition of HSE is important because untreated mortality approaches 70%, and even with therapy, complete recovery is achieved by only a minority of patients.[7]

HSE is transmitted through trigeminal or olfactory routes or via reactivation of latent virus, causing neural injury through direct and immune mediated mechanism.[8] It predominantly affects the temporal lobes and limbic system with heightened susceptibility immunocompromised individuals or those on immunosuppressive therapies, and rare cases have been reported following whole-brain radiation.[9] Advanced age and comorbidities likely predisposed the patient to HSV-1 reactivation, making it the most plausible trigger for encephalitis in this case. These comorbidities initially obscured the diagnosis, but the characteristic MRI pattern and confirmatory CSF PCR established HSE, particularly relevant in elderly patients where comorbidities may mask the clinical picture.

Early initiation of intravenous acyclovir against the HSE remains a central theme of the emergency management whereby postponement of over 48 hours has always led to high mortality rate and long-term disability. The drug has a high CSF penetration and is an inhibitor of viral DNA polymerase, therefore, blocking the viral replication [10] though resistance is rare in healthy people and thus alternative agents like foscarnet or cidofovir can be used.[6]

## CONCLUSION

HSE is a rare, but critical neurological emergency that can be fatally high without treatment. The chances of HSV 1 reactivation in patients with comorbid disease, e.g. diabetes and hypertension, emphasize the need to pay attention to the clinical picture.

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