Cureus
Part of SPRINGER NATURE

Open Access Case Report

Sensorimotor Axonal Polyneuropathy Revealing Chronic Hepatitis B Virus Infection in the Postpartum Period: A Case Report and Review of the Literature

Review began 10/12/2026
Review ended 11/10/2026
Published 11/24/2026

© **Copyright** 2026
Zouaoui et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.120178

Mehdi Zouaoui [1], Ilyass Lachgar [1], Ibtissam Bamaarouf [1], Youssef Hnach [2], Mbarek Azouaoui [1], Nourdin Aqodad [1]

1. Department of Hepato-Gastroenterology, Mohammed VI University Hospital, Agadir, MAR 2. Department of Hepato-Gastroenterology, Oued Eddahab Military Hospital, Agadir, MAR

Corresponding author: Ilyass Lachgar, ilyasslachgar@gmail.com

Abstract

Extrahepatic manifestations of chronic hepatitis B virus (HBV) infection are well described; however, peripheral nervous system involvement is a rare and probably underdiagnosed manifestation. We report a case of sensorimotor axonal polyneuropathy that led to the diagnosis of a previously unrecognized chronic HBV infection during the postpartum period.

A 32-year-old woman, gravida 2 para 2, who delivered vaginally at home in the context of preeclampsia, developed partial functional impairment of both lower limbs during the postpartum period, accompanied by a static-kinetic cerebellar syndrome, bilateral horizontal nystagmus, and a peripheral neurogenic syndrome involving all four limbs, without digestive symptoms or clinical signs of liver disease. Workup revealed a positive hepatitis B surface antigen, positive total anti-HBc antibodies, negative HBe antigen, and an elevated HBV viral load, despite normal liver function tests. Antineutrophil cytoplasmic antibodies (ANCA) and hepatitis C virus (HCV) serology were negative. Complement levels, cryoglobulinemia testing, and nerve/muscle biopsy were not performed. Cerebrospinal fluid (CSF) analysis was normal, with no evidence of albuminocytologic dissociation. Vitamin B1 deficiency was identified, whereas vitamin B12 levels were normal and cold agglutinins were negative. Brain CT angiography and MRI were normal. Nerve conduction studies were consistent with sensorimotor axonal polyneuropathy, predominantly affecting the lower limbs. Antiviral therapy with tenofovir was initiated, and clinical and paraclinical follow-up was planned.

This presentation falls within the spectrum of extrahepatic neurological manifestations of HBV infection. The associated vitamin B1 deficiency represents an important confounding factor that must be considered in the diagnostic reasoning, together with the immunological particularities of the postpartum period. The treatment decision was guided by current international recommendations regarding early antiviral therapy for HBV infection in the presence of symptomatic extrahepatic manifestations. In patients presenting with unexplained peripheral neuropathy, particularly in a suggestive clinical context, screening for chronic HBV infection should be systematic, even in the absence of hepatic abnormalities. Nevertheless, a comprehensive etiological workup is essential to exclude relevant differential diagnoses before HBV infection is considered the sole underlying cause.

Categories: Neurology, Infectious Disease, Gastroenterology
Keywords: cryoglobulinaemia, extrahepatic manifestation, guillain-barré syndrome, hbv, hepatitis b, nerve conduction study, peripheral nervous system, polyneuropathy, postpartum, vitamin b1 deficiency

Introduction

Chronic hepatitis B virus (HBV) infection remains one of the leading causes of liver disease worldwide. According to the most recent WHO estimates, approximately 254 million people were living with chronic HBV infection in 2022, accounting for about 1.1 million deaths annually, mainly attributable to cirrhosis and hepatocellular carcinoma. The 2026 global hepatitis report confirms the persistently high disease burden, with approximately 240 million chronic carriers reported in 2024, fewer than 5% of whom were receiving antiviral treatment [1,2]. Although liver involvement remains the most feared complication of this infection, approximately 20% of patients develop extrahepatic manifestations, including polyarteritis nodosa (PAN), non-rheumatoid arthritis, glomerulonephritis, non-Hodgkin lymphoma, and cryoglobulinemic vasculitis. These manifestations, which are independent of viral genotype, may precede, accompany, or occur in the absence of overt hepatic disease, significantly contributing to patient morbidity and impaired quality of life [3].

Peripheral nervous system involvement is among the least well-characterized extrahepatic manifestations of HBV infection. It may present as mononeuritis multiplex in the setting of necrotizing vasculitis, such as HBV-associated PAN, historically the neurological presentation most classically associated with HBV; as

polyneuropathy associated with mixed cryoglobulinemia, which is considerably less common than in hepatitis C virus (HCV) infection; or, exceptionally, as Guillain-Barré syndrome (GBS) [3,4]. The postpartum period represents a distinct pathophysiological setting, characterized by rapid immune reconstitution following the relative immunological tolerance of pregnancy, which may unmask or exacerbate underlying infectious or dysimmune conditions [5].

We report the case of a patient who developed sensorimotor axonal polyneuropathy associated with a cerebellar syndrome and nystagmus in the immediate postpartum period following a delivery complicated by preeclampsia, leading to the diagnosis of previously unrecognized chronic HBV infection. This case provides an opportunity to discuss, in light of a review of the literature, the spectrum of HBV-associated peripheral neurological manifestations, their differential diagnoses, particularly nutritional causes, in the specific context of the postpartum period, and the current diagnostic and therapeutic implications of these manifestations.

Case Presentation

History

A 32-year-old woman, gravida 2 para 2 (with two living children, both delivered vaginally), with no significant past medical history apart from preeclampsia during the current pregnancy, delivered vaginally at home. She was hospitalized in the immediate postpartum period for completion of postpartum care and management of preeclampsia. Her initial clinical course was favorable, allowing her to be discharged. However, she was readmitted approximately three weeks after delivery due to rapidly progressive partial functional impairment of both lower limbs. There were no associated gastrointestinal symptoms, including abdominal pain, changes in bowel habits, gastrointestinal bleeding, or vomiting. Nevertheless, as discussed below, the absence of digestive symptoms does not exclude inadequate micronutrient intake or increased nutritional requirements during pregnancy and the peripartum period.

Physical examination

On admission, the patient was conscious and hemodynamically and respiratorily stable (blood pressure: 120/70 mmHg; heart rate: 113 beats/min; oxygen saturation: 100% on room air). Her conjunctivae were normally colored, with no jaundice or asterixis, findings that were specifically assessed given the hepatotropic viral infection subsequently suspected. Abdominal examination was unremarkable, with a soft, non-distended abdomen, no visible collateral venous circulation, no tenderness, guarding or rigidity, and no percussion dullness. There were no clinical stigmata of chronic liver disease or portal hypertension.

Neurological examination revealed a static-kinetic cerebellar syndrome, bilateral horizontal nystagmus, and a peripheral neurogenic syndrome involving all four limbs. The coexistence of these three findings, namely cerebellar involvement, nystagmus, and polyneuropathy, is particularly relevant to the diagnostic discussion below, as this clinical constellation may also be suggestive of vitamin B1 deficiency.

Laboratory, virological, and imaging findings

The results of the etiological workup are summarized in Table *1*. They revealed HBV infection, with a positive hepatitis B surface antigen, positive total anti-HBc antibodies, and negative HBe antigen, associated with a moderate viral load (8,590 IU/mL), despite normal liver function tests, with AST and ALT levels within normal limits. This serological profile, characterized by HBe antigen negativity, moderate viremia, and normal transaminase levels, is consistent with the so-called "indeterminate" or "gray zone" phase of chronic HBV infection, the management of which has recently been reassessed in international guidelines, as discussed.

Parameter	Patient result	Normal value (reference range)
HBsAg	Positive	Negative
Total anti-HBc antibodies	Positive	Negative
HBeAg	Negative	Negative
HBV viral load (DNA, PCR)	8590 IU/mL	Not detectable
HCV serology	Negative	Negative
HIV serology	Negative	Negative
Syphilis serology (TPHA/VDRL)	Negative	Negative
ANCA	Negative	Negative
Cold agglutinins	Negative	Negative (titer < 1:32)
AST	28 U/L	5–34 U/L
ALT	31 U/L	5–36 U/L
CSF white cell count	1/mm³	0–5/mm³
CSF red cell count	9/mm³	0/mm³ (absent)
CSF protein	0.6 g/L	0.15–0.45 g/L
CSF glucose	0.5 g/L	0.40–0.70 g/L
CSF culture	Sterile	Sterile
Vitamin B1 (thiamine)	4 ng/mL	7–30 ng/mL
Vitamin B12	843 pg/mL	200–900 pg/mL
Creatine kinase (CK)	32 U/L	26–140 U/L

TABLE 1: Summary of laboratory findings

HBsAg: hepatitis B surface antigen; anti-HBc: antibodies against the hepatitis B core antigen; HBeAg: hepatitis B e antigen; DNA: deoxyribonucleic acid; PCR: polymerase chain reaction; HCV: hepatitis C virus; HIV: human immunodeficiency virus; TPHA: Treponema pallidum hemagglutination assay; VDRL: Venereal Disease Research Laboratory (syphilis serology test); ANCA: antineutrophil cytoplasmic antibodies; AST: aspartate aminotransferase; ALT: alanine aminotransferase; CSF: cerebrospinal fluid; CK: creatine kinase

HIV, syphilis, and HCV serologies were negative, with the latter making HCV-associated cryoglobulinemia an unlikely alternative diagnosis. Testing for antineutrophil cytoplasmic antibodies (ANCA) was also negative, thereby reducing the likelihood of ANCA-associated vasculitis. Cold agglutinin testing was negative, making an immune-mediated hemolytic process or an underlying IgM monoclonal lymphoid disorder unlikely as an alternative explanation for the polyneuropathy. However, complement levels (C3 and C4), cryoglobulinemia testing, and nerve/muscle biopsy had not been performed at the time of manuscript preparation, representing an important limitation that is discussed below.

Cerebrospinal fluid (CSF) analysis was normal, with no protein elevation or albuminocytologic dissociation, and both direct examination and culture were negative. Vitamin assessment revealed vitamin B1 deficiency, while vitamin B12 levels were normal. Creatine kinase levels were normal, arguing against an associated myopathic process. Brain CT angiography and MRI revealed no abnormalities that could explain the cerebellar syndrome or nystagmus. However, these normal imaging findings do not definitively exclude thiamine-deficiency-related brainstem or cerebellar involvement, as MRI findings may remain normal in some mild or incomplete forms of thiamine deficiency (Table *2*).

Study	Result
Brain CT angiography	Normal, no vascular abnormality
Brain MRI	Normal, no brainstem or cerebellar lesion

TABLE 2: Brain imaging findings

CT: computed tomography (with angiographic acquisition of the intracranial vessels); MRI: magnetic resonance imaging

Nerve conduction studies

The findings of the nerve conduction studies (NCS/EMG), performed approximately three weeks after delivery, are summarized in Table *3*.

Nerve studied	Parameter	Result
Upper-limb nerves (motor)	Motor amplitude	Normal
Upper-limb nerves (motor)	Distal latency	Normal
Upper-limb nerves (motor)	Motor conduction velocity	Normal
Right tibial nerve	Motor response	Absent
Left tibial nerve	Motor response	Absent
Right peroneal nerve	Motor amplitude	Collapsed
Right peroneal nerve	Distal latency	Normal
Right peroneal nerve	Motor conduction velocity	Normal
Left peroneal nerve	Motor amplitude	Collapsed
Left peroneal nerve	Distal latency	Normal
Left peroneal nerve	Motor conduction velocity	Normal
Nerves explored (lower limbs)	F-wave latency	Normal
Upper-limb sensory nerves	Sensory amplitude	Decreased
Upper-limb sensory nerves	Sensory conduction velocity	Decreased
Lower-limb sensory nerves	Sensory amplitude	Decreased
Lower-limb sensory nerves	Sensory conduction velocity	Decreased

TABLE 3: Nerve conduction study findings

NCS/EMG: nerve conduction study/electromyography; F-wave: late response elicited by supramaximal motor nerve stimulation, reflecting conduction along the proximal portion of the motor nerve (roots and plexus); right/left: side of the nerve examined

These findings were consistent with a sensorimotor axonal polyneuropathy, predominantly affecting the lower limbs. The preserved F-wave latencies and the absence of conduction block or temporal dispersion make an acute demyelinating process, such as an inflammatory polyradiculoneuropathy, unlikely, a point discussed in detail below.

Diagnostic synthesis and initial management

Taken together, the combination of sensorimotor axonal polyneuropathy affecting all four limbs, a static-kinetic cerebellar syndrome, and bilateral horizontal nystagmus, occurring during the postpartum period in a patient with previously undiagnosed chronic HBV infection, and with no alternative cause identified on brain imaging or CSF analysis, was considered suggestive of an HBV-related peripheral neurological manifestation. The negative ANCA results make ANCA-associated vasculitis unlikely, while negative HCV

serology makes hepatitis C-related cryoglobulinemia an unlikely alternative cause. Similarly, the absence of cold agglutinins makes an underlying IgM monoclonal lymphoid disorder less likely. Collectively, these findings narrow the differential diagnosis and support a possible association with HBV infection. However, in the absence of complement measurements, cryoglobulinemia testing, and nerve/muscle biopsy, the presence of HBV-related vasculitis or cryoglobulinemia cannot be definitively established at this stage. The concomitant vitamin B1 deficiency should therefore be considered a potential contributing factor rather than the sole explanation for the neurological presentation.

Treatment and follow-up

Based on the available evidence and in accordance with current guidelines, antiviral therapy with tenofovir was initiated. Treatment had only recently been started at the time of manuscript preparation; therefore, no data on the clinical, virological, or electrophysiological response are yet available. Clinical, laboratory (viral load and transaminase levels), and electrophysiological follow-up are planned to assess the response to antiviral therapy and the progression of the polyneuropathy, as well as to guide any further escalation of management, including completion of the immunological workup or initiation of immunomodulatory therapy, if clinical improvement is not achieved.

Discussion

Epidemiology and pathophysiology of extrahepatic manifestations of HBV

Chronic HBV infection is associated with extrahepatic manifestations in approximately 20% of patients, with no established correlation between these manifestations and viral genotype [3]. These manifestations are classically attributed to immunological mechanisms, particularly the deposition of circulating immune complexes formed by viral antigens, notably HBsAg, and their corresponding antibodies. This process triggers complement activation and inflammation of the vessel walls across vessels of various calibers. This mechanism is best documented in HBV-associated PAN, in which immune complex and complement deposits have been demonstrated in the walls of affected medium-sized vessels [4].

Historically, PAN was considered the archetypal HBV-associated vasculitis, with up to 30% to 50% of PAN cases attributed to HBV infection in studies published before the widespread introduction of HBV vaccination and systematic blood donor screening. The widespread implementation of HBV vaccination has since considerably reduced the incidence of this association in countries with high vaccine coverage, although it persists in regions with high viral endemicity [4].

Spectrum of HBV-related peripheral neurological involvement

1. Mononeuritis Multiplex and HBV-Associated Polyarteritis Nodosa

HBV-associated PAN is a necrotizing vasculitis affecting medium-sized arteries and capable of involving multiple organs, including the skin, gastrointestinal tract, kidneys, and peripheral nervous system. Neurologically, it typically manifests as mononeuritis multiplex, often with an abrupt and asymmetric onset, initially predominantly sensory and subsequently progressing to motor involvement, resulting from ischemia of the vasa nervorum. In the largest published series to date, which included 115 patients with HBV-associated PAN, peripheral neurological involvement was among the most frequent and disabling manifestations of the disease, warranting a treatment approach combining antiviral therapy, a short course of corticosteroids, and plasma exchange [4].

2. HBV-Related Cryoglobulinemic Polyneuropathy

Mixed cryoglobulinemia, an immune complex-mediated small-vessel vasculitis, is predominantly associated with HCV, which accounts for approximately 90% of cases of cryoglobulinemic vasculitis. Its association with HBV is considerably less common, being reported in only 0.5% to 5.5% of mixed cryoglobulinemia cases in published series and affecting approximately 2% to 4% of patients with chronic hepatitis B [6]. When present, HBV-related cryoglobulinemia may nevertheless be complicated by sensory or sensorimotor, distal, length-dependent polyneuropathy, which is often painful and ascending. This occurs through a mechanism comparable to that described in HCV-related cryoglobulinemic neuropathy, involving both ischemia resulting from the deposition of cryoprecipitable immune complexes in the vasa nervorum and potentially direct antineuronal reactivity [6,7]. A recently published case of cryoglobulinemia associated with chronic HBV infection demonstrated marked clinical and laboratory improvement after a few months of antiviral treatment with tenofovir alafenamide, without the need for additional immunosuppressive therapy [5]. In our patient, negative HCV serology makes the most common cause of mixed cryoglobulinemia unlikely. However, cryoglobulinemia testing was not performed, meaning that this possibility remains uninvestigated rather than excluded.

3. Guillain-Barré Syndrome

GBS has been reported in association with both acute and chronic HBV infection, particularly during episodes of viral reactivation, although this association remains rare and accounts for approximately 1% of all GBS cases [8,9]. Electrophysiologically, GBS is classically classified into a demyelinating form (acute inflammatory demyelinating polyradiculoneuropathy (AIDP)) and axonal forms (acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN)), based on well-established electrodiagnostic criteria that include conduction velocities, distal latencies, F-waves, and the assessment of conduction block or temporal dispersion [10,11]. The diagnosis of GBS is classically based on the combination of acute, progressive motor weakness, areflexia, and cerebrospinal fluid albuminocytologic dissociation, although the latter may be absent during the first few days of illness [12].

4. Other Rare Presentations

Other forms of peripheral nervous system involvement have been reported more anecdotally in HBV infection: plexopathies, radiculopathies, and isolated cranial nerve neuropathies. These presentations remain exceptional and rest essentially on isolated observations, which limits the strength of any pathophysiological conclusions that can be drawn from them [3].

Diagnostic approach and electrophysiological interpretation

The evaluation of distal symmetric polyneuropathy classically follows a structured approach combining medical history, clinical examination, nerve conduction studies, and targeted laboratory investigations. American Academy of Neurology guidelines emphasize the importance of a first-line workup that includes at least blood glucose measurement, vitamin B12 levels, and serum protein electrophoresis, supplemented, as clinically indicated, by viral serologies and an immunological panel [13]. In our patient, nerve conduction studies enabled precise characterization of the neuropathy in terms of its nature (axonal rather than demyelinating) and distribution (sensorimotor, predominantly distal, and affecting the lower limbs). These findings subsequently guided the etiological workup toward acquired axonal neuropathies, including those associated with hepatotropic virus-related vasculitis, as well as nutritional, toxic, and metabolic causes.

From a methodological standpoint, distinguishing axonal from demyelinating neuropathy relies on a combination of electrophysiological findings. Relatively preserved conduction velocities and distal latencies, together with a proportional reduction in response amplitudes, favor an axonal process. In contrast, disproportionate slowing of conduction velocities, prolonged distal latencies, conduction block or temporal dispersion, and prolonged or absent F waves favor a demyelinating process [10,11]. In our patient, the markedly reduced motor response amplitudes, with preserved latencies and conduction velocities in the peroneal nerves, together with normal F-wave latencies, strongly support an axonal process.

Differential diagnoses and confounders specific to the postpartum period

1. Vitamin B1 Deficiency, Wernicke's Encephalopathy, and Beriberi

Vitamin B1 (thiamine) deficiency is an important differential diagnosis in patients presenting with a combination of cerebellar syndrome, nystagmus, and peripheral polyneuropathy. This clinical presentation may reflect an overlap between Wernicke's encephalopathy, classically characterized by the triad of confusion, ataxia, and oculomotor disturbances, and dry beriberi, the peripheral neuropathic form of thiamine deficiency [14]. Although traditionally associated with chronic alcoholism, vitamin B1 deficiency is also well documented during pregnancy and the postpartum period, particularly in the setting of prolonged hyperemesis gravidarum. A recent Moroccan case series of 12 patients with hyperemesis gravidarum-related Wernicke's encephalopathy reported clinical presentations combining nystagmus, ataxia, and peripheral nervous system involvement, with potentially normal brain imaging and no other identifiable cause [15,16]. Although our patient reported no vomiting or other gastrointestinal symptoms, inadequate dietary intake or increased thiamine requirements related to pregnancy and lactation cannot be excluded. Therefore, the documented biochemical deficiency should be considered a potential contributing factor, although not necessarily the sole cause, of the neurological manifestations observed.

2. Postpartum Period and Immunological Vulnerability

Pregnancy is characterized by a state of relative immune tolerance, which is necessary for the acceptance of the semi-allogeneic fetus and involves, in particular, attenuation of cell-mediated pro-inflammatory responses. Conversely, the postpartum period represents a phase of rapid immune reconstitution, characterized by rising cortisol and prolactin levels and a marked decline in estrogen and progesterone levels. These changes may be accompanied by an inflammatory rebound capable of unmasking or exacerbating previously quiescent infectious or dysimmune conditions [5,17]. This phenomenon, which is well documented in several systemic autoimmune diseases, including systemic lupus erythematosus, rheumatoid arthritis, and postpartum thyroiditis, may provide a plausible pathophysiological framework for explaining the emergence, in the weeks following delivery, of a previously latent neurological manifestation of an underlying, previously undiagnosed chronic HBV infection [17].

Therapeutic implications

This case highlights the importance of systematic HBV screening in patients presenting with unexplained peripheral neuropathy, even in the absence of elevated liver enzymes. The updated 2025 EASL guidelines have broadened the indications for antiviral therapy beyond the traditional criteria based solely on transaminase levels and viral load, explicitly recognizing symptomatic extrahepatic manifestations as an additional indication for treatment initiation [18]. This change is supported by the findings of the randomized ATTENTION trial, which evaluated early antiviral treatment with tenofovir alafenamide in patients with moderate-to-high viremia and normal or near-normal transaminase levels, a clinical scenario directly comparable to that of our patient. The trial demonstrated a significant reduction in the incidence of serious clinical complications in the early-treatment group compared with the control group (incidence rates of 0.33 versus 1.57 per 100 person-years; hazard ratio: 0.21; 97.5% CI: 0.04-1.20; p = 0.027) [19].

When HBV-associated vasculitis, such as PAN or cryoglobulinemia, is confirmed, management classically combines antiviral therapy aimed at suppressing viral replication, which is essential for achieving sustained remission of vasculitis, with plasma exchange and a short course of corticosteroids in severe cases or those associated with a risk of functional impairment or life-threatening complications. Conventional immunosuppressants and rituximab are generally reserved for refractory cases because of the risk of promoting viral replication [4,6].

Prognosis

The prognosis of HBV-related peripheral neuropathies largely depends on the underlying pathophysiological mechanism and the timeliness of treatment initiation. Mild-to-moderate cryoglobulinemic forms generally respond favorably to antiviral therapy alone, with clinical improvement and clearance of circulating cryoglobulins occurring within a few weeks to several months, as illustrated by recently published case reports [5]. In contrast, severe vasculitic forms, such as PAN, carry a risk of permanent peripheral neurological sequelae if treatment is not initiated promptly, highlighting the importance of early diagnosis and management [4]. In our case, the limited follow-up available at the time of manuscript preparation does not allow any conclusions to be drawn regarding the patient's clinical or electrophysiological outcome.

Limitations

This report has several limitations inherent to a single case observation. Negative ANCA results make ANCA-associated vasculitis unlikely, negative HCV serology excludes the most common cause of mixed cryoglobulinemia, and negative cold agglutinin testing makes an underlying IgM monoclonal lymphoid disorder less likely. Collectively, these findings strengthen, but do not establish, the hypothesis of an HBV-related neurological manifestation. However, the absence of complement measurements, cryoglobulinemia testing, and nerve/muscle biopsy at the time of manuscript preparation prevents a definitive distinction between a specific vascular etiology, such as PAN or HBV-related cryoglobulinemia, and a less well-characterized parainfectious process involving the peripheral nervous system. Second, because antiviral therapy with tenofovir had only recently been initiated, the lack of follow-up data on the clinical and electrophysiological course under treatment precludes any definitive conclusions regarding the causal relationship between HBV infection and the observed polyneuropathy or the response to treatment. These findings will need to be reported in a subsequent update of this case. Finally, the coexistence of documented vitamin B1 deficiency represents a potential confounding factor that cannot be entirely excluded based on the available data. This further supports the possibility of a multifactorial etiology rather than exclusive attribution of the neurological manifestations to HBV infection.

Conclusions

This case report illustrates an initial peripheral neurological presentation of chronic HBV infection during the postpartum period, an unusual setting for this type of manifestation. It highlights the importance of systematically considering chronic HBV infection in patients presenting with unexplained polyneuropathy, even in the absence of hepatic abnormalities. At the same time, it underscores the need for a comprehensive etiological workup to investigate potential associated vasculitis and nutritional causes, particularly in the specific context of the postpartum period, before attributing the neurological manifestations exclusively to HBV infection. Finally, this case highlights recent changes in international guidelines favoring earlier initiation of antiviral therapy when symptomatic extrahepatic manifestations of HBV infection are identified, regardless of the patient's conventional hepatic status.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ilyass Lachgar, Youssef Hnach, Ibtissam Bamaarouf

Acquisition, analysis, or interpretation of data: Ilyass Lachgar, Mehdi Zouaoui, Mbarek Azouaoui, Nourdin Aqodad

Drafting of the manuscript: Ilyass Lachgar, Mehdi Zouaoui, Ibtissam Bamaarouf

Critical review of the manuscript for important intellectual content: Ilyass Lachgar, Mehdi Zouaoui, Youssef Hnach, Mbarek Azouaoui, Nourdin Aqodad

Supervision: Ilyass Lachgar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. World Health Organization. Hepatitis B. Fact sheet . (2025). Accessed: November 12, 2026: https://www.who.int/news-room/fact-sheets/detail/hepatitis-b.
2. World Health Organization. Global Hepatitis Report . (2026). Accessed: November 12, 2026: https://www.who.int/teams/global-hiv-hepatitis-and-stis-programmes/hepatitis/reports/global-hepatitis-report-2026.
3. Mazzaro C, Adinolfi LE, Pozzato G, et al.: Extrahepatic manifestations of chronic HBV infection and the role of antiviral therapy. J Clin Med. 2022, 11:23-5. 10.3390/jcm11216247
4. Guillevin L, Mahr A, Callard P, Godmer P, Pagnoux C, Leray E, Cohen P: Hepatitis B virus-associated polyarteritis nodosa: clinical characteristics, outcome, and impact of treatment in 115 patients. Medicine (Baltimore). 2005, 84:313-22. 10.1097/01.md.0000180792.80212.5e
5. Kolli H, Ali MJ, Campoverde Reyes KJ, Lau DT: Case of cryoglobulinaemia associated with chronic hepatitis B. BMJ Case Rep. 2022, 15:16-8. 10.1136/bcr-2022-249630
6. Ferri C: Mixed cryoglobulinemia. Orphanet J Rare Dis. 2008, 3:25. 10.1186/1750-1172-3-25
7. Alpa M, Ferrero B, Cavallo R, et al.: Anti-neuronal antibodies in patients with HCV-related mixed cryoglobulinemia. Autoimmun Rev. 2008, 8:56-8. 10.1016/j.autrev.2008.07.027
8. Wei J, Duan S: Severe Guillain-Barré syndrome associated with chronic hepatitis B: a case report and literature review. Medicine (Baltimore). 2021, 100:e27989. 10.1097/MD.0000000000027989
9. Yimam KK, Merriman RB, Todd Frederick R: A rare case of acute hepatitis B virus infection causing Guillain-Barré syndrome. Gastroenterol Hepatol (N Y). 2013, 9:121-3.
10. Hadden RD, Cornblath DR, Hughes RA, Zielasek J, Hartung HP, Toyka KV, Swan AV: Electrophysiological classification of Guillain-Barré syndrome: clinical associations and outcome. Plasma Exchange/Sandoglobulin Guillain-Barré Syndrome Trial Group. Ann Neurol. 1998, 44:780-8. 10.1002/ana.410440512
11. Uncini A, Kuwabara S: The electrodiagnosis of Guillain-Barré syndrome subtypes: where do we stand? . Clin Neurophysiol. 2018, 129:2586-93. 10.1016/j.clinph.2018.09.025
12. Asbury AK, Cornblath DR: Assessment of current diagnostic criteria for Guillain-Barré syndrome . Ann Neurol. 1990, 27:S21-4. 10.1002/ana.410270707
13. England JD, Gronseth GS, Franklin G, et al.: Practice parameter: evaluation of distal symmetric polyneuropathy: role of laboratory and genetic testing (an evidence-based review). Report of the American Academy of Neurology, American Association of Neuromuscular and Electrodiagnostic Medicine, and American Academy of Physical Medicine and Rehabilitation. Neurology. 2009, 72:185-92. 10.1212/01.wnl.0000336370.51010.a1
14. Kumar N: Nutritional neuropathies. Neurol Clin. 2007, 25:209-55. 10.1016/j.ncl.2006.11.001
15. Abouelbaqua K, Rebahi H, Louhab N, Kissani N, El Adib AR: Wernicke encephalopathy related to hyperemesis gravidarum: a retrospective study of 12 cases. Case Rep Crit Care. 2025, 2025:7607058. 10.1155/crcc/7607058
16. Chiossi G, Neri I, Cavazzuti M, Basso G, Facchinetti F: Hyperemesis gravidarum complicated by Wernicke encephalopathy: background, case report, and review of the literature. Obstet Gynecol Surv. 2006, 61:255-68. 10.1097/01.ogx.0000206336.08794.65
17. Singh N, Perfect JR: Immune reconstitution syndrome and exacerbation of infections after pregnancy . Clin Infect Dis. 2007, 45:1192-9. 10.1086/522182
18. European Association for the Study of the Liver: EASL Clinical Practice Guidelines on the management of hepatitis B virus infection. J Hepatol. 2025, 83:502-83. 10.1016/j.jhep.2025.03.018
19. Lim YS, Yu ML, Choi J, et al.: Early antiviral treatment with tenofovir alafenamide to prevent serious clinical adverse events in adults with chronic hepatitis B and moderate or high viraemia (ATTENTION): interim results from a randomised controlled trial. Lancet Gastroenterol Hepatol. 2025, 10:295-305. 10.1016/S2468-1253(24)00431-X