Case Report

Hepatitis E virus-associated Guillain-Barré syndrome: A case report

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ABSTRACT

Guillain-Barré syndrome (GBS) is an acute immune-mediated neuropathy often triggered by infections. Hepatitis E virus (HEV), a common cause of viral hepatitis, has been increasingly linked to extrahepatic manifestations, including neurological syndromes. We report a 31-year-old man who developed progressive limb weakness and respiratory difficulty following acute diarrheal illness. Neurological examination revealed quadriparesis with reduced reflexes; nerve conduction studies showed axonal and demyelinating motor neuropathy. Cerebrospinal fluid analysis demonstrated lymphocytic pleocytosis, and HEV-immunoglobulin M was positive with concurrent hepatic dysfunction. The patient was treated with intravenous immunoglobulin and supportive care, showing gradual recovery. This case highlights HEV-associated GBS as a rare but important diagnostic consideration, particularly in endemic regions. Early recognition of HEV as a trigger in GBS can guide timely management and improve outcomes, underscoring the need for heightened clinical awareness of its extrahepatic complications.

Key words: Case report, Guillain-Barré syndrome, Hepatitis E virus, Intravenous immunoglobulin, Neurological manifestation

Guillain-Barré syndrome (GBS) is an acute inflammatory disorder of the peripheral nervous system caused by a cross-reactive immune response following viral, bacterial, or parasitic infections, a phenomenon known as molecular mimicry [1]. The acute progression of limb weakness, often with sensory and cranial nerve involvement 1–2 weeks after immune stimulation, proceeds to its peak clinical deficit in 2–4 weeks [2]. About two-thirds of adult patients report preceding symptoms of a respiratory or gastrointestinal tract infection within 4 weeks of the onset of weakness, and one-third of cases have no symptoms of a prior infection [3]. Hepatitis E virus (HEV) infection is one of the most common causes of acute viral hepatitis and acute jaundice syndrome worldwide [4]. The clinical presentation of HEV infection could be classified into hepatic and extra-hepatic forms of the disease, with the hepatic form further subdivided into acute hepatitis, fibrosis, or cirrhosis [5]. The extrahepatic manifestation of HEV includes neurological, hematological, and renal disorders [4]. Interestingly, 16% of acute hepatitis E infection involves neurological manifestations, with the highest detection rates found in neuralgic amyotrophy and in GBS [4]. Since HEV infections are asymptomatic in the majority of patients, they might account for some cases of GBS without preceding symptoms of an infection [6]. In addition, in some patients, direct infiltration of the nerve roots due to neurotropic properties of the virus cannot be excluded [7].

Here, we report a case of GBS associated with HEV infection. Although GBS is a well-recognized post-infectious neuropathy, its association with HEV remains underreported and often overlooked. GBS is mostly related to enteric viruses, and it is unlikely to be associated with viruses affecting the liver; hence, GBS related to the HEV is a rare association. This case contributes to the limited literature and highlights the importance of heightened clinical awareness in endemic regions.

CASE REPORT

A 31-year-old gentleman without any known comorbidity presented with a history of diarrhea associated with vomiting for 10 days. He had bilateral lower limb weakness that gradually increased over 7 days and spread to the trunk and upper limbs, associated with breathing difficulties.

On admission, the patient was afebrile with a temperature of 37.6°C, pulse rate of 80/min, blood pressure of 130/80 mmHg, respiratory rate of 18/min, and oxygen saturation of 98% on room air. He was

Access this article online

Received - 16 March 2026
Initial Review - 02 April 2026
Accepted - 23 April 2026

DOI: 10.32677/ijcr.v12i5.8172

Quick Response code

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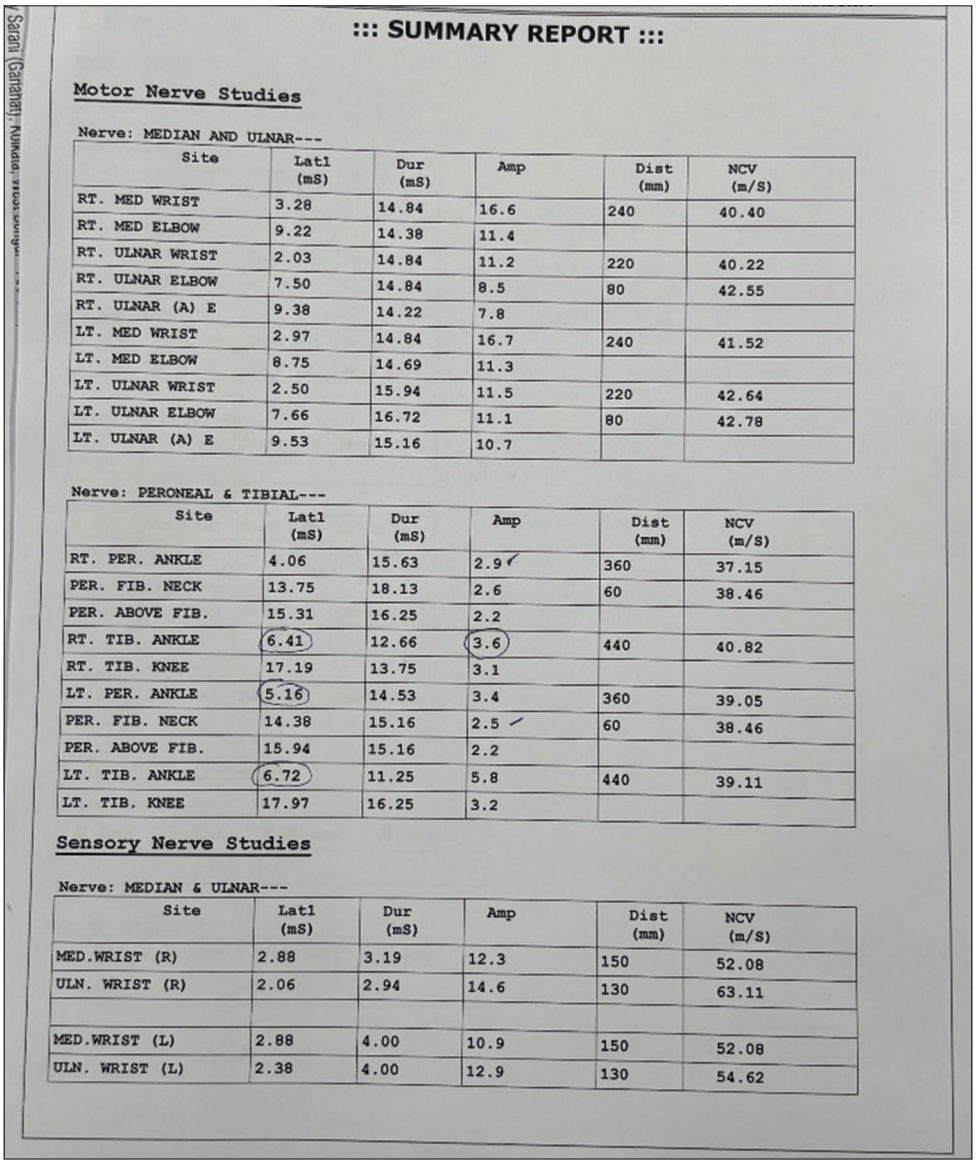
thoroughly examined, and relevant investigations were sent. Physical examination showed normal muscle tone, power 3/5 in all four limbs, and deep tendon reflexes decreased bilaterally, with planters bilaterally equivocal.

A comprehensive viral hepatitis panel for hepatitis A, B, C, D, and E viruses using an enzyme-linked immunosorbent assay was performed. All were negative except for HEV infection, which came positive for HEV-immunoglobulin M (IgM), confirming acute HEV infection. A cerebrospinal fluid (CSF) study was done in the 1st week of illness, which showed 5 cells, glucose – 64 mg/dL, protein – 27 mg/dL, absent coagulum, and lymphocytes-100%, suggesting a non-bacterial inflammatory process. Nerve conduction studies were done in the 2nd week of presentation, which demonstrated mixed axonal and demyelinating motor neuropathy of all four limbs (lower limb > upper limb) with bilateral S1 radiculopathy, consistent with GBS (Fig. 1). In addition, the patient was screened for Scrub typhus IgM, Leptospira IgM, and anti-nuclear antibody, all of which were negative. Liver function tests showed elevated total bilirubin (3.7 mg/dL), elevated direct bilirubin (2.1 mg/dL), elevated levels of aspartate aminotransferase (172 U/L), alanine aminotransferase (125 U/L), and alkaline phosphatase (180U/L). The patient reported no history of blood transfusions, risky sexual behavior, or sharing needles.

He was managed conservatively with intravenous Ig (IVIg) 2 g/kg divided in equal doses over 5 days, IV antibiotics, nebulization, along with other supportive measures, including physiotherapy, in line of the GBS. He gradually improved with the above management and was discharged in a hemodynamically stable condition after 6 days post-admission.

DISCUSSION

HEV infection is endemic in many developing countries, where it is transmitted mainly through the fecal-oral route through contaminated water or food [7]. The virus is well recognized to induce hepatitis; however, neurological syndromes associated with HEV infection cause significant morbidity [8]. Limited data exists regarding the neurological consequences of HEV infection, with most studies originating from India and involving HEV genotype 1 [9]. However, several developed countries



::: SUMMARY REPORT :::

Motor Nerve Studies

Nerve: MEDIAN AND ULNAR---

Site	Lat1 (mS)	Dur (mS)	Amp	Dist (mm)	NCV (m/S)
RT. MED WRIST	3.28	14.84	16.6	240	40.40
RT. MED ELBOW	9.22	14.38	11.4		
RT. ULNAR WRIST	2.03	14.84	11.2	220	40.22
RT. ULNAR ELBOW	7.50	14.84	8.5	80	42.55
RT. ULNAR (A) E	9.38	14.22	7.8		
LT. MED WRIST	2.97	14.84	16.7	240	41.52
LT. MED ELBOW	8.75	14.69	11.3		
LT. ULNAR WRIST	2.50	15.94	11.5	220	42.64
LT. ULNAR ELBOW	7.66	16.72	11.1	80	42.78
LT. ULNAR (A) E	9.53	15.16	10.7		

Nerve: PERONEAL & TIBIAL---

Site	Lat1 (mS)	Dur (mS)	Amp	Dist (mm)	NCV (m/S)
RT. PER. ANKLE	4.06	15.63	2.9	360	37.15
PER. FIB. NECK	13.75	18.13	2.6	60	38.46
PER. ABOVE FIB.	15.31	16.25	2.2		
RT. TIB. ANKLE	6.41	12.66	3.6	440	40.82
RT. TIB. KNEE	17.19	13.75	3.1		
LT. PER. ANKLE	5.16	14.53	3.4	360	39.05
PER. FIB. NECK	14.38	15.16	2.5	60	38.46
PER. ABOVE FIB.	15.94	15.16	2.2		
LT. TIB. ANKLE	6.72	11.25	5.8	440	39.11
LT. TIB. KNEE	17.97	16.25	3.2		

Sensory Nerve Studies

Nerve: MEDIAN & ULNAR---

Site	Lat1 (mS)	Dur (mS)	Amp	Dist (mm)	NCV (m/S)
MED.WRIST (R)	2.88	3.19	12.3	150	52.08
ULN. WRIST (R)	2.06	2.94	14.6	130	63.11
MED.WRIST (L)	2.88	4.00	10.9	150	52.08
ULN. WRIST (L)	2.38	4.00	12.9	130	54.62

Figure 1: Nerve conduction studies

have recently reported an increase in HEV infections (primarily genotype 3) resulting in GBS [9]. In our case, we did not check the genotype of the Hepatitis E virus. A study conducted in the Netherlands found that 10 GBS patients had a higher concentration of anti-HEV IgM antibodies (5.0%) compared to a healthy control (0.5%); with HEV RNA being detected in the blood of three of the patients [10].

The clear mechanisms by which HEV can induce GBS are still unknown, but two possible pathogenesis causes have been proposed according to published studies; one of which is by direct viral damage due to HEV replication in the neurological system, and the other is by indirect immune response, also called molecular mimicry [11]. Current view holds that when infectious organisms, especially those having the same epitopes as the host's peripheral nerves, invade the human, the host initiates an immune response against the foreign infectious organisms and mistakenly attacks the myelin or axon [12].

GBS is an extrahepatic manifestation of HEV infection, which should be suspected in patients with neurological manifestations with increased liver enzymes [4]. In our case, CSF examination demonstrated lymphocytic pleocytosis, although with normal protein levels, which suggested non-bacterial causes of infection and inflammation of the central nervous system. Furthermore, in our patient, nerve conduction studies were suggestive of axonal and demyelinating motor neuropathy of all four limbs. The prognosis of HEV-associated GBS is generally good, with most patients recovering fully or partially within 6 months [7]. Our patient responded well to IVIg, consistent with reports of a favorable outcome. He recovered fully within 21 days and was able to walk without support.

This case highlights the need for clinicians in endemic regions to consider HEV as a potential trigger for GBS, especially in patients with concurrent hepatic abnormalities. The global burden of HEV infections is approximately 20 million cases each year, with 3.3 million symptomatic cases and 44,000 deaths annually [13]. Highest prevalence is seen in South Asia, subSaharan Africa, and parts of East Asia, where poor sanitation and contaminated water are major drivers [13]. In India, HEV accounts for 30–60% of sporadic acute viral hepatitis cases in endemic areas, making it a leading cause of acute viral hepatitis in young adults [13].

Routine screening for HEV in GBS patients may improve diagnostic accuracy and epidemiological understanding. Increasing recognition of HEV-associated neurological disease highlights the importance of preventive measures, including improved sanitation and potential vaccination strategies.

CONCLUSION

HEV-associated GBS is an underrecognized entity that can mimic other infectious or immune-mediated neuropathies. It should be considered in patients presenting with GBS, especially in endemic regions or those with concurrent hepatic dysfunction. Early identification of HEV as a trigger in GBS patients with hepatic abnormalities can guide appropriate management, improve outcomes, and enhance epidemiological surveillance. This case underscores the need for clinicians to maintain a high index of suspicion for HEV in endemic areas and highlights the broader public health importance of preventive measures against HEV transmission.

REFERENCES

1. Fritz-Weltin M, Frommherz E, Isenmann N, Niedermeier L, Csernalabics B, Boettler T, *et al*. Hepatitis E virus as a trigger for Guillain-Barré syndrome. BMC Neurol 2021;21:304.
2. Fokke C, Van Den Berg B, Drenthen J, Walgaard C, Van Doorn PA, Jacobs BC. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain 2014;137:33-43.
3. Willison HJ, Jacobs BC, Van Doorn PA. Guillain-Barré syndrome. Lancet 2016;388:717-27.
4. Ahmed A, EL-Sadig SM, Siddig EE. Guillain-Barre syndrome associated with hepatitis E virus infection: A case report. Clin Case Rep 2023;11:e7863.
5. Willauer AN, Sherman KE. Hepatitis E virus: Has anything changed? Curr Opin Gastroenterol 2023;39:169-74.
6. Webb GW, Dalton HR. Hepatitis E: An underestimated emerging threat. Ther Adv Infect Dis 2019;6:2049936119837162.
7. Rodrigues RA, Sequeira M, Barros F, Alves T, Gonçalves J. Acute hepatitis E-associated Guillain-Barré syndrome. Cureus 2023;15:e48778.
8. Ripellino P, Pasi E, Melli G, Staedler C, Fraga M, Moradpour D, *et al*. Neurologic complications of acute hepatitis E virus infection. Neurol Neuroimmunol Neuroinflamm 2019;7:e643.
9. Kamar N, Bendall RP, Peron JM, Cintas P, Prudhomme L, Mansuy JM, *et al*. Hepatitis E virus and neurologic disorders. Emerg Infect Dis 2011;17:173-9.
10. Van Den Berg B, Van Der Eijk AA, Pas SD, Hunter JG, Madden RG, Tio-Gillen AP, *et al*. Guillain-Barré syndrome associated with preceding hepatitis E virus infection. Neurology 2014;82:491-7.
11. Liu H, Ma Y. Hepatitis E virus-associated Guillain-Barre syndrome: Revision of the literature. Brain Behav 2020;10:e01496.
12. Hughes RA, Cornblath DR. Guillain-Barré syndrome. Lancet 2005;366:1653-66.
13. World Health Organization. Global Hepatitis Report 2024: Action for Access in Low- and Middle-Income Countries. Geneva: WHO; 2024.

Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Biswas R, Chakraborty N. Hepatitis E virus-associated Guillain-Barré syndrome: A case report. Indian J Case Reports. 2026;12(5):305-307.