

A Case of Cholestatic Acute Hepatitis B Virus Infection with Severe Jaundice: Rapid Resolution with Corticosteroid Therapy

Dr. Jhumur Ghosh¹, Dr. Nafsin Nur Morshed Bhuiyan² and Jaya Ghosh³

¹Associate Professor, Department of Hepatology, MH Samorita Hospital and Medical College, Tejgaon, Dhaka - 1208, BANGLADESH.

²Lecturer, Department of Forensic Medicine and Toxicology, MH Samorita Hospital and Medical College, Tejgaon, Dhaka - 1208, BANGLADESH.

³Clinical Trial Coordinator, Radiation Oncology Department, Tata Medical Center, Newtown, Rajarhat, Kolkata - 700160, INDIA.

³Corresponding Author: jaya95ghosh@gmail.com

ORCID

<https://orcid.org/0009-0001-4449-0705>



www.jrasb.com || Vol. 5 No. 3 (2026): June Issue

Received: 17-05-2026

Revised: 21-06-2026

Accepted: 26-06-2026

ABSTRACT

Cholestatic hepatitis is an uncommon but clinically significant manifestation of acute hepatitis B virus (HBV) infection, characterized by prolonged jaundice, impaired bile flow, and severe pruritus. The role of corticosteroids in HBV-related cholestasis remains controversial due to concerns about immunosuppression and viral reactivation. We report a 52-year-old hypertensive man with acute HBV infection who developed persistent cholestasis with a peak bilirubin of 32.1 mg/dL. Despite treatment with tenofovir alafenamide, ursodeoxycholic acid, and cholestyramine, no improvement was observed. Initiation of oral prednisolone (40 mg daily) led to rapid resolution of pruritus within one week and significant bilirubin reduction to 5.8 mg/dL in two weeks. Steroids were tapered over five weeks, with complete biochemical recovery. At 12-weeks follow-up, the patient achieved HBsAg seroconversion and undetectable HBV DNA. This case suggests corticosteroids may benefit selected patients with refractory cholestatic hepatitis B.

Keywords- Acute hepatitis B; Intrahepatic cholestasis; Corticosteroid; Prednisolone; HBsAg seroconversion; Tenofovir alafenamide.

I. INTRODUCTION

Acute viral hepatitis is a widespread liver condition caused by hepatotropic viruses such as hepatitis A (HAV), B (HBV), C (HCV), D (HDV), and E (HEV). HAV and HEV are primarily transmitted through the fecal-oral route, while HBV, HCV, and HDV spread via blood and body fluids, with the potential to cause both acute and chronic liver disease¹. Globally, viral hepatitis remains a major health concern, accounting for approximately 1.1 million deaths in 2022,

largely due to long-term complications like cirrhosis and hepatocellular carcinoma².

HBV is highly prevalent in regions including sub-Saharan Africa, Southeast Asia, and parts of the Western Pacific, where infection rates exceed 8%. An estimated 254 million individuals worldwide live with chronic HBV infection^{3,4}. Most acute HBV cases are asymptomatic or present without jaundice, while others develop symptoms such as fatigue, nausea, jaundice, and elevated liver enzymes. Although rare, acute liver failure can occur and carries significant mortality risk⁵.

Cholestatic hepatitis is an uncommon complication of acute Hepatitis B infection, characterized by impaired intrahepatic bile flow that results in progressive hyperbilirubinemia, pruritus, and prolonged jaundice disproportionate to the extent of hepatocellular injury.⁶ Although corticosteroids have been shown to shorten the course of prolonged cholestasis associated with Hepatitis A infection, their role in HBV-related cholestasis remains considerably less well defined and is further complicated by the theoretical risks of viral reactivation and immunosuppression.⁷ This balance between achieving symptomatic relief through immunomodulation and preserving virological control constitutes the central clinical challenge in the management of cholestatic HBV infection. Here, we report a case of acute HBV-associated intrahepatic cholestasis refractory to conventional therapy that demonstrated a rapid clinical response to oral prednisolone administered concomitantly with antiviral treatment, thereby adding to the limited evidence supporting this therapeutic strategy.

II. CASE REPORT

A 52-year-old hypertensive, non-diabetic man presented with an 8-weeks history of progressively worsening jaundice associated with severe pruritus. He had initially been evaluated at a tertiary care center, where acute hepatitis B virus (HBV) infection was diagnosed on the basis of markedly elevated liver enzymes and positive serological markers (Table 1). He had no prior history of liver disease and denied recognized risk factors, including intravenous drug use, blood transfusion, recent invasive procedures, or unprotected sexual exposure. Despite the absence of identifiable risk factors, the clinical presentation and laboratory findings were consistent with newly acquired acute HBV infection, although the precise source of transmission remained uncertain.

On physical examination, the patient had marked icterus involving both the sclera and skin, along with mild hepatosplenomegaly. There were no stigmata of chronic liver disease, such as spider angiomas, lymphadenopathy, or hepatic encephalopathy. Laboratory investigations demonstrated severe conjugated hyperbilirubinemia, with a total bilirubin level of 29.9 mg/dL, significantly elevated alanine aminotransferase (ALT) of 770 IU/L and aspartate aminotransferase (AST) of 267 IU/L, and mildly elevated alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). Serum albumin and international normalized ratio (INR) were within normal limits, indicating preserved hepatic synthetic function. Abdominal ultrasonography revealed a hypoechoic liver without evidence of biliary obstruction.

Although liver biopsy was considered, it was ultimately deferred because of the markedly elevated bilirubin level and the associated risk of coagulopathy-related complications, the clear serological diagnosis of acute HBV infection, the characteristic clinical and biochemical profile of intrahepatic cholestasis, and the absence of features suggestive of an alternative diagnosis requiring histological confirmation. This decision was reviewed and endorsed by the treating hepatology team.

Initial management consisted of tenofovir alafenamide (TAF) 25 mg once daily, ursodeoxycholic acid (UDCA) 300 mg twice daily, and cholestyramine 4 g twice daily. Antiviral therapy with TAF was initiated in view of the markedly elevated bilirubin level (>10 mg/dL) and high HBV DNA viral load (7.73×10^5 IU/mL), in accordance with current guidance recommending antiviral therapy in severe acute HBV infection characterized by significant hyperbilirubinemia, even in the absence of overt synthetic dysfunction.⁸ UDCA was prescribed to enhance bile acid secretion and mitigate cholestatic injury, while cholestyramine was added for symptomatic relief of pruritus.

After two weeks of treatment, the patient showed no clinical improvement. Repeat laboratory testing demonstrated a further rise in total bilirubin to 32.1 mg/dL, with persistent severe pruritus significantly impairing quality of life (Table 2). In view of the failure of conventional cholestatic therapy, the absence of an alternative diagnosis, and previously documented use of corticosteroids in HAV-related prolonged cholestasis, oral prednisolone 40 mg/day was added to the ongoing antiviral regimen. Blood glucose monitoring was initiated because of the patient's hypertensive status and corticosteroid exposure.

Within one week of initiating prednisolone, the patient experienced marked symptomatic improvement, particularly in pruritus. Serum bilirubin declined rapidly to 5.8 mg/dL within two weeks, at which point the prednisolone dose was reduced to 20 mg/day. Gradual tapering was continued over the following weeks, and prednisolone was discontinued after a total treatment duration of five weeks. At the completion of steroid therapy, total bilirubin had normalized to 1.8 mg/dL, ALT had decreased to 40 IU/L, and ALP to 68 IU/L (Table 2). No corticosteroid-related adverse effects—including hyperglycemia, blood pressure instability, or clinical deterioration—were observed during the treatment period.

At 12-weeks follow-up, the patient remained completely asymptomatic, with sustained loss of HBsAg, undetectable HBV DNA (<10 IU/mL by real-time PCR), and no biochemical evidence of hepatitis relapse. HBsAg seroconversion in the setting of acute HBV infection represents a favorable immunological outcome and is consistent with effective viral clearance.

Table 1: Viral markers of patient.

Viral Markers	Results
HBsAg	Positive
Anti-HBc IgM	Positive
HBV DNA (real-time PCR)	7.73×10^5 IU/mL
Anti-HEV IgM	Negative
Anti-HAV IgM	Negative
Anti-HCV	Negative

HBV = hepatitis B virus; PCR = polymerase chain reaction.

Table 2: Liver function tests of patient.

Parameters	23 Oct 2025	31 Oct 2025	13 Nov 2025	29 Nov 2025	19 Dec 2025
S. Bilirubin (mg/dL)	13.5	29.9	32.1	5.8	1.8
ALT/SGPT (IU/L)	710	770	684	238	40
AST/SGOT (IU/L)	—	267	—	—	—
ALP (IU/L)	185	187	158	137	68
GGT (IU/L)	—	78	—	—	—
PT (seconds)	12	13.6	12.9	13	12
Albumin (g/dL)	3.7	3.8	3.6	3.8	3.9

ALT = alanine aminotransferase; AST = aspartate aminotransferase; ALP = alkaline phosphatase; GGT = gamma-glutamyl transferase; PT = prothrombin time.

III. DISCUSSIONS

This case report describes an uncommon presentation of intrahepatic cholestasis complicating acute hepatitis B virus (HBV) infection, which showed rapid resolution after the addition of oral prednisolone to ongoing antiviral therapy. Several important clinical issues emerge from this case, including the epidemiology and pathophysiology of cholestatic HBV, the potential risks and benefits of corticosteroid therapy, evidence from similar published cases, and the clinical significance of subsequent HBsAg seroconversion.

HBV infection continues to represent a major global health burden, with an estimated 254 million people living with chronic infection and approximately 1.2 million new infections occurring annually.² The burden is particularly high in low- and middle-income countries, especially across sub-Saharan Africa and Asia. Cholestatic hepatitis is an uncommon manifestation of acute HBV infection, reported in approximately 5–10% of symptomatic cases.^{6,9} It is typically characterized by marked hyperbilirubinemia, bilirubinuria, severe pruritus, and prolonged jaundice despite preserved extrahepatic biliary flow. This clinical course often results in prolonged hospitalization,

significant patient discomfort, and increased healthcare costs.

The pathogenesis of intrahepatic cholestasis in acute HBV is thought to be predominantly immune mediated. Cytotoxic T-cell-driven hepatocyte injury leads to the release of inflammatory cytokines, which suppress critical bile acid transporters, including NTCP, BSEP, and MRP2.^{10,11,12} The resulting impairment of bile excretion causes intracellular accumulation of bile acids, which may further aggravate hepatocellular injury. In addition, bile acids themselves can influence immune signaling pathways, suggesting a bidirectional interaction between cholestasis and immune dysregulation.

The use of corticosteroids in active HBV infection remains controversial because of the potential risks of immunosuppression, including enhanced viral replication and disease flare.¹³ In the present case, these risks were likely mitigated by several favorable factors, namely concurrent antiviral therapy with tenofovir alafenamide, preserved hepatic synthetic function, and the use of a short tapered course of prednisolone. The absence of clinical deterioration and the eventual achievement of HBsAg seroconversion support effective viral control and subsequent immune recovery.¹⁴

Although evidence supporting corticosteroid therapy in HBV-related cholestasis remains limited, available case reports suggest that selected patients may experience rapid clinical improvement when adequate antiviral coverage is provided. Tanaka et al. described a similar case of cholestatic acute HBV infection in which bilirubin levels declined rapidly after short-course prednisolone, followed by HBsAg clearance, findings that closely parallel our case.¹⁵ Likewise, Coppola et al. reported favorable outcomes in a small series of severe acute HBV cases treated with corticosteroids, emphasizing the importance of simultaneous antiviral therapy.¹⁶

In contrast, the evidence base for corticosteroid use in hepatitis A virus (HAV)-related prolonged cholestasis is more substantial. A systematic review by Alebaji et al. demonstrated consistent clinical and biochemical improvement with prednisolone or methylprednisolone across multiple case reports, with normalization of bilirubin generally occurring within four to eight weeks.⁷ While the mechanisms of cholestasis in HAV and HBV share similarities, a crucial distinction lies in the absence of ongoing viral replication in HAV, which is typically a self-limited infection. This makes corticosteroid therapy inherently safer in HAV than in HBV and underscores why concurrent antiviral therapy is essential when corticosteroids are considered in HBV-associated cholestasis.

Ursodeoxycholic acid (UDCA) has also been explored as a therapeutic option in cholestatic hepatitis. Although some studies suggest modest biochemical benefits through hydrophilic bile acid replacement and hepatoprotective effects,¹⁷ its efficacy in severe acute HBV-related cholestasis remains uncertain. This was reflected in our patient, who showed no meaningful clinical or biochemical response after two weeks of UDCA therapy.

Finally, the sustained HBsAg seroconversion observed in this patient strongly indicates successful viral clearance. The rapid symptomatic and biochemical response following corticosteroid therapy further supports an immune-mediated mechanism of cholestasis and highlights the potential role of carefully selected immunomodulatory therapy in specific cases.

IV. CONCLUSION

Acute HBV with intrahepatic cholestasis is challenging when standard therapy fails. A short corticosteroid course under antiviral cover may be effective, reflecting immune-mediated disease. Use potent antivirals like tenofovir alafenamide to prevent reactivation. Recommend careful selection, close monitoring, and further research to define optimal dosing, duration, and clear treatment guidelines.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENT

The authors are grateful to the patient for providing informed written consent for publication of this case report.

REFERENCES

- [1] Shepard CW, Simard EP, Finelli L, Fiore AE, Bell BP. Hepatitis B virus infection: epidemiology and vaccination. *Epidemiol Rev.* 2006;28:112–125. doi: 10.1093/epirev/mxj009.
- [2] World Health Organization. Global progress report on HIV, viral hepatitis and sexually transmitted infections, 2023. Geneva: WHO; 2023. Available from: <https://www.who.int/publications/i/item/9789240053779>
- [3] Hou J, Liu Z, Gu F. Epidemiology and prevention of hepatitis B virus infection. *Int J Med Sci.* 2005;2(1):50–57. doi: 10.7150/ijms.2.50.
- [4] Liang TJ. Hepatitis B: the virus and disease. *Hepatology.* 2009;49(5 Suppl):S13–21. doi: 10.1002/hep.22881.
- [5] Keefe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. *Clin Gastroenterol Hepatol.* 2008;6(12):1315–1341. doi: 10.1016/j.cgh.2008.08.021.
- [6] Shivangi, Mishra MK, Gupta S, Razdan K, Sudan S, Sehgal S. Clinical diagnosis of viral hepatitis: current status and future strategies. *Diagn Microbiol Infect Dis.* 2024;108(3):116151. doi: 10.1016/j.diagmicrobio.2023.116151.
- [7] Alebaji MB, Mehair AS, Shahrour OI, Elkhatib FA, Alkaabi EH, Alkuwaiti NS. Prolonged cholestasis following acute hepatitis A infection: case report and a review of literature. *Cureus.* 2023;15(5):e38511. doi: 10.7759/cureus.38511.
- [8] European Association for the Study of the Liver. EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection. *J Hepatol.* 2017;67(2):370–398. doi: 10.1016/j.jhep.2017.03.021.
- [9] Alter MJ. Epidemiology of hepatitis B in Europe and worldwide. *J Hepatol.* 2003;39 Suppl 1:S64–69. doi: 10.1016/s0168-8278(03)00141-7.
- [10] Trauner M, Meier PJ, Boyer JL. Molecular pathogenesis of cholestasis. *N Engl J Med.* 1998;339(17):1217–1227. doi: 10.1056/NEJM19981023391707.
- [11] Crocenzi FA, Mottino AD, Roma MG. Regulation of synthesis and trafficking of canalicular

- transporters and its alteration in acquired hepatocellular cholestasis. Experimental considerations for future therapeutics. *Curr Med Chem.* 2004;11(4):501–524. doi: 10.2174/0929867043455825.
- [12] Ding C, Hong Y, Che Y, He T, Wang Y, Zhang S, Wu J, Xu W, Hou J, Hao H, Cao L. Bile acid restrained T cell activation explains cholestasis aggravated hepatitis B virus infection. *FASEB J.* 2022;36(9):e22468. doi: 10.1096/fj.202200234R.
- [13] Lok AS, Liang RH, Chiu EK, Wong KL, Chan TK, Todd D. Reactivation of hepatitis B virus replication in patients receiving cytotoxic therapy: report of a prospective study. *Gastroenterology.* 1991;100(1):182–188. doi: 10.1016/0016-5085(91)90599-g.
- [14] Terrault NA, Lok ASF, McMahon BJ, Chang KM, Hwang JP, Jonas MM, et al. Update on prevention, diagnosis, and treatment of chronic hepatitis B: AASLD 2018 hepatitis B guidance. *Hepatology.* 2018;67(4):1560–1599. doi: 10.1002/hep.29800.
- [15] Tanaka H, Tsukuma H, Yamano H, Oshima A, Shibata H. Prednisolone therapy for acute cholestatic hepatitis B. *J Gastroenterol Hepatol.* 1995;10(5):581–584. doi: 10.1111/j.1440-1746.1995.tb01355.x.
- [16] Coppola N, Tonziello G, Colombatto P, Pisaturo M, Messina V, Moriconi F, et al. Lamivudine-resistant HBV strain rtM204V in acute hepatitis B. *J Infect.* 2013;67(4):322–329. doi: 10.1016/j.jinf.2013.04.014.
- [17] Paumgartner G, Beuers U. Ursodeoxycholic acid in cholestatic liver disease: mechanisms of action and therapeutic use revisited. *Hepatology.* 2002;36(3):525–531. doi: 10.1053/jhep.2002.36088.