Infection and Drug Resistance

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

A Case Report of Rare MOG Antibody-Associated Optic Neuritis Related to Hepatitis E Virus Infection

Xiou Yang[1,*], Xiaojing Zhang[2,*], Wenwen Yu[1], Changjiao Hu[1], Sitong Fan[1], Yonghu Zhang[1], Zenghua Xie[1], Hongyan Ye[1], Chao Ji[1], Yafeng Zheng[2], Wei Gai[2], Fang Wang[1]

[1]Infectious Disease Department, Beilun People's Hospital, Beilun Branch of the First Affiliated Hospital of Zhejiang University, Ningbo, Zhejiang, People's Republic of China; [2]WillingMed Technology (Beijing) Co., Ltd, Beijing, People's Republic of China

*These authors contributed equally to this work

Correspondence: Fang Wang, Infectious Disease Department, Beilun People's Hospital, Beilun Branch of the First Affiliated Hospital of Zhejiang University, Ningbo, Zhejiang, People's Republic of China, Email f8023f1314@126.com

Infection and Drug Resistance downloaded from https://www.dovepress.com/
For personal use only.

Abstract: Hepatitis E infection is a highly prevalent viral infection, with neurological manifestations being the most common extrahepatic symptoms. However, cases of hepatitis E virus (HEV) infection presenting with optic neuropathy symptoms remain scarce. We reported a case of a 49-year-old male who presented with fatigue, poor appetite, slightly yellowish urine, and fever. The patient had no significant past medical history. Upon admission, liver and kidney function tests were abnormal, and serological testing was positive for HEV IgM antibodies. After targeted therapy for liver protection and jaundice reduction, liver and kidney function indices returned to normal. However, after discharge, the patient gradually developed blurred vision. Cranial magnetic resonance imaging (MRI) revealed bilateral optic nerve swelling with abnormal signals and slight accumulation of fluid in the optic nerve sheath. Cerebrospinal fluid (CSF) testing was positive for HEV antibodies and Myelin oligodendrocyte glycoprotein (MOG) antibody IgG, after confirm no other related factors causing optic neuritis present, so it is considered as MOG antibody-associated optic neuritis, and this was confirmed through effective treatment. After five days of immunoglobulin therapy, along with corticosteroids and neurotrophic drugs, the bilateral optic nerve swelling improved, and the fluid accumulation in the optic nerve sheath reduced. This case indicates that HEV infection may lead to MOG antibody-associated optic neuritis. Although there are currently no specific drugs for treating HEV infection, immunotherapy can play a crucial role.

Keywords: hepatitis E virus, MOG antibody-associated disease, optic neuritis, immunotherapy, immunocompetent

Introduction

Hepatitis E virus (HEV) is an unenveloped, positive-sense single-stranded RNA virus belonging to the Hepeviridae family. It can cause acute hepatitis, which is usually self-limiting and asymptomatic or mild, requiring no specific treatment, and the virus can be spontaneously cleared within 4–6 weeks.[1,2] Individuals with compromised immune systems are often unable to clear HEV through their immune responses in a short time, potentially progressing to chronic hepatitis.[3] Most HEV infections are asymptomatic, with less than 5% of patients developing typical acute hepatitis symptoms such as fever, stomach pain, nausea, vomiting, and jaundice.[4,5] HEV infection can present with multisystem and multi-organ extrahepatic manifestations, including the nervous system, kidneys, hematologic system, and pancreas.[6] Neurological damage is the most common extrahepatic manifestation of HEV infection,[7] with neuralgic amyotrophy (primarily bilateral muscle atrophy) and Guillain-Barré syndrome (GBS) being the most common, followed by myalgia and encephalitis/myelitis.[8,9] To date, only one case report has associated HEV infection with optic neuropathy. The patient initially experienced blurred vision and later developed complete vision loss, ultimately diagnosed as acute HEV infection leading to central nervous system multifocal disseminated inflammatory lesions.[10] Cases of HEV infection presenting with optic neuropathy symptoms remain scarce. Here, we present a rare case of Myelin oligodendrocyte glycoprotein (MOG) antibody-associated optic neuritis related to HEV infection in an immunocompetent patient.

https://doi.org/10.2147/IDR.S570904
Received: 28 September 2025
Accepted: 29 December 2025
Published: 14 January 2026

© 2026 Yang et al. This work is published and licensed by Dove Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.php and incorporate the Creative Commons Attribution – Non Commercial (unported, v4.0) License (http://creativecommons.org/licenses/by-nc/4.0/). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php).

Case Presentation

The patient is a 49-year-old male with a healthy lifestyle and regular physical activity. On April 17, 2024, he experienced fatigue, poor appetite, slightly yellowish urine, and a low-grade fever of 37.7°C without any apparent cause. Blood tests showed elevated liver function indices: alanine aminotransferase (ALT) at 3337 U/L (Ref: 9–50), aspartate aminotransferase (AST) at 2769 U/L (Ref: 15–40), and total bilirubin (TBIL) at 46 μmol/L (Ref: ≤ 23) (Figure 1). Urinalysis was abnormal with positive results for urine bilirubin (1+) and urobilinogen (1+). On April 19, serological testing for hepatitis A, C, D, and E antibodies revealed positive IgM antibodies for hepatitis E virus (HEV), leading to a diagnosis of acute hepatitis E. The patient was admitted for the first time on April 20 for treatment.

The patient, with no significant past medical history, exhibited clear consciousness, low energy, poor appetite and sleep, normal bowel movements, yellowish urine, and unspecified weight changes since the onset of symptoms. During hospitalization, coagulation function was normal. Treatment included magnesium isoglycyrrhizinate, ademetionine, bicyclol tablets, and ursodeoxycholic acid tablets. ALT, AST, and TBIL levels gradually decreased to normal (Figure 1). By April 30, the patient's clinical symptoms improved, and he was discharged in good condition.

On May 3, the patient was readmitted due to gradually worsening blurred vision, accompanied by posterior ocular pain for four days. One day before admission, the ophthalmologic examination revealed visual acuity vision of the right eye (VOD) 0.6, vision of the left eye (VOS) 0.6, intraocular pressure Tod: 18 mmHg, Tos: 20 mmHg. Both eyes showed no conjunctival congestion, vitreous opacity, optic disc edema with blurred margins, dilated veins, and flat retinas. Fundus photography showed bilateral optic disc edema. Optic disc optical coherence tomography (OCT) showed thickened nerve fiber layer, suggesting optic disc edema. Relative afferent pupillary defect (RAPD) was negative. Upon admission, his pulse was 62 beats/min (Ref: 60–100), respiratory rate 17 breaths/min (Ref: 12–20), blood pressure 128/74 mmHg, and body temperature 36.7°C. The patient was conscious, with no significant conjunctival congestion or eyelid edema, a soft and non-tender abdomen, and no palpable liver or spleen below the ribs. There was no edema in the lower extremities. Neurological examination showed equal, round pupils with sensitive light reflexes, and restricted bilateral eye abduction. Other neurological examinations, including cranial nerves, sensory nerves, and motor nerves, were normal. Blood tests, urinalysis, coagulation function, D-dimer, and erythrocyte sedimentation rate (ESR) were normal. Tests for homocysteine, C-reactive protein (CRP), glucose, cardiac enzymes, amylase, calcium, magnesium, and phosphorus were also normal. Liver and kidney function [ALT: 91 U/L (Ref: 9–50 U/L), GGT: 192 U/L (Ref: 10–60 U/L), direct bilirubin: 8.4 μmol/L (Ref: ≤8.0 μmol/L), creatinine: 47 μmol/L (Ref: 59–104 μmol/L), uric acid: 178 μmol/L (Ref: 208–428 μmol/L)], lipid analysis (total cholesterol: 6.02 mmol/L, LDL cholesterol: 3.86 mmol/L, apolipoprotein B: 1.11 g/L), and immunological tests (immunoglobulin M: 3.17 g/L) showed some abnormalities. Tests for TP-TRUST, *Treponema pallidum* antibodies, HIV antibodies, glycated hemoglobin, rheumatoid factor, antistreptolysin O, brain natriuretic peptide (BNP), thyroid function, fecal analysis, and fungal examination were

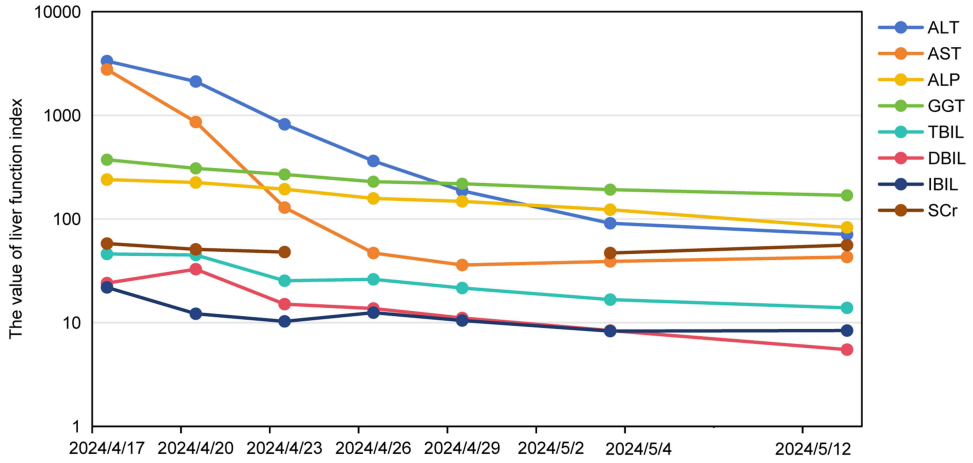

Figure 1 Changes of liver function indexes in patients during hospitalization. ALT: alanine aminotransferase (Ref: 9–50 U/L); AST: aspartate aminotransferase (Ref: 15–40 U/L); ALP: alkaline phosphatase (Ref: 45–125 U/L); GGT: γ-glutamyl transferase (Ref: 10–60 U/L); TBIL: total bilirubin (Ref: ≤23.0 μmol/L); DBIL: direct bilirubin (Ref: ≤8.0 μmol/L); IBIL: indirect bilirubin (Ref: 3.4–13.7 μmol/L); Scr: serum creatinine (Ref: 59–104 μmol/L).

https://doi.org/10.2147/IDR.S570904

normal. Hepatitis B tests showed positive results for surface antibody, e antibody, and core antibody. Tumor markers were negative. Cervical spine magnetic resonance imaging (MRI) was normal. Lumbar puncture and cerebrospinal fluid (CSF) analysis showed clear, colorless fluid with positive Pandy's test (2+), red blood cells: 102/μL, white blood cells: 10/μL, neutrophils: 32%, lymphocytes: 63%, monocytes: 5%. CSF biochemistry showed protein: 971.0 mg/L, glucose: 4.25 mmol/L, chloride: 123.3 mmol/L; lactate dehydrogenase (LDH): 26 U/L, Adenosine deaminase (ADA): 0 U/L. *Cryptococcal*, bacterial cultures, and acid-fast staining were negative. Sarcoid optic neuropathy may present with manifestations such as hilar lymphadenopathy or skin nodules, potentially elevated serum Angiotensin Converting Enzyme (ACE) levels, granulomas around the optic nerve on imaging. This patient showed no hilar lymphadenopathy or skin nodules. Furthermore, MRI revealed no perineural granulomas, leading to the exclusion of sarcoid optic neuropathy. Additionally, the absence of necrotic lesions on MRI ruled out toxoplasmic optic neuropathy as a primary consideration. Nevertheless, CSF demyelinating antibody testing revealed positive MOG IgG antibody (Live Cell-Based Assay, normal Ref: negative) at 1:10 titer, and oligoclonal bands were negative. HEV IgM antibody [Hepatitis E Virus IgM Antibody Detection Kit (Enzyme-Linked Immunosorbent Assay) of Wantai Biopharm, and Shanghai Kehua Bio-Engineering] was positive. Cranial MRI showed bilateral optic nerve swelling with abnormal signals and slight fluid accumulation in the optic nerve sheath, consistent with optic neuritis (Figure 2A). MOG antibody-associated optic neuritis, likely related to HEV infection, was considered.

The patient received methylprednisolone 500 mg daily (with gradual tapering after symptom relief), human immunoglobulin 25 g daily for five days, neurotrophic treatment (mecobalamin injection 1 mL daily, thiamine tablets 25 mg three times daily), gastric protection (rabeprazole tablets 20 mg daily), calcium supplementation (calcium carbonate D3 tablets 600 mg daily), and comprehensive liver protection and electrolyte balance treatment.

After treatment, the patient's visual acuity improved to VOD: 0.8, VOS: 0.8 on May 9, OCT showed normal result. His optic disc edema had reduced, and visual field defects showed significant improvement compared to previous findings, and the symptoms of eye pain have improved. A lumbar puncture on May 10 showed decreased CSF protein

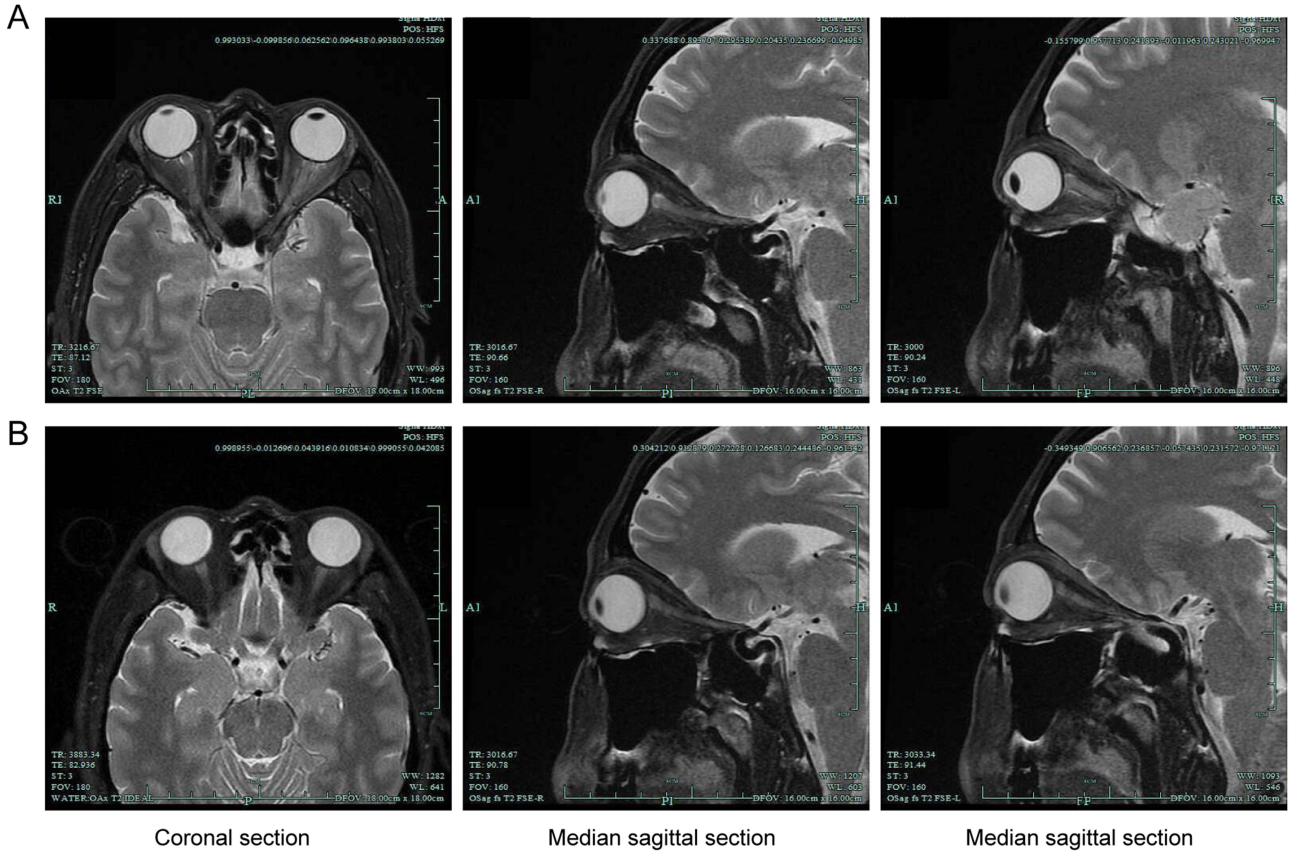

Figure 2 The patient's imaging results. (**A**) The result of cranial MRI on May 2. (**B**) The result of orbital MRI on May 13.

https://doi.org/10.2147/IDR.S570904

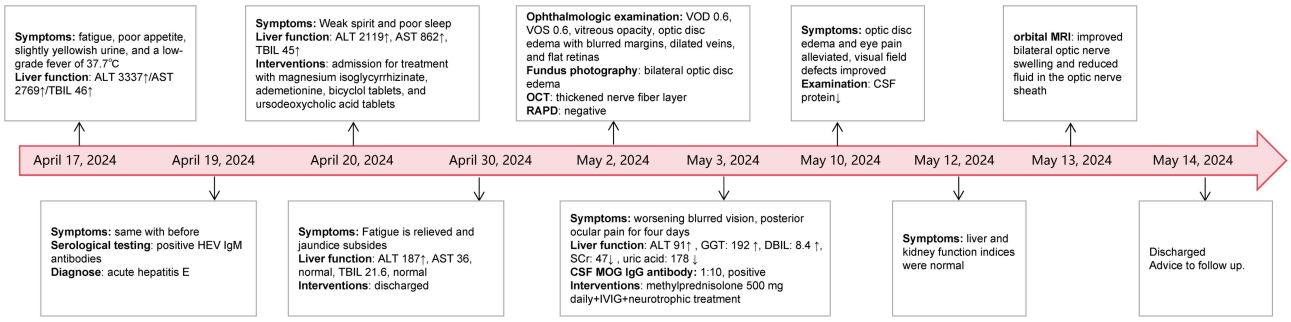

Figure 3 The clinical course of the patient. ALT: alanine aminotransferase (Ref: 9–50 U/L); AST: aspartate aminotransferase (Ref: 15–40 U/L); GGT: γ-glutamyl transferase (Ref: 10–60 U/L); TBIL: total bilirubin (Ref: ≤23.0 μmol/L); DBIL: direct bilirubin (Ref: ≤8.0 μmol/L); Scr: serum creatinine (Ref: 59–104 μmol/L).
Abbreviations: HEV, hepatitis E virus; VOD, vision of the right eye; VOS, vision of the left eye; OCT, optical coherence tomography; RAPD, relative afferent pupillary defect; CSF, cerebrospinal fluid; MOG, Myelin oligodendrocyte glycoprotein; IVIG, intravenous immunoglobulin; MRI, magnetic resonance imaging.

levels. By May 12, liver and kidney function indices were normal. An orbital MRI on May 13 compared to the May 3 cranial MRI showed improved bilateral optic nerve swelling and reduced fluid in the optic nerve sheath (Figure 2B). The patient's symptoms improved, and he was discharged on May 14 with advice to follow up in the neurology outpatient clinic. As of this writing, the patient has no discomfort and is fully recovered. The clinical course of the patient were showed in Figure 3.

Discussion

Neurological involvement in HEV infection is not uncommon, but many aspects remain unclear. Known neurological conditions associated with HEV infection include radiculopathies and plexopathies (Guillain-Barré syndrome, neuralgic amyotrophy, and meningoradiculitis), central nervous system disorders (meningoencephalitis, cerebral ischemia, seizures or epilepsy, transverse myelitis), neuropathies (polyneuritis, peripheral neuropathies, idiopathic facial nerve palsy, oculomotor nerve palsy, vestibular neuritis), and neuromuscular junction and muscle diseases (myositis, myasthenia gravis).[11,12] This study reports a rare case of MOG antibody-associated optic neuritis related to HEV infection, providing additional reference for neurological complications in HEV infection.

MOG is a minor component of the outermost layer of the myelin sheath in the central nervous system (CNS), expressed on the surface of oligodendrocytes, contributing to myelin adhesion and integrity.[13,14] MOG antibody-associated disease (MOGAD) is a CNS autoimmune disorder diagnosed by positive serum MOG-IgG antibodies and characteristic pathological features.[15] The exact pathogenesis remains unclear, with some suggesting infections as initial triggers inducing autoimmunity.[16] However, no specific pathogens have been identified, and many MOGAD patients do not report infection-related antecedent symptoms.[16] In the pathogenesis of MOGAD, MOG-specific B cells, plasma cells, and their produced antibodies (anti-MOG antibodies) play a central role. Research indicates that these MOG-specific B cells and antibodies can activate effector T cells via antigen-presenting cells within the central nervous system.[17,18] Subsequently, they cross the blood-brain barrier, bind to MOG antigens expressed on target structures, and initiate a series of pathogenic processes.[19–22] Specifically, anti-MOG antibodies (IgG1) bind to MOG expressed on the surface of myelin and oligodendrocytes, leading to myelin damage and demyelination through mechanisms such as antibody-dependent cellular cytotoxicity or complement activation.[19,20] Concurrently, activated MOG-specific B cells and plasma cells further activate CD4+ T cells and macrophages, promoting the release of pro-inflammatory cytokines such as IL-6, IL-17, and TNF-α, which exacerbates inflammatory responses and myelin injury.[21,22] The characteristic pathological hallmarks of MOGAD include perivenous and confluent white matter demyelination, MOG-dominant myelin loss, intracortical demyelination, inflammatory infiltrates predominantly composed of CD4+ T cells and granulocytes, complement deposition within active lesions, partial axonal preservation, and reactive gliosis. These pathological features are further supported by the presence of MOG-specific CD4+ T cells within patient lesions, confirming their critical role in disease pathogenesis.[21,22]

MOGAD can present as various clinical syndromes, including acute disseminated encephalomyelitis (ADEM), autoimmune encephalitis, optic neuritis, and transverse myelitis (TM). Optic neuritis is the most common initial manifestation in adults (50% of cases), and about 80% of MOGAD patients develop optic neuritis at some point, characterized by varying degrees of vision loss and almost always accompanied by eye pain, often exacerbated by eye movement.[23,24] Studies suggest that MOG antibody-associated optic neuritis may be related to vascular damage, with MOG antibodies potentially causing retinal destruction and degeneration through perivascular inflammation rather than direct damage to retinal ganglion cells and their axons.[25]

In this case, the patient was diagnosed with acute hepatitis E based on positive serological HEV antibodies, with normalized liver and kidney function indices after symptomatic treatment. However, the patient subsequently developed blurred vision, with no significant eyelid congestion or edema, but restricted bilateral eye abduction. Serological and CSF tests were positive for HEV, and CSF MOG antibody testing was also positive. Cranial MRI showed bilateral optic nerve swelling with abnormal signals and slight fluid accumulation in the optic nerve sheath (Figure 2A), consistent with MOG antibody-associated optic neuritis, likely related to acute HEV infection. Due to limited testing capabilities at our hospital, HEV RNA detection was not available, which constitutes a limitation of this study as it resulted in insufficient evidence. However, during the period when the patient exhibited symptoms of HEV infection and optic neuritis, no other potential causative factors for optic neuritis were identified. Moreover, the patient's optic neuritis symptoms significantly improved with symptomatic therapy, providing supportive evidence for the association between HEV infection and optic neuritis.

Studies have shown that hepatitis-related viral infections can stimulate immune responses, leading to abnormal autoantibodies and various autoimmune diseases.[26,27] HEV is typically an acute infection, and the relationship between HEV and autoimmunity remains controversial. However, HEV infection has been associated with peripheral nervous system autoimmune diseases, such as Guillain-Barré syndrome.[28,29] Additionally, HEV infection is linked to central nervous system inflammatory diseases, with some patients developing vision loss following HEV infection.[10] These findings suggest that HEV infection may be associated with autoimmune diseases and optic neuritis. This case provides valuable evidence for exploring the relationship between HEV and autoimmunity.

Existing studies have extensively explored the association between infections and MOGAD, indicating that various pathogenic infections-including viral, bacterial, *mycoplasma*, and *chlamydial* infections-may be linked to the onset of the disease. Common clinical manifestations include optic neuritis, myelitis, and acute disseminated encephalomyelitis.[30] Viruses implicated in MOGAD comprise SARS-CoV-2, human immunodeficiency virus (HIV), varicella-zoster virus, human herpesvirus type 6, Zika virus, mumps virus, herpes simplex virus (genital), adenovirus, rubella virus, and dengue virus.[30] Pathogen-induced autoimmunity is thought to arise through mechanisms such as molecular mimicry, bystander activation, superantigen activity, and epitope spreading.[31,32] Among these, molecular mimicry is one of the most widely cited theories. It occurs when pathogen-derived peptides share structural homology with human peptides, leading to the production of cross-reactive antibodies and effector immune cells that can target host tissues via autoimmune mechanisms.[33] In the context of MOGAD, pathogens including SARS-CoV-2, *Mycoplasma*, influenza A virus, varicella-zoster virus, and *Chlamydia* have been suggested to trigger the production of anti-MOG antibodies through molecular mimicry.[33–36] In the present case, CSF demyelinating antibody testing revealed a positive result for MOG-IgG, suggesting that HEV infection may also have induced MOGAD via a molecular mimicry pathway.

Acute HEV infection is generally self-limiting, with most patients fully recovering. During the acute phase, patients should rest, maintain a light diet, and receive hepatoprotective, transaminase-lowering, and jaundice-reducing medications, typically without antiviral treatment. For this case, after diagnosing acute hepatitis E, the patient was treated with magnesium isoglycyrrhizinate, ademetionine, bicyclol tablets, and ursodeoxycholic acid tablets. Following the diagnosis of MOG antibody-associated optic neuritis, immunotherapy, including steroids and neurotrophic drugs, was administered, resulting in improvement and discharge.

Conclusion

This report describes a rare case of MOG antibody-associated optic neuritis related to HEV infection in an immunocompetent patient. Unlike previous reports of HEV-related neurological infections, this patient initially presented with elevated liver enzymes, followed by blurred vision. MRI and CSF tests confirmed HEV-related MOG antibody-

associated optic neuritis. This case, along with previous studies, suggests considering HEV infection in patients with acute neurological impairment and significantly elevated liver enzymes. Following a diagnosis of acute hepatitis E, clinicians should be vigilant for potential neurological complications.

Ethics Approval and Consent to Participate

This study was approved by Ethics Committee of Beilun People's hospital, Beilun Branch of the first affiliated hospital of Zhejiang university (2024LP024). The studies were conducted in accordance with the local legislation and institutional requirements. Written informed consent was obtained from the individual for the publication of any potentially identifiable images or data included in this article. This study was approved for publication by the Ethics Committee of Beilun People's hospital, Beilun Branch of the first affiliated hospital of Zhejiang university.

Funding

There is no funding to report.

Disclosure

The authors declare no competing interests in this work.

References

1. Aggarwal R. Hepatitis E: clinical presentation in disease-endemic areas and diagnosis. *Semin Liver Dis*. 2013;33(1):30–40. doi:10.1055/s-0033-1338112
2. Kamar N, Bendall R, Legrand-Abravanel F, et al. Hepatitis E. *Lancet*. 2012;379(9835):2477–2488. doi:10.1016/S0140-6736(11)61849-7
3. Kamar N, Selves J, Mansuy JM, et al. Hepatitis E virus and chronic hepatitis in organ-transplant recipients. *N Engl J Med*. 2008;358(8):811–817. doi:10.1056/NEJMoa0706992
4. Zhang J, Zhang XF, Huang SJ, et al. Long-term efficacy of a hepatitis E vaccine. *N Engl J Med*. 2015;372(10):914–922. doi:10.1056/NEJMoa1406011
5. Zhu FC, Zhang J, Zhang XF, et al. Efficacy and safety of a recombinant hepatitis E vaccine in healthy adults: a large-scale, randomised, double-blind placebo-controlled, Phase 3 trial. *Lancet*. 2010;376(9744):895–902. doi:10.1016/s0140-6736(10)61030-6
6. Pischke S, Hartl J, Pas SD, Lohse AW, Jacobs BC, Van der Eijk AA. Hepatitis E virus: infection beyond the liver? *J Hepatol*. 2017;66(5):1082–1095. doi:10.1016/j.jhep.2016.11.016
7. Kamar N, Abravanel F, Lhomme S, Rostaing L, Izopet J. Hepatitis E virus: chronic infection, extra-hepatic manifestations, and treatment. *Clin Res Hepatol Gastroenterol*. 2015;39(1):20–27. doi:10.1016/j.clinre.2014.07.005
8. Dalton HR, Kamar N, van Eijk JJJ, et al. Hepatitis E virus and neurological injury. *Nat Rev Neurol*. 2016;12(2):77–85. doi:10.1038/nrneurol.2015.234
9. Bazerbachi F, Haffar S, Garg SK, Lake JR. Extra-hepatic manifestations associated with hepatitis E virus infection: a comprehensive review of the literature. *Gastroenterol Rep*. 2016;4(1):1–15. doi:10.1093/gastro/gov042
10. Rahmig J, Grey A, Berning M, et al. Disseminated inflammation of the central nervous system associated with acute hepatitis E: a case report. *BMC Neurol*. 2020;20(1):391. doi:10.1186/s12883-020-01952-5
11. Rawla P, Raj JP, Kannemkuzhiyil AJ, Aluru JS, Thandra KC, Gajendran M. A systematic review of the extra-hepatic manifestations of Hepatitis E Virus infection. *Med Sci*. 2020;8(1):9. doi:10.3390/medsci8010009
12. Jha AK, Kumar G, Dayal VM, Ranjan A, Suchismita A. Neurological manifestations of hepatitis E virus infection: an overview. *World J Gastroenterol*. 2021;27(18):2090–2104. doi:10.3748/wjg.v27.i18.2090
13. Schluesener HJ, Sobel RA, Linington C, Weiner HL. A monoclonal antibody against a myelin oligodendrocyte glycoprotein induces relapses and demyelination in central nervous system autoimmune disease. *J Immunol*. 1987;139(12):4016–4021.
14. Reindl M, Di Pauli F, Rostásy K, Berger T. The spectrum of MOG autoantibody-associated demyelinating diseases. *Nat Rev Neurol*. 2013;9(8):455–461. doi:10.1038/nrneurol.2013.118
15. Banwell B, Bennett JL, Marignier R, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: international MOGAD Panel proposed criteria. *Lancet Neurol*. 2023;22(3):268–282. doi:10.1016/s1474-4422(22)00431-8
16. Gklinos P, Dobson R. Myelin oligodendrocyte glycoprotein-antibody associated disease: an updated review of the clinical spectrum, pathogenetic mechanisms and therapeutic management. *Antibodies*. 2024;13(2):43. doi:10.3390/antib13020043
17. Flach AC, Litke T, Strauss J, et al. Autoantibody-boosted T-cell reactivation in the target organ triggers manifestation of autoimmune CNS disease. *Proc Natl Acad Sci U S A*. 2016;113(12):3323–3328. doi:10.1073/pnas.1519608113
18. Kinzel S, Lehmann-Horn K, Torke S, et al. Myelin-reactive antibodies initiate T cell-mediated CNS autoimmune disease by opsonization of endogenous antigen. *Acta Neuropathol*. 2016;132(1):43–58. doi:10.1007/s00401-016-1559-8
19. Kaneko K, Sato DK, Nakashima I, et al. CSF cytokine profile in MOG-IgG+ neurological disease is similar to AQP4-IgG+ NMOSD but distinct from MS: a cross-sectional study and potential therapeutic implications. *J Neurol Neurosurg Psychiatry*. 2018;89(9):927–936. doi:10.1136/jnnp-2018-317969
20. Mariotto S, Gajofatto A, Batzu L, et al. Relevance of antibodies to myelin oligodendrocyte glycoprotein in CSF of seronegative cases. *Neurology*. 2019;93(20):e1867–e1872. doi:10.1212/wnl.0000000000008479

21. Spadaro M, Winklmeier S, Beltrán E, et al. Pathogenicity of human antibodies against myelin oligodendrocyte glycoprotein. *Ann Neurol*. 2018;84(2):315–328. doi:10.1002/ana.25291
22. Takai Y, Misu T, Kaneko K, et al. Myelin oligodendrocyte glycoprotein antibody-associated disease: an immunopathological study. *Brain*. 2020;143(5):1431–1446. doi:10.1093/brain/awaa102
23. van Pelt ED, Wong YY, Ketelslegers IA, Hamann D, Hintzen RQ. Neuromyelitis optica spectrum disorders: comparison of clinical and magnetic resonance imaging characteristics of AQP4-IgG versus MOG-IgG seropositive cases in the Netherlands. *Eur J Neurol*. 2016;23(3):580–587. doi:10.1111/ene.12898
24. Jeyakumar N, Lerch M, Dale RC, Ramanathan S. MOG antibody-associated optic neuritis. *Eye*. 2024;38(12):2289–2301. doi:10.1038/s41433-024-03108-y
25. Yao Y, Li X, Xu Y, et al. The difference of the retinal structural and microvascular characteristics in patients with MOGAD-ON and AQP4-ON. *BMC Neurol*. 2022;22(1):323. doi:10.1186/s12883-022-02848-2
26. Maslennikov R, Ivashkin V, Efremova I, Shirokova E. Immune disorders and rheumatologic manifestations of viral hepatitis. *World J Gastroenterol*. 2021;27(18):2073–2089. doi:10.3748/wjg.v27.i18.2073
27. Elfert KA, Qasim HM, Faisal MM, et al. Hepatitis E Viral association with autoimmune hepatitis: a viral trigger or cross-reactivity. *Case Rep Gastroenterol*. 2021;15(1):115–122. doi:10.1159/000509494
28. Kamar N, Bendall RP, Peron JM, et al. Hepatitis E virus and neurologic disorders. *Emerg Infect Dis*. 2011;17(2):173–179. doi:10.3201/eid1702.100856
29. Zheng X, Yu L, Xu Q, Gu S, Tang L. Guillain-Barre syndrome caused by hepatitis E infection: case report and literature review. *BMC Infect Dis*. 2018;18(1):50. doi:10.1186/s12879-018-2959-2
30. Xiang H, Cai M. Infections combined with myelin oligodendrocyte glycoprotein antibody-associated disease: a case report and systematic review of the literature. *Cureus*. 2024;16(10):e71229. doi:10.7759/cureus.71229
31. Vanheusden M, Broux B, Welten SPM, et al. Cytomegalovirus infection exacerbates autoimmune mediated neuroinflammation. *Sci Rep*. 2017;7(1):663. doi:10.1038/s41598-017-00645-3
32. Christen U. Pathogen infection and autoimmune disease. *Clin Exp Immunol*. 2019;195(1):10–14. doi:10.1111/cei.13239
33. Vahabi M, Ghazanfari T, Sepehrnia S. Molecular mimicry, hyperactive immune system, and SARS-COV-2 are three prerequisites of the autoimmune disease triangle following COVID-19 infection. *Int Immunopharmacol*. 2022;112:109183. doi:10.1016/j.intimp.2022.109183
34. Zeng XD, Chen H, Hu WG. Myelin Oligodendrocyte Glycoprotein (MOG) antibody-associated meningoencephalitis due to Mycoplasma pneumoniae infection. *Neurol Res*. 2023;45(2):124–126. doi:10.1080/01616412.2022.2124794
35. Nasa P, Mortada M, Singh A, Malhotra V, Syed H. Transient myelin oligodendrocyte glycoprotein antibody-positive acute disseminated encephalomyelitis following influenza A infection: a rare case. *Saudi J Med Med Sci*. 2021;9(3):271–275. doi:10.4103/sjmms.sjmms_791_20
36. Yadav A, Sharma R, Ganiger MP, Sharma G. Anti-myelin oligodendrocyte glycoprotein antibody positive acute disseminated encephalomyelitis post-varicella zoster virus infection. *Ann Indian Acad Neurol*. 2021;24(5):797–799. doi:10.4103/aian.AIAN_1054_20

Infection and Drug Resistance

Dovepress
Taylor & Francis Group

Publish your work in this journal

Infection and Drug Resistance is an international, peer-reviewed open-access journal that focuses on the optimal treatment of infection (bacterial, fungal and viral) and the development and institution of preventive strategies to minimize the development and spread of resistance. The journal is specifically concerned with the epidemiology of antibiotic resistance and the mechanisms of resistance development and diffusion in both hospitals and the community. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit http://www.dovepress.com/testimonials.php to read real quotes from published authors.

Submit your manuscript here: https://www.dovepress.com/infection-and-drug-resistance-journal