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Meningoencephalitis in a Patient Presenting with a Preliminary Diagnosis of Acute Ischemic Stroke: A Case Report

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Abstract *Central nervous system (CNS) infections may present with acute focal neurological deficits and may be misdiagnosed as ischemic stroke. HSV-1 encephalitis is a recognized stroke mimic, whereas Haemophilus influenzae CNS infection is uncommon in adults. We report a case of HSV-1 encephalitis initially presenting as acute ischemic stroke, with concurrent detection of H. influenzae DNA in cerebrospinal fluid (CSF), highlighting the challenges of interpreting molecular diagnostic findings in CNS infections.*

Keywords: HSV-1; encephalitis; stroke mimic; Haemophilus influenzae; cerebrospinal fluid PCR; central nervous system infection

1. Introduction

Herpes simplex virus type 1 (HSV-1) encephalitis is the leading cause of sporadic fatal viral encephalitis in adults. It typically affects the temporal lobes and may present with altered consciousness, seizures, behavioral changes, and focal neurological deficits. Acute encephalitis remains an important neurological condition associated with significant morbidity in adults, and infectious causes represent a major proportion of identified etiologies [6,7]. Because of these focal manifestations, HSV-1 encephalitis may closely resemble acute ischemic stroke. Early diagnosis and initiation of antiviral therapy are essential, as delayed treatment is associated

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with increased neurological morbidity and mortality. However, diagnosis may be challenging when clinical findings, neuroimaging, and cerebrospinal fluid (CSF) characteristics are not typical. In contrast, *Haemophilus influenzae* has become an uncommon cause of adult CNS infection due to vaccination programs and improved antimicrobial strategies. Nevertheless, invasive infections may still occur and can result in severe neurological complications. The increasing use of molecular diagnostic methods has improved pathogen detection; however, interpretation of positive molecular results may be difficult when culture results and inflammatory CSF findings are limited. We describe a case of HSV-1 encephalitis initially misdiagnosed as acute ischemic stroke, with concurrent CSF detection of *H. influenzae*, highlighting the diagnostic challenges of atypical CNS infections and the importance of integrating molecular results with clinical evaluation.

2. Case Report

A 42 –year – old male patient presented to us with complaints of jaundice, band-like pain in the epigastric region and nausea. The patient had A 66-year-old man with a history of hypertension was transferred to the intensive care unit (ICU) of our hospital from another medical center with a preliminary diagnosis of acute ischemic stroke. His medical history included coronary artery bypass grafting (CABG) performed seven years earlier. According to information obtained from his relatives, the patient developed sudden deterioration in consciousness. He had been monitored for approximately three days at the referring hospital with a presumed diagnosis of acute ischemic stroke complicated by aspiration pneumonia. No meningeal signs were reported during this period. On ICU admission, the patient was somnolent with a Glasgow Coma Scale (GCS) score of 10 (E3M4V3). His body temperature was 38.5–39°C. Oxygen saturation was 92–93% despite oxygen supplementation, and respiratory support was escalated to high-flow nasal cannula (HFNC) therapy (flow 40 L/min, FiO_2 45–50%). Neurological examination revealed left-sided weakness and hemiplegia. No neck stiffness or meningeal irritation signs were present initially. Laboratory evaluation showed a C-reactive protein (CRP) level of 103 mg/L, leukocyte count of 10.4 $\times 10^9$/L, and procalcitonin level of 0.12 ng/mL. Renal and hepatic function tests were normal. HIV, hepatitis B surface antigen, and anti-HCV tests were negative. Initial brain MRI performed at the referring hospital demonstrated hyperintense abnormalities involving the right mediobasal temporal region on FLAIR, T2-weighted, and diffusion-weighted sequences. Because of the acute onset of focal neurological symptoms, these findings were interpreted as acute ischemic infarction. Aspiration pneumonia was initially suspected, and

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ceftriaxone plus metronidazole therapy was started. Due to clinical deterioration, treatment was escalated to meropenem, and metronidazole was discontinued. Blood and sputum cultures were obtained. Teicoplanin was temporarily added because of concern for severe infection and possible infective endocarditis. Transthoracic echocardiography showed no vegetation, structurally normal cardiac valves, and a left ventricular ejection fraction of 40%. Blood cultures remained sterile. Repeat neuroimaging and lumbar puncture were initially delayed because of respiratory instability and the risk associated with patient transport while requiring HFNC support. On hospital day 3, sputum culture yielded multidrug-resistant *Acinetobacter baumannii*, resistant to carbapenems, aminoglycosides, quinolones, and colistin. Progressive respiratory deterioration, persistent fever, increasing inflammatory markers, and hemodynamic instability supported the diagnosis of true hospital-acquired pneumonia rather than colonization. Because therapeutic options were limited, high-dose ampicillin-sulbactam combined with colistin was initiated as salvage therapy despite reported in-vitro resistance. This decision was based on infection severity, the lack of effective alternatives, and the potential role of combination therapy in severe MDR infections. On hospital day 6, meningeal irritation signs developed, including neck stiffness and a positive Kernig sign. Lumbar puncture was performed. CSF analysis revealed clear fluid with low opening pressure. Protein concentration was 81 mg/dL, glucose level was 73 mg/dL (serum glucose 110 mg/dL), leukocyte count was 1–3/mm³, and erythrocyte count was 4–6/mm³. CSF multiplex PCR detected HSV-1 and *Haemophilus influenzae* DNA. CSF cultures remained negative. The minimal CSF inflammatory response was considered possibly related to previous antimicrobial exposure and the patient's age. However, the significance of molecular detection required interpretation together with clinical findings, imaging results, and treatment response. Intravenous acyclovir (10 mg/kg every 8 hours) was initiated, and meropenem was continued to provide antibacterial coverage for possible *H. influenzae* CNS infection. Dexamethasone was administered according to bacterial meningitis treatment recommendations. By hospital day 10, neurological status improved with a GCS score of 13, fever resolved, and inflammatory markers decreased. Respiratory status improved, allowing transition from HFNC to

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conventional oxygen therapy.Follow-up MRI demonstrated bilateral temporal, insular, cingulate, and frontal cortical T2/FLAIR hyperintensities without significant diffusion restriction. The progression from unilateral temporal involvement to bilateral limbic abnormalities favored an inflammatory encephalitic process rather than a vascular territory infarction. Repeat CSF analysis on hospital day 20 showed negative PCR results for HSV-1 and *H. influenzae*. CSF cultures remained sterile.

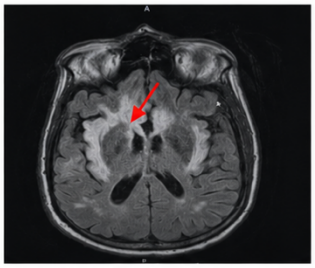

Figure 1.
Axial FLAIR MRI at initial presentation showing hyperintensity in the right mediobasal temporal region (red arrow), initially interpreted as ischemic infarction.

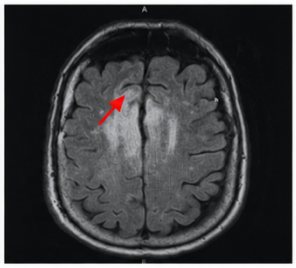

Figure 2.
Axial FLAIR MRI at initial presentation showing subtle cortical hyperintensity in the right frontal region (red arrow).

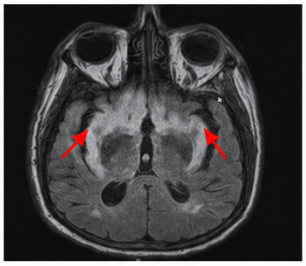

Figure 3.
Follow-up axial FLAIR MRI demonstrating progression of hyperintensity in the bilateral medial temporal lobes and insular regions (red arrows).

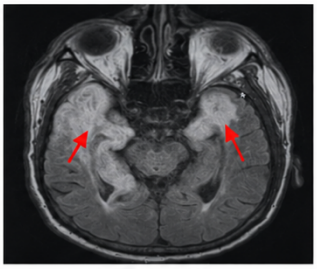

Figure 4.
Follow-up axial FLAIR MRI showing bilateral temporal lobe hyperintensities, more pronounced on the medial aspects (red arrows).

2.1. Limitations

This report has several limitations. First, although *H. influenzae* DNA was detected by CSF multiplex PCR, culture confirmation was not obtained, and the exact clinical contribution of this finding cannot be determined with complete certainty. Second, early lumbar puncture and repeat neuroimaging were delayed because of respiratory instability and HFNC dependency. Third, EEG monitoring could not be performed because bedside EEG was unavailable and the patient's condition did not allow safe transport. Finally, molecular testing was performed using an institutional multiplex PCR assay rather than a commercially available syndromic panel, limiting direct comparison with validated commercial platforms.

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3. Discussion

This case highlights the diagnostic challenges of HSV-1 encephalitis presenting with features initially suggestive of acute ischemic stroke. HSV-1 encephalitis is a recognized stroke mimic because it may cause focal neurological deficits and temporal lobe abnormalities that overlap with cerebrovascular disease. Previous reports have described patients who were initially diagnosed with ischemic stroke before HSV-1 encephalitis was confirmed by CSF PCR [3-5]. Therefore, HSV-1 encephalitis should remain in the differential diagnosis of patients with acute neurological deficits, particularly when the clinical course or imaging findings are not fully consistent with a vascular event. In this patient, the initial MRI findings were interpreted as acute ischemic infarction because of sudden-onset hemiplegia and unilateral temporal involvement. However, follow-up imaging demonstrated bilateral temporal, insular, cingulate, and frontal cortical abnormalities without significant diffusion restriction. This pattern, together with the clinical progression, favored an inflammatory encephalitic process rather than a vascular territory infarction. The diagnosis was further complicated by the absence of early meningeal findings and the inability to perform immediate lumbar puncture and repeat imaging because of respiratory instability. This emphasizes that HSV-1 encephalitis may initially present without classic clinical features and that diagnosis should be guided by the overall clinical picture rather than a single finding. Another important feature of this case was the limited CSF inflammatory response. Despite positive molecular detection, CSF analysis showed minimal pleocytosis, normal glucose concentration, and negative culture results. Previous antimicrobial therapy before lumbar puncture may have reduced culture sensitivity. In addition, older patients with HSV encephalitis may occasionally demonstrate a limited inflammatory CSF response despite active infection [1,2]. Therefore, normal or near-normal CSF findings should not exclude HSV-1 encephalitis when clinical suspicion remains high. The detection of *Haemophilus influenzae* DNA in CSF represented an additional diagnostic challenge. Adult *H. influenzae* meningitis is uncommon; however, invasive CNS infection may still occur and can result in severe neurological complications [10]. In this case, culture confirmation was not obtained, and the limited CSF inflammatory response made interpretation of this finding difficult. The combination of meningeal signs, positive molecular detection, clinical course, and subsequent PCR negativity after treatment suggested that this finding may have represented a true CNS infection, although its clinical significance cannot be established with complete certainty. Nevertheless, molecular diagnostic results require cautious interpretation. Detection of microbial DNA does not always prove active infection and may reflect residual nucleic acid after partial treatment, non-viable organisms, or, rarely, false-positive results. For this reason, molecular findings should be interpreted together with clinical presentation, CSF

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parameters, imaging findings, and response to therapy. Multiplex PCR testing has improved the diagnosis of CNS infections, particularly in patients with atypical presentations or previous antimicrobial exposure [8,9]. In our case, testing was performed using an institutional multiplex PCR assay because a commercially available BioFire FilmArray meningitis/encephalitis panel was not accessible in our setting. The assay was performed under routine laboratory quality procedures, including internal quality controls and contamination prevention measures. However, the absence of culture confirmation and the lack of direct comparison with a commercially validated syndromic panel remain important limitations. The patient's clinical course was additionally complicated by MDR *Acinetobacter baumannii* pneumonia during ICU hospitalization. Progressive respiratory deterioration, persistent fever, increasing inflammatory markers, and hemodynamic instability supported true hospital-acquired pneumonia rather than simple colonization. Due to limited therapeutic options, high-dose ampicillin-sulbactam combined with colistin was used as salvage therapy despite reported in-vitro resistance. This decision was based on infection severity, the lack of effective alternatives, and the potential role of combination therapy in severe MDR *A. baumannii* infections [12]. This case highlights the importance of maintaining diagnostic flexibility in critically ill patients with neurological deterioration. Early consideration of CNS infection and timely CSF molecular testing may prevent delays in appropriate antiviral and antibacterial therapy. A structured diagnostic approach is recommended in patients with suspected encephalitis [11].

4. Conclusion

HSV-1 encephalitis should be considered in elderly patients presenting with acute focal neurological deficits, even when initial imaging suggests ischemic stroke and CSF findings are minimally abnormal. This case demonstrates the diagnostic value of CSF PCR in atypical CNS infections and emphasizes that molecular pathogen detection requires careful interpretation within the clinical context. Early recognition and targeted therapy remain essential to improve outcomes in patients with severe CNS infections.

Declaration of conflicting interests

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