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Case Report

Internal Medicine Section

Herpes Simplex Virus Encephalitis Presenting as Acute Hemiplegia and Altered Sensorium: A Case Report

DEEPAK RAJ[1], R UMASHANKAR[2], RAZOOK FAREEDH[3], MV AISHWARYA LAKSHMI[4], SAKETH RAMINENI[5]

ABSTRACT

Herpes Simplex Virus Encephalitis (HSVE) is the most common cause of sporadic fatal viral encephalitis worldwide and carries substantial morbidity and mortality when antiviral therapy is delayed. The disease typically involves the temporal lobes and presents with fever, altered mental status, seizures, and focal neurological deficits. However, early clinical and radiological features may closely resemble acute ischemic stroke, leading to diagnostic uncertainty. We report a 46-year-old male patient who presented with an acute onset of fever, global aphasia, right-sided hemiplegia, and altered sensorium. Magnetic Resonance Imaging (MRI) demonstrated unilateral cortical T2/FLAIR hyperintensity with diffusion restriction predominantly involving the left temporal and fronto-parietal regions, without conformity to a specific vascular territory. Electroencephalography (EEG) revealed Periodic Lateralised Epileptiform Discharges (PLED) over the left hemisphere. Normal opening pressure, slight lymphocytic pleocytosis (68 cells/mm^3), normal protein and glucose, and negative Gram stain, AFB smear, and fungal investigations were all observed in the CSF study. A normal first CSF does not rule out the diagnosis because early HSVE might appear with little or normal CSF abnormalities. Empirical intravenous acyclovir was initiated promptly. The patient demonstrated neurological stabilisation following antiviral therapy. This case highlights the importance of considering HSVE in patients presenting with acute febrile focal neurological deficits mimicking stroke. Early recognition and timely antiviral therapy significantly reduce mortality and improve neurological outcomes.

Keywords: Acyclovir, Aphasia, Hemiparesis

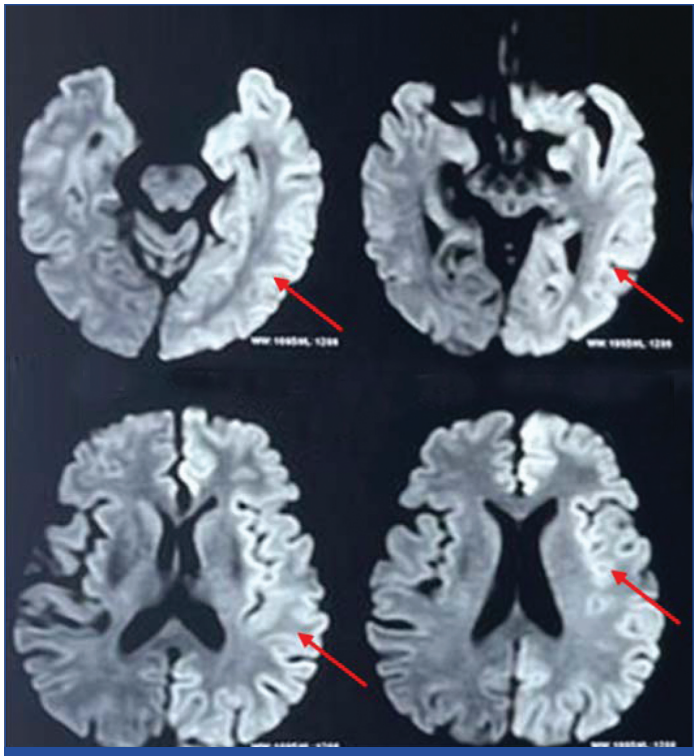
CASE REPORT

A 46-year-old male presented in emergency room with a two-day history of high-grade fever (102.2 F) followed by an acute onset of confusion and reduced responsiveness. He was later found to have global aphasia and inability to move the right upper and lower limbs. There was no prior history of cerebrovascular disease, seizures, head trauma, diabetes mellitus, or chronic neurological illness, and no recent systemic infection or travel history was reported.

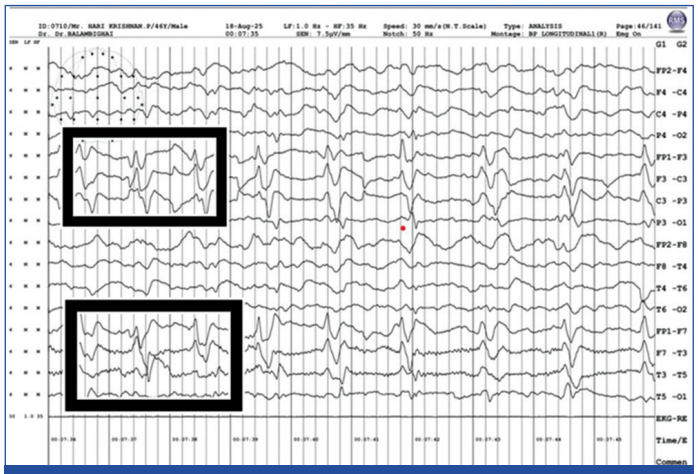
On examination, the patient was febrile (102°F) with increased pressure (200/100 mmHg). His Glasgow Coma Scale score was E4V1M6. Neurological examination revealed global aphasia and right-sided flaccid hemiplegia, with brisk deep tendon reflexes on the right and an extensor plantar response. Brainstem reflexes were preserved, and no meningeal signs were elicited. On investigations MRI of the brain demonstrated T2/FLAIR hyperintensity with diffusion restriction involving the left temporal, insular, fronto-parietal, and occipital cortices, with additional involvement of the left thalamus and corresponding low Apparent Diffusion Coefficient (ADC) values [Table/Fig-1]. The lesions did not confirm to a specific arterial vascular territory, and no large vessel occlusion was identified on angiographic sequences, favouring a diagnosis of viral encephalitis rather than ischemic infarction. EEG revealed left hemispheric PLEDs predominantly over the temporal region, along with background slowing. These findings are indicative of focal cortical irritability and are classically associated with HSVE involving the temporal lobe [Table/Fig-2]. Cerebrospinal fluid analysis revealed a normal opening pressure. The total leukocyte count was 68 cells/mm^3 with a lymphocytic predominance (85% lymphocytes, 15% neutrophils). The protein level was 24.2 mg/dL, and the glucose was 65 mg/dL, with a corresponding blood glucose of 108 mg/dL. Red blood cells were present at 120 cells/mm^3 and minimal cellularity, with negative Gram stain, AFB smear, and fungal studies. It is recognised that early HSVE may demonstrate minimal pleocytosis, and normal initial CSF findings do not exclude the diagnosis. Although HSV polymerase chain reaction is considered the diagnostic gold standard, it could not be performed in this case due to logistic constraint. However, the diagnosis of probable HSVE was made based on the characteristic clinical presentation, typical MRI findings involving the temporal and insular cortex, and supportive EEG abnormalities. Empirical intravenous acyclovir (10 mg/kg every 8 hours) was initiated promptly, along with supportive management including blood pressure control, seizure prophylaxis, and measures to reduce intracranial pressure, while broad-spectrum antibiotics were discontinued following negative microbiological results. By three to five days of antiviral therapy, the patient demonstrated improvement in alertness with stabilisation of neurological deficits, and no further seizures were observed. Although residual right-sided weakness remained, the patient showed some neurological recovery at discharge, with sensorium improving and focal impairments stabilising. Logistical limitations prevented the performance of follow-up neuroimaging.

DISCUSSION

Acute hemiplegia with altered sensorium closely mimicking an acute ischemic stroke represents an uncommon but clinically important presentation of HSVE. In the present case, the patient initially presented with high-grade fever, rapidly progressive encephalopathy, global aphasia, and right-sided hemiplegia, strongly suggesting an acute cerebrovascular event. However, MRI brain demonstrated cortical T2/FLAIR hyperintensities with diffusion restriction involving the left temporal, insular, fronto-parietal, occipital cortices, and thalamus without confinement to a defined vascular territory, favouring an infectious encephalitic process rather than arterial infarction. Similar stroke-like presentations of HSVE have increasingly been reported in recent literature. AlRashed F et al., described a patient presenting with acute focal neurological deficits suggestive of stroke in whom subsequent neuroimaging and clinical progression established HSVE as the underlying diagnosis [1]. Their report emphasised that HSV CNS infection may initially



[Table/Fig-1]: Illustrates MRI brain findings. The red arrow demonstrates unilateral hemisphere diffusion restriction involving the left fronto parietal temporal and occipital cortices, with additional involvement of left thalamus, accompanied by corresponding low Apparent Diffusion Coefficient (ADC) values.



[Table/Fig-2]: Illustrates the EEG findings, with the black box highlighting Periodic Lateralised Epileptiform Discharges (PLEDs) over the affected hemisphere.

mimic acute ischemic stroke because of abrupt onset hemiparesis and aphasia, particularly when early imaging demonstrates cortical diffusion restriction. Grzonka P et al., in a recent systematic review of HSV CNS infections, further highlighted that focal neurological deficits including hemiplegia, aphasia, and cortical signs may dominate the early clinical presentation and frequently lead to an initial misdiagnosis of stroke [2]. Compared with previously reported cases, the patient demonstrated more extensive multifocal cortical involvement with thalamic extension and preceding fever, which increased the suspicion for encephalitis early during evaluation.

Neuroimaging played a crucial role in differentiating HSVE from acute ischemic stroke in the present case. Diffusion restriction extending beyond arterial vascular territories, particularly involving the temporal and insular cortices, strongly supported viral encephalitis. Roçi E et al., reported a 49-year-old man presenting with abrupt aphasia and hemiplegia who was initially treated as acute ischemic stroke until MRI revealed unilateral temporal-insular diffusion restriction compatible with HSVE [3]. Similarly, a recent case report described HSV meningoencephalitis presenting with acute Broca's aphasia and stroke-like deficits, where diffusion restriction involving the temporal and insular cortex ultimately favoured encephalitis over infarction [4]. These reports closely resemble the imaging pattern observed in our patient, although the current case demonstrated more extensive cortical involvement including occipital and thalamic regions. Such multifocal cortical diffusion abnormalities extending beyond vascular territories are atypical for acute infarction and should raise suspicion for encephalitis, particularly in the presence of fever and encephalopathy.

The EEG provided additional diagnostic support in the current case. EEG demonstrated PLEDs predominantly over the left temporal region, correlating closely with MRI abnormalities and suggesting focal cortical irritability involving the dominant temporal lobe. Sutter R et al., demonstrated that EEG plays an important role in both the diagnosis and prognostication of acute encephalitis, with temporal periodic discharges and focal slowing being strongly associated with herpes simplex encephalitis [5]. Similar EEG abnormalities have also been described in stroke-mimic presentations of HSVE, where early EEG findings supported prompt initiation of antiviral therapy before virological confirmation [1,3,4]. In contrast to previously reported cases where EEG abnormalities evolved later during hospitalisation, the early identification of temporal PLEDs in our patient significantly strengthened the clinicoradiological diagnosis and facilitated timely initiation of acyclovir therapy.

Cerebrospinal fluid findings in HSVE may be subtle or even misleading during the early phase of illness and therefore should not exclude the diagnosis when clinical suspicion remains high. In the present case, CSF analysis revealed mild lymphocytic pleocytosis, normal glucose, protein, and red blood cells suggestive of early haemorrhagic temporal lobe involvement. Ahmed WA et al., reported a case of PCR-confirmed herpes simplex encephalitis with initially normal brain MRI and normocellular cerebrospinal fluid, highlighting that early investigations in HSVE may occasionally appear deceptively benign despite active CNS infection [6]. Matthews E et al., further emphasised that early HSVE may demonstrate minimal inflammatory CSF abnormalities despite significant neurological dysfunction and characteristic neuroimaging findings [7]. Compared with these reports, our patient demonstrated moderate inflammatory CSF abnormalities together with characteristic MRI and EEG findings, which facilitated early clinicoradiological diagnosis despite the unavailability of HSV PCR testing. These findings reinforce that HSVE often remains a predominantly clinicoradiological diagnosis in resource-limited settings and requires a high index of suspicion. Early initiation of intravenous acyclovir remains the cornerstone of HSVE management and is strongly associated with improved neurological outcomes. In the present case, empirical acyclovir therapy was initiated immediately after MRI and EEG findings suggested viral encephalitis, resulting in stabilisation of focal deficits and gradual improvement in sensorium within a few days. Similar favourable responses following early antiviral treatment have been described in recent stroke-mimic HSVE cases, where prompt recognition prevented inappropriate thrombolysis and improved neurological recovery [1,3,4]. Aboelezz A and Mahmoud SH demonstrated that delayed acyclovir initiation remains one of the strongest predictors of mortality and poor neurological outcome in HSVE [8]. Similarly, Duerlund LS et al., in a recent prospective cohort study, reaffirmed that early antiviral therapy significantly improves survival and functional recovery in HSV-1 encephalitis [9]. Cleaver J et al., additionally highlighted that delayed recognition and treatment of HSVE may predispose survivors to significant neuroinflammatory and post-infectious autoimmune neurological complications [10]. The present case therefore reinforces that HSVE should always be considered in febrile patients presenting with acute hemiplegia, aphasia, encephalopathy, and cortical diffusion restriction extending beyond vascular territories. Early MRI, EEG evaluation, and prompt empirical acyclovir therapy may be both diagnostic and life-saving.

CONCLUSION(S)

The HSVE can closely mimic acute ischemic stroke during the early stages because of abrupt focal neurological deficits and marked diffusion restriction on MRI. Recognition of associated clinical features such as fever, encephalopathy, multifocal cortical involvement beyond vascular territories, and supportive EEG findings is essential to avoid diagnostic delay. Early empirical acyclovir therapy should be initiated whenever HSVE is suspected, even before virological confirmation is available. Prompt clinicoradiological diagnosis and treatment significantly reduce mortality and improve neurological outcomes.

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PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
2. Associate Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
3. Assistant Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
4. Assistant Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
5. Assistant Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Deepak Raj,
No. 40/68, TVK 2nd Link Road, Kodungaiyur, Chennai-118, Chennai,
Tamil Nadu, India.
E-mail: deepakraj.maa@gmail.com

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