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Hepatitis E–Associated Lower Limb Weakness: Case Report and Review

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1. Abstract

Hepatitis E virus (HEV) is a well-recognized cause of acute viral hepatitis in developing countries, but autochthonous infections are increasingly reported in developed regions. Although hepatic involvement is the primary manifestation, neurological complications are emerging as important extrahepatic features.

We report a case of a 26-year-old woman who presented with progressive bilateral lower limb weakness associated with elevated liver enzymes. Serological testing confirmed acute HEV infection through positive anti-HEV IgM antibodies. Extensive evaluation excluded other infectious, autoimmune, metabolic, and structural causes of neurological disease. The patient was managed conservatively, with complete neurological recovery and normalization of liver function over a few months.

This case highlights the importance of considering HEV infection in patients with unexplained neurological symptoms, particularly when liver abnormalities are present.

2. Introduction

Hepatitis E virus is an important cause of acute hepatitis worldwide. While traditionally associated with outbreaks in developing countries, locally acquired HEV infections are increasingly recognized in industrialized nations. These cases are most commonly linked to genotype 3, which is considered zoonotic and often associated with exposure to swine reservoirs or contaminated food sources.

In recent years, HEV has also been associated with chronic infection in immunocompromised individuals. Beyond hepatic disease, extrahepatic manifestations are increasingly reported, particularly neurological syndromes such as Guillain-Barré syndrome, neuralgic amyotrophy, and transverse myelitis. The exact mechanism remains uncertain but may involve immune-mediated nerve injury or viral neurotropism.

3. Case Presentation

A 26-year-old Hispanic woman presented with progressive weakness of both lower limbs over several days. She had no significant travel history outside the United States and no known exposure to livestock. Her medical history was notable only for alcohol use.

On examination, motor strength in the lower extremities was reduced to 3/5 bilaterally. Sensory function was intact, and there were no abnormalities in bowel or bladder control. No focal neurological deficits were identified in the upper limbs or cranial nerves.

Cerebrospinal fluid analysis was normal. Magnetic resonance imaging of the brain and entire spinal cord revealed no structural abnormalities or inflammatory lesions.

Laboratory testing demonstrated significant liver enzyme elevation, with markedly increased aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma-glutamyl transferase (GGT). Mild hyperbilirubinemia was also present.

Serological studies confirmed acute hepatitis E infection with positive anti-HEV IgM antibodies. Tests for hepatitis A, B, and C viruses, cytomegalovirus, Epstein–Barr virus, and toxoplasmosis were negative. Autoimmune and metabolic liver disease evaluations, including ceruloplasmin, alpha-1 antitrypsin, and vitamin B12 levels, were within normal limits.

Abdominal ultrasound revealed hepatomegaly with diffuse increased echogenicity, consistent with hepatic inflammation. Liver biopsy showed moderate steatohepatitis.

The patient was treated conservatively without antiviral therapy. Over the following weeks, neurological symptoms gradually improved, and complete recovery was achieved within three months. Liver function tests normalized within two months.

4. Discussion

This case supports the growing evidence that HEV infection may present with neurological complications in addition to hepatic disease. Autochthonous HEV infection in developed countries is most often caused by genotype 3 and is considered zoonotic in origin.

Neurological manifestations associated with HEV infection are increasingly recognized and include Guillain-Barré syndrome, neuralgic amyotrophy, and transverse myelitis. The pathogenesis remains unclear but may involve immune-mediated nerve injury triggered by viral infection or, less commonly, direct neuronal invasion.

In this patient, the presence of acute HEV infection together with progressive lower limb weakness and exclusion of alternative etiologies strongly suggests an HEV-associated neurological syndrome. The temporal relationship between infection and symptom onset further supports this association.

Most reported cases of HEV-related neurological disease are self-limiting, with gradual recovery following resolution of infection, as observed in this case.

5. Conclusion

Neurological complications are an emerging extrahepatic manifestation of hepatitis E virus infection. Clinicians should consider HEV infection in patients presenting with unexplained neurological symptoms, especially when accompanied by abnormal liver function tests.

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Early recognition is important, and HEV testing should be included in the diagnostic evaluation of unexplained hepatitis or neurological syndromes, regardless of travel history or geographic setting.

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