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Hepatitis E Virus Infection Presenting With Lower Extremity Weakness: A Case Report and Literature Review

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1. Abstract

Hepatitis E virus (HEV) is a well-recognized cause of acute viral hepatitis, particularly in developing regions. However, autochthonous infections are increasingly reported in developed countries and are often linked to zoonotic transmission. In recent years, neurological complications associated with HEV infection have emerged, including Guillain-Barré syndrome, neuralgic amyotrophy, and transverse myelitis. We report a case of a young adult female presenting with progressive lower extremity weakness and significantly elevated liver enzymes, in whom HEV IgM serology was positive. The patient recovered fully with supportive management, highlighting the importance of considering HEV in patients with unexplained neurological symptoms accompanied by hepatic dysfunction.

2. Introduction

Hepatitis E virus infection is traditionally associated with waterborne outbreaks in endemic regions. However, locally acquired HEV infection is increasingly recognized in industrialized countries, mainly caused by genotype 3 and transmitted zoonotically, particularly from swine reservoirs.

Beyond hepatic disease, HEV has been implicated in a growing spectrum of extrahepatic manifestations, particularly neurological syndromes. Reported complications include Guillain-Barré syndrome, neuralgic amyotrophy, and acute transverse myelitis. Although the exact mechanism remains unclear, immune-mediated nerve injury or direct neurotropic viral effects have been proposed. Because neurological involvement may precede or overshadow hepatic symptoms, recognition of this association is clinically important.

3. Case Presentation

A 26-year-old Hispanic female with a history of alcohol use presented with progressive weakness of both lower extremities. She denied recent travel outside the United States and had no known exposure to livestock or pigs. There was no bowel or bladder dysfunction, and sensation remained intact.

Neurological examination revealed reduced motor strength (3/5) in both lower limbs. Cranial nerves and upper limb strength were normal. Reflexes were preserved, and no sensory deficits were identified.

Laboratory studies demonstrated marked liver enzyme elevation, including significantly increased AST and moderately elevated ALT. Gamma-glutamyl transferase was also elevated. Serological testing confirmed positive hepatitis E virus IgM antibodies. Testing for hepatitis A, B, and C viruses was negative. Additional infectious and autoimmune markers, including CMV, EBV, HSV, and toxoplasmosis, were unremarkable.

Magnetic resonance imaging of the brain and spinal cord showed no structural abnormalities. Cerebrospinal fluid analysis was normal. Abdominal ultrasound revealed hepatomegaly with increased echogenicity, consistent with hepatic inflammation. Liver biopsy demonstrated steatohepatitis without advanced fibrosis.

The patient was managed conservatively with supportive care, as no specific antiviral therapy was indicated. Over a three-month follow-up period, her neurological symptoms gradually resolved. Liver function tests normalized within two months.

4. Discussion

This case highlights an emerging but underrecognized association between HEV infection and neurological dysfunction. While HEV is classically associated with acute hepatitis, increasing evidence suggests that genotype 3 infection can lead to neurological complications, even in patients without travel history or known exposure risks.

The mechanism of neurological injury in HEV infection remains uncertain. Proposed explanations include immune-mediated nerve damage triggered by viral infection, similar to mechanisms seen in other post-infectious neuropathies such as Guillain-Barré syndrome. Alternatively, neurotropic variants of HEV may directly infect neural tissue.

Importantly, neurological symptoms may occur simultaneously with or independently of hepatic manifestations, making diagnosis challenging. In this case, elevated liver enzymes provided an important diagnostic clue leading to serological testing and confirmation of HEV infection.

Most reported cases of HEV-associated neurological disease occur in middle-aged adults, but this case demonstrates that younger patients may also be affected. Recognition of this association is crucial, especially in patients presenting with unexplained neuropathy or weakness accompanied by liver enzyme

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abnormalities.

Management remains primarily supportive, as most immunocompetent patients recover spontaneously. However, chronic infection and severe neurological complications may occur in immunocompromised individuals.

5. Conclusion

Hepatitis E virus infection should be considered in the differential diagnosis of patients presenting with unexplained neurological symptoms, particularly when accompanied by abnormal liver function tests. Early recognition can prevent unnecessary investigations and support appropriate clinical management. Broader awareness of HEV-related neurological manifestations is essential, even in non-endemic regions and in patients without travel history.

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