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Miis Akel
Dr. Kiran C. Patel College of Osteopathic Medicine, Nova Southeastern University, USA

Dhruv Ratra
Dr. Kiran C. Patel College of Osteopathic Medicine, Nova Southeastern University, USA

Sahil Shah DO
Larkin Community Hospital, Palm Springs Campus, USA

Sergio Hernandez Borges MD
Larkin Community Hospital, Palm Springs Campus, USA

Corresponding Author:
Miis Akel
Dr. Kiran C. Patel College of Osteopathic Medicine, Nova Southeastern University, USA

HSV encephalitis in the context of substance abuse: A case report

Miis Akel, Dhruv Ratra, Sahil Shah DO and Sergio Hernandez Borges MD

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Abstract
This case report discusses the presentation, diagnosis, and management of a 24-year-old male with no significant past medical history, who presented with substance abuse-related symptoms, including a drug overdose and seizure-like activity. He was later diagnosed with herpes simplex virus type 1 (HSV-1) encephalitis. Diagnostic workup revealed elevated creatine kinase levels indicative of rhabdomyolysis and positive urinary toxicology for THC. Neurology consultation and imaging findings confirmed HSV-1 encephalitis, and the patient was treated with intravenous acyclovir. This case emphasizes the importance of considering infectious causes in patients with a history of substance abuse presenting with neurological symptoms and highlights the complexity of managing dual pathologies in such patients.

Keywords: Herpes simplex virus encephalitis, substance abuse disorder, rhabdomyolysis, acyclovir treatment, neurological symptoms

Introduction
Herpes simplex virus type 1 (HSV-1) encephalitis is the most common cause of sporadic viral encephalitis worldwide, responsible for significant morbidity and mortality. In the United States, its annual incidence is estimated at 1 in 250,000 to 500,000, and without treatment, the condition is associated with mortality rates as high as 70% [1]. Even with antiviral therapy, up to 20% of patients may experience long-term neurological sequelae, including cognitive impairment, seizures, and behavioral changes [2].

HSV-1 encephalitis primarily affects the central nervous system (CNS) through viral reactivation in the trigeminal or olfactory nerve tracts, typically targeting the temporal lobes. This localized infection in the temporal lobe is responsible for many of the hallmark symptoms of the disease, such as altered mental status, aphasia, focal seizures, and memory disturbances [1]. While fever, headache, and confusion are common initial symptoms, the clinical presentation can vary, making early diagnosis challenging. A rapid diagnosis is critical, as early treatment with intravenous acyclovir significantly improves outcomes. Delays in treatment can lead to severe brain damage or death [3].

Magnetic resonance imaging (MRI) is the preferred imaging modality for detecting characteristic temporal lobe abnormalities associated with HSV-1 encephalitis. Hyperintensities in the mesial temporal lobes, orbital frontal regions, or insular cortex are frequently observed, especially in T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences [4]. However, the gold standard for diagnosis remains the detection of HSV-1 DNA in cerebrospinal fluid (CSF) via polymerase chain reaction (PCR). PCR testing is highly sensitive and specific, with reported sensitivity of up to 98% and specificity of 95-100% [5].

The pathogenesis of HSV-1 encephalitis is complex, involving both direct viral cytotoxicity and immune-mediated responses that contribute to neuronal damage. Immune responses, particularly the activation of pro-inflammatory cytokines, can worsen brain damage, leading to edema, necrosis, and hemorrhage in affected regions [6]. As a result, early initiation of antiviral therapy is essential for limiting the extent of neurological damage [1].

Substance abuse can complicate the presentation and diagnosis of HSV-1 encephalitis,

as many of the neurological symptoms of drug intoxication-such as confusion, seizures, and agitation-overlap with those seen in encephalitis. Furthermore, substance use, particularly opioids and cannabinoids, has been associated with altered immune function, potentially increasing susceptibility to infections [7]. This case report discusses a young male with a history of substance abuse who presented with symptoms suggestive of both overdose and viral encephalitis, presenting diagnostic challenges due to the co-existing substance use disorder.

Case Presentation

A 24-year-old male with a history of marijuana and opioid use (Percocet) was brought to the emergency department following an unwitnessed fall. His mother found him awake and scratching his face obsessively. During transportation in the ambulance, the patient became combative, which required sedation with intravenous Keppra and Ativan due to suspected seizure-like activity. His mother noted similar episodes requiring hospitalization in the past year, though there was no known psychiatric history.

Upon arrival at the emergency department, the patient was somnolent, making it difficult to obtain a full history. Initial laboratory studies revealed an elevated creatine kinase (CK) level of 1334 U/L, suggestive of rhabdomyolysis. A toxicology screen was positive for tetrahydrocannabinol (THC). A computed tomography (CT) scan of the brain showed no acute intracranial abnormalities. Given the patient's altered mental state and the possibility of a drug-related neurological event, he was admitted to the internal medicine service for further evaluation.

Neurology was consulted, and a magnetic resonance imaging (MRI) scan of the brain revealed hyperintensity in the left hippocampus [Figure 1-2]. Suspecting herpes simplex virus encephalitis, a cerebrospinal fluid (CSF) analysis was performed, which confirmed the presence of HSV-1 DNA via polymerase chain reaction (PCR) (5). The patient was promptly started on intravenous acyclovir for a 10-day course.

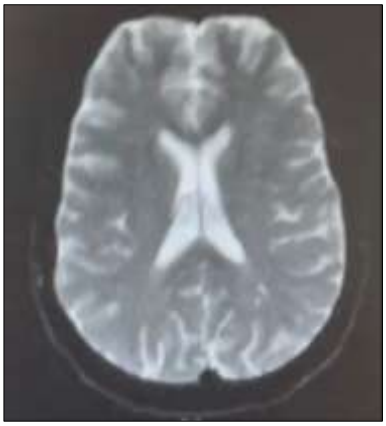

Fig 1: (Axial View, FLAIR)

This axial FLAIR image demonstrates hyperintensity in the periventricular white matter and possibly extending into the surrounding cortical areas. The ventricles are well-demarcated and appear normal in size, without signs of hydrocephalus. The absence of significant mass effect suggests that the hyperintense areas are due to inflammation rather than a mass lesion. FLAIR images are particularly sensitive to changes in the parenchyma, and these findings are suggestive of early viral encephalitis, potentially including HSV.

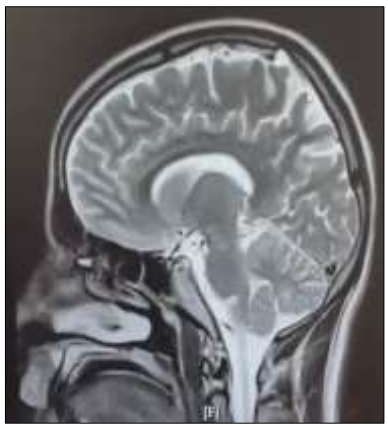

Fig 2: (Sagittal View, T2-weighted)

In this sagittal section, there is a clear definition of the brainstem and cerebellum, which appear unremarkable. The corpus callosum is intact, with normal anatomical structures visible, including the midbrain and pons. No significant lesions are observed in the sagittal plane.

Throughout the hospitalization, the patient was closely monitored for withdrawal symptoms related to his opioid and marijuana use. Addiction Medicine specialists managed his withdrawal using the Clinical Opiate Withdrawal Scale (COWS) and Clinical Institute Withdrawal Assessment for Alcohol (CIWA) scores. Clonazepam was prescribed to manage agitation and opioid withdrawal, sertraline was initiated for depressive symptoms, and olanzapine was used to address persistent agitation. Trileptal was added for seizure prophylaxis.

Given the elevated CK levels and the risk of rhabdomyolysis, nephrology was consulted. The patient was treated with intravenous fluids, and his creatine kinase levels, fluid intake, and output were closely monitored until his levels normalized. His respiratory status was also observed, with oxygen administered intermittently in the step-down unit for potential metabolic acidosis and respiratory compromise due to the overdose.

After completing 10 days of intravenous acyclovir, the patient showed resolution of his encephalitis symptoms. His CK levels returned to normal, and his withdrawal symptoms subsided with appropriate management. The patient was discharged in stable condition with outpatient follow-up for substance use disorder.

Discussion

HSV-1 encephalitis is a life-threatening condition that requires prompt diagnosis and early intervention to prevent significant neurological damage and death. In this case, the clinical picture was further complicated by the patient's history of substance abuse, which can mimic some of the neurological symptoms seen in encephalitis. Early suspicion for HSV-1 encephalitis was raised when imaging revealed typical hippocampal hyperintensity, and PCR testing of the CSF confirmed the diagnosis. Immediate initiation of antiviral therapy with acyclovir was critical in ensuring the patient's recovery [1].

Substance use, particularly marijuana and opioids, can present with neurological and psychiatric symptoms such as confusion, agitation, and in some cases, seizures [7]. This

overlap in symptoms made it challenging to initially differentiate between a substance-related event and an infectious process. THC intoxication, for instance, has been associated with altered mental status and seizures, which further complicated the clinical evaluation [7]. However, the patient's response to antiviral therapy provided confirmation that HSV-1 encephalitis was the primary diagnosis.

The patient's elevated creatine kinase levels, a marker of rhabdomyolysis, were likely due to prolonged seizure activity. Rhabdomyolysis is a known complication in patients with seizures and can lead to acute kidney injury if not properly managed. Early nephrology involvement and aggressive fluid management were key to preventing further complications [6].

This case highlights the importance of a multidisciplinary approach to managing patients with complex medical histories. The involvement of internal medicine, neurology, nephrology, pulmonology, and addiction medicine specialists was essential in providing comprehensive care for this patient. Collaboration between specialties ensured that both the encephalitis and the substance use disorder were addressed, ultimately leading to a positive outcome [1, 5].

Conclusion

This case highlights the challenges of diagnosing and managing HSV encephalitis in a patient with a history of substance abuse. Early recognition and treatment of infectious causes, along with appropriate management of withdrawal symptoms, were crucial in achieving a positive outcome. Multidisciplinary care is essential for patients with co-existing medical conditions, such as substance use disorders, to ensure both the primary condition and any underlying issues are addressed thoroughly. Further research is needed to better understand the relationship between substance abuse and viral encephalitis, particularly in how these factors interact to complicate diagnosis and treatment.

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