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CASE REPORT

A Case of HSV Encephalitis Misdiagnosed as Worsening Psychiatric Condition: A Case Report

Elham Abdulhakim Mohammed, Medhanet Azene Gebeyehu, Muhudin Arusi Dari, Yodit Alemayehu

Department of Internal Medicine, Eka Kotebe General Hospital, St. Peter's Specialized Hospital, ALERT Hospital, Addis Ababa, Ethiopia

Correspondence: Elham Abdulhakim Mohammed, Eka Kotebe General Hospital, Addis Ababa, 1971, Ethiopia, Email elh1990@gmail.com

Abstract: Herpes simplex virus (HSV) encephalitis is uncommon but serious condition that can lead to significant morbidity and mortality if not promptly diagnosed and treated. Atypical presentations are becoming more common with improved diagnostic methods, but they remain underexplored. We present the case of a 29-year-old known patient with schizophrenia for the past four years on olanzapine 5 mg PO BID who initially presented to psychiatry emergency with difficulty in proper communication, auditory hallucination, blurring of the eyes, and depressive symptoms for which olanzapine was discontinued, trifluoperazine 5 mg PO daily was initiated, and lorazepam 1 mg p. o. nocturnal was added after the diagnosis of schizophrenia relapse. Despite the above management, the patient presented with abnormal body movements characterized by up-rolling of the eyes, drooling of saliva, jaw jerking followed by repetitive flexion and extension of the upper and lower extremities, and postictal loss of consciousness. He also had low-grade fever and headache for 5 days. The patient was started on phenobarbitone 100 mg PO nocturnal and escalated. However, the frequency of seizures increased. Clinical evaluation, MRI, and EEG eventually confirmed HSV encephalitis. The patient was administered acyclovir 500 mg intravenously every 8 hours for 21 days. After antiviral therapy, the patient was discharged with antiepileptic and antipsychotic medications 18 days after the seizure-free period with significant improvement. Here, we illustrate that HSV encephalitis can present in unfamiliar manner and reinforce the need for a low index of suspicion and early empirical use of acyclovir until definitive laboratory test results are available.

Keywords: schizophrenia, status epilepticus, viral encephalitis

Introduction

Herpes Simplex Virus Encephalitis (HSVE) is uncommon but critical condition primarily caused by Herpes Simplex Virus type 1 (HSV-1), though HSV-2 can also be responsible. The literature surrounding HSVE is vast, primarily focusing on diagnostic criteria, clinical presentations, and the typical management of the disease. Major and minor criteria were used to diagnose encephalitis and encephalopathy, respectively.[1] Major is required and includes patients presenting with change in mentation (defined as decreased or altered level of consciousness, lethargy, or personality change) lasting ≥24 hours with no alternative cause identified. Minor criteria are fever ≥38°C of 72 hours at presentation, abnormal body movement (focal or generalized) not fully attributable to a pre-existing seizure disorder, new onset of focal neurologic deficit, cerebrospinal fluid analysis of WBC count ≥5 mm3, structural brain abnormality on neuroimaging showing evidence for encephalitis that is either new from prior studies or appears acute in onset, abnormal electroencephalography finding suggestive of encephalitis and not attributable to another cause.[1] The clinical manifestations of HSVE include headache, lethargy, confusion, behavioral changes, and focal neurological (temporal lobe) symptoms.[2] Cognitive and behavioral changes can be misdiagnosed as psychiatric disorders. However, gaps remain in understanding the full spectrum of clinical presentations and the subtle early indicators of HSVE, which can often lead to delay or misdiagnosis. For example, the overlap between cognitive and behavioural symptoms in HSVE and those of primary psychiatric illnesses poses significant diagnostic challenges.[3]

This case report addresses these gaps by presenting an atypical case of HSVE, where the patient's primary presentation included prominent psychiatric symptoms without the hallmark neurological findings typically associated with the condition. The insights from this case underscore the importance of considering HSVE in the differential

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diagnosis of patients presenting with unexplained psychiatric symptoms, ultimately contributing to a better detection of cases and improving patient outcomes.

Case

We present the case of a 29-year-old known schizophrenia patient for the past four years on olanzapine 5 mg PO twice daily came to the psychiatry emergency department of our hospital with a history of inability to communicate properly, blurring of the eyes, and auditory hallucinations that were not present prior to the current presentation. The initial psychiatric mental state examination showed that the patient was alert and conscious, with good eye contact and dressed casually. He displayed a blunt affect and refused to respond to questions. His symptoms remained persistent upon repeated examinations, during which he exhibited social isolation, poor self-care, mumbling, rigidity, auditory hallucinations, persecutory delusions, and stuttering speech.[4] Based on these findings, he was diagnosed with a relapse of schizophrenia. The treatment plan was to start trifluoperazine 5 mg orally daily and lorazepam 1 mg orally at night for 7 days. He was admitted to the psychiatry ward where he stayed for 110 days and in the meantime developed abnormal body movement characterized by up-rolling of the eyes, drooling of saliva, jaw jerking followed by repetitive flexion and extension of the upper and lower extremities and post-ictal loss of consciousness. He also had low-grade fever and headache of 5 days' duration. For this, he was started on phenobarbitone 100 mg PO nocturnal and escalated. He was also started with anti-meningeal doses of ceftriaxone and vancomycin. Seizure frequency increased despite this and status epilepticus was diagnosed. Medical team was consulted and up on admission to medical ward he was lethargic with a Glasgow Coma Score of E3 V2 M5, he was tachycardic with a pulse rate of 128 bpm and a blood sugar level of 119 mg/dl. Meningeal signs were negative, and there were no localizing signs. MRI of the brain showed T2 prolongation involving right medial and peripheral temporal lobe expansion and right side focal abnormal T2 prolongation associated with volume loss and ex vacuo dilatation of posterior horn of ipsilateral lateral ventricle as seen in Figure 1a and b is coronal T2W image showing T2 prolongation involving right medial and peripheral temporal lobe with expansion. There was also involvement of right insular lobe and right medial inferior frontal lobe and right peripheral posterior temporal and parietal lobes showing no restricted diffusion, cortical and subcortical focal T2 hyperintense lesion in Figure 1c associated with volume loss, and corresponding DWI showed restricted diffusion. It concluded that the right side temporal and posterior inferior frontal lobe herpes encephalitis is indicative of herpes encephalitis.

EEG showed generalized sharp and slow wave complexes on bifrontal leads and ictal discharge characterized by spikes and slow wave ranging for few seconds and also showed abnormal wake EEG tracing due to the presence of interictal epileptiform discharge as depicted in Figure 2 and ictal generalized epileptiform discharges as seen in Figure 3.

The initial WBC count was 12,260, with a neutrophil predominance of 87.7%. Liver function, renal function test results, and serum electrolyte levels were within normal ranges. The STAT-PAK® for HIV testing was nonreactive. Lumbar puncture CSF analysis revealed a cell count of 30 with lymphocyte predominance, protein level of 167 mg/dl,

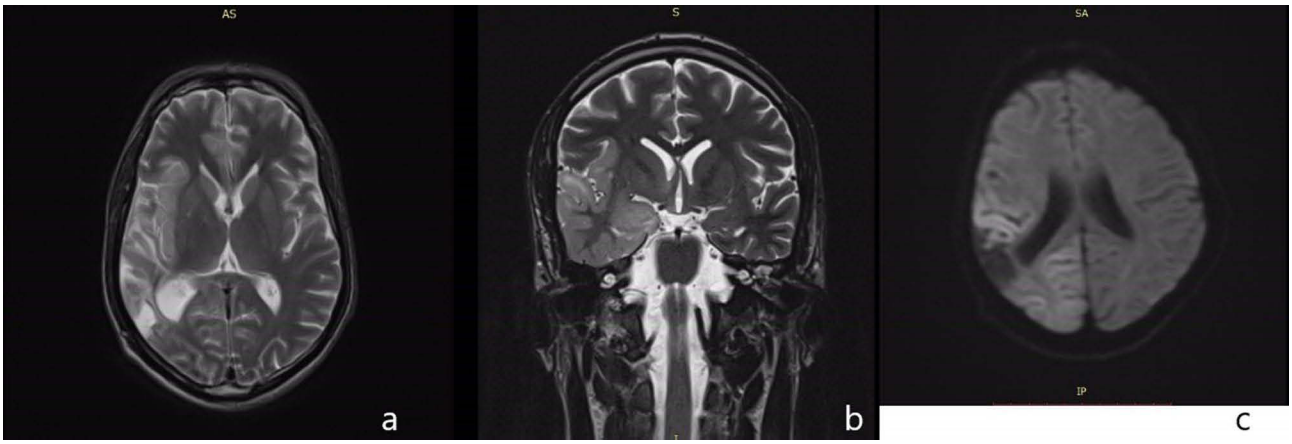

Figure 1 (**a**) Axial T2W (**b**) Coronal T2W (**c**) Axial DWI.

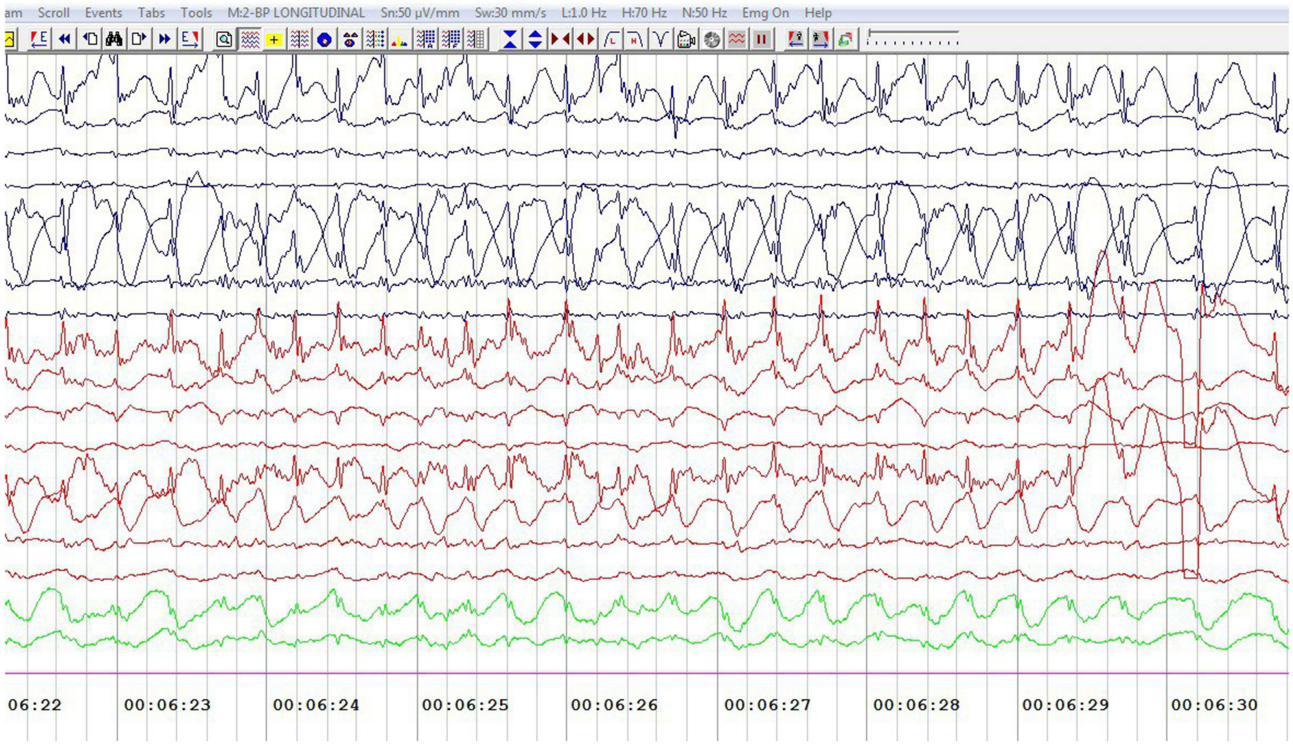

Figure 2 Interictal spike and slow wave.

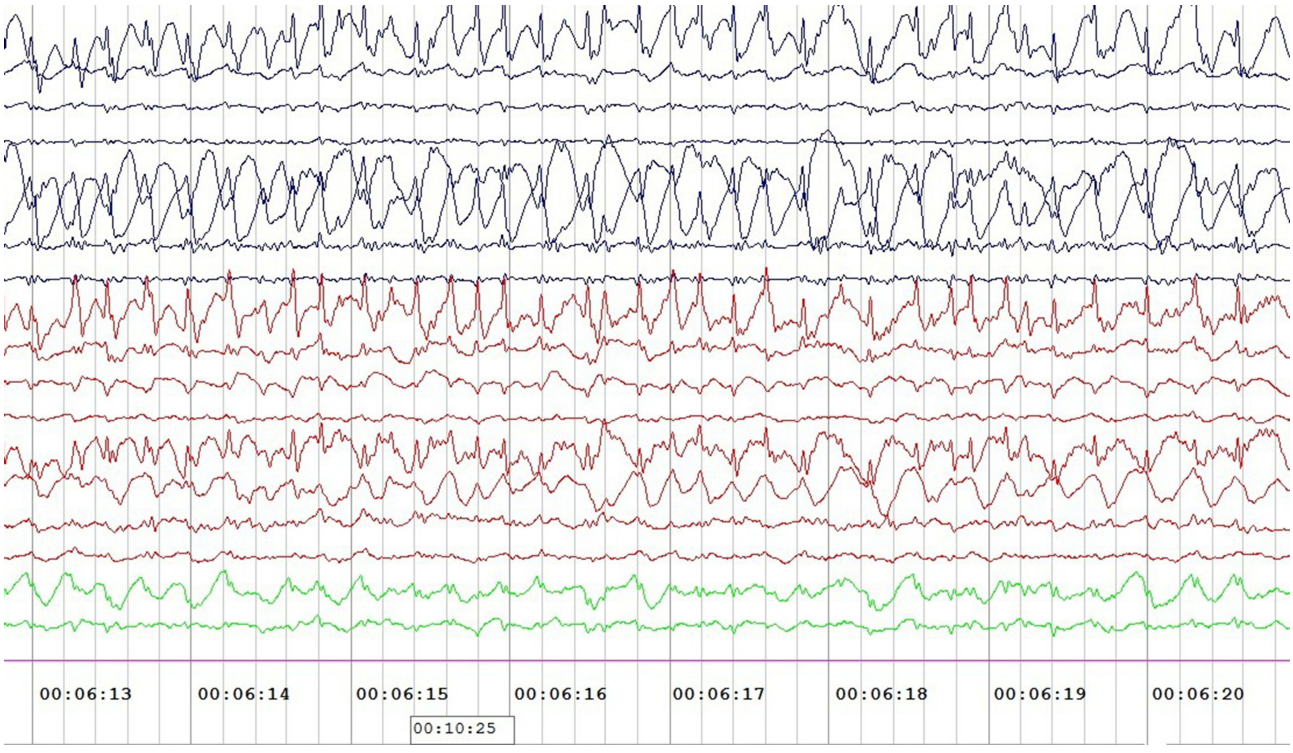

Figure 3 Ictal generalized spike and slow wave.

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glucose 71 mg/dl. No Gram or AFB staining was detected. We were unable to do CSF PCR tests due to financial constraints. The patient was administered acyclovir (500 mg, intravenously every 8 hrs) for 21 days. He was also started on phenytoin 100 mg PO BID and sodium valproate 500 mg PO BID to control seizures. After antiviral therapy, the patient was discharged with phenytoin 100 mg po BID, sodium valproate 500 mg po BID, haloperidol 1 mg po PRN & fluoxetine 20 mg po daily, 18 days after the seizure-free period with significant improvement.

After discharge, he had two visits to our neurology referral clinic within 5 months; he was seizure-free and does self-care activity but did not return to his previous job (teaching) due to his cognitive decline.

Discussion

The HSV encephalitis pathogenesis varies between studies. Usually, in the young, infection is from HSV-1 infection accounting for one-third of cases and the rest are from reactivation of an acquired infection from before which is seen in older patients.[5] Physical examination mostly shows fever, lethargy, decreased level of consciousness, focal neurologic signs, seizures in the majority of patients, and aphasia if the dominant temporal lobe is involved which makes diagnosis relatively early in these patients. The clinical signs tend to fluctuate more in patients with herpes encephalitis and can manifest within several days.[5] There are also a number of patients also demonstrate a quietly progressing prodromal phase at the early stage of the disease, after which they rapidly deteriorate.[6] Some patients show behavioral or psychiatric disorders, such as amnestic or language disturbances, as in our case.[6]

Psychosis frequently occurs in cases of organic delirium and it is a common mistake to assume that patients exhibiting psychotic features have a functional psychiatric disorder, in our case he was on follow-up for schizophrenia for almost 4 years. Indicators of an organic cause include a fast-changing course, no history of psychotic illness in the patient or their family, the presence of non-auditory hallucinations, and the absence of mood-congruent delusions. Health care providers must always keep in mind treatable medical conditions when approaching a patient presenting with a new psychotic episode, because failing to do so can result in preventable adverse outcomes.[6]

The patient's cerebrospinal fluid analysis usually has a normal in 5–10% or slightly elevated protein levels. Glucose levels are normal or slightly low, and there could be an elevated white blood cell count with lymphocytic predominance, which was the finding in our case.[7]

Currently, brain MRI is the diagnostic modality of choice. However, it should be noted that early in the clinical course of the disease, CT scans and MRI may be negative which does not rule out herpes encephalitis. In our case, Brain MRI was suggestive of right temporal and posterior inferior frontal lobe T2 and T1 prolongation, indicating herpes encephalitis and oedema.[8]

The most effective treatment is intravenous acyclovir. With treatment, mortality significantly decreases to less than 30% but is 70% or higher if left untreated. To prevent mortality related to Herpes Encephalitis, empirical treatment should be initiated immediately when HSV encephalitis is considered.[7] Permanent neurologic sequelae appear in more than half of patients, even after treatment, and can differ from mild to severe disability. About 10% of patients experience relapse. In our case, the patient was a teacher, and he could not return to his job because of cognitive deficits post-treatment. Lifelong morbidity includes motor and sensory deficits, aphasia, and amnestic syndromes. Appropriate pharmaceutical therapy and rehabilitation can significantly reduce the medical and psychosocial ramifications of herpes encephalitis and significantly improve the quality of life of many patients.[9] Rehabilitation can be lengthy.

Ethical Approval

Our institution does not require ethical approval for publication of single-case report.

Consent for Publication

Written informed consent for the publication of this case report was obtained from the brother on behalf of the patient.

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Disclosure

The authors report no conflicts of interest in this work.

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