Case Report

Clinical recovery in probable acute hepatitis B with severe hepatocellular injury: A case report

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ABSTRACT

Acute hepatitis is characterized by hepatocellular injury and may result from viral, drug-induced, autoimmune, ischemic, or other causes. We report the 30-day clinical, biochemical, serological, and ultrasonographic course of a 26-year-old woman with probable acute hepatitis B-associated severe hepatocellular injury. She presented with jaundice, fatigue, digestive discomfort, and constipation. Baseline investigations revealed marked hepatocellular injury, with alanine aminotransferase (ALT) 2,526 U/L, aspartate aminotransferase (AST) 2,086 U/L, total bilirubin 7.3 mg/dL, and direct bilirubin 4.9 mg/dL. Hepatitis B surface antigen was reactive (44.49 S/CO), while ultrasonography showed mild hepatomegaly, periportal edema, and gallbladder wall thickening without biliary obstruction. After approximately 10 days of hospitalization and supportive care, including nutritional supplementation, substantial biochemical improvement was observed by day 30: ALT 58 U/L, AST 59 U/L, total bilirubin 1.3 mg/dL, and direct bilirubin 0.59 mg/dL. Jaundice and presenting symptoms resolved. However, immunoglobulin M hepatitis B core antibody, hepatitis B e antigen/hepatitis B e antibody, and hepatitis B virus (HBV) DNA were not obtained; therefore, acute HBV infection could not be definitively confirmed.

Key words: Acute hepatitis, Case report, Hepatitis B virus, Hepatocellular injury, Hyperbilirubinemia, Liver function tests

Acute hepatitis is an inflammatory condition of the liver characterized by hepatocellular injury and variable impairment of hepatic synthetic and excretory function. It commonly presents with jaundice, fatigue, anorexia, and abdominal discomfort and is associated with elevations in aminotransferases and bilirubin [1,2]. Among causes of acute hepatitis, hepatitis B virus (HBV) remains important worldwide. HBV is a non-cytopathic, hepatotropic DNA virus, and liver injury is mainly mediated by the host cytotoxic T-lymphocyte response against infected hepatocytes [3]. In immunocompetent adults, fewer than 5% of acute HBV infections progress to chronic infection, compared with most perinatal or early-childhood infections [4]. Positive hepatitis B surface antigen (HBsAg) confirms HBV infection but does not distinguish acute from chronic infection with an acute flare; immunoglobulin M (IgM) hepatitis B core antibody (anti-HBc) is useful when acute infection is suspected [5]. Laboratory biomarkers are essential for assessing liver injury and monitoring progression. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) indicate hepatocellular injury, with ALT being more liver-specific [2,6]. Bilirubin, gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) provide information on excretory and hepatobiliary function, whereas albumin, total protein, and the A/G ratio provide information about synthetic function and systemic inflammation [6,7]. Abdominal ultrasonography complements biochemical assessment by identifying findings such as hepatomegaly, periportal edema, and gallbladder wall thickening, while excluding biliary obstruction and other structural causes of jaundice [8]. Given the variable course of acute hepatitis B and limited documentation of recovery trajectories in routine practice, structured reporting of serial biochemical, serological, and radiological findings remains valuable [9].

This report presents a 26-year-old woman with probable acute hepatitis B and severe hepatocellular injury followed prospectively for 30 days, illustrating the timeline of clinical and biochemical recovery and the role of serial monitoring.

CASE REPORT

A 26-year-old woman presented with progressive jaundice, generalized weakness and fatigue, reduced

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appetite, digestive discomfort, and persistent constipation. At presentation, physical examination was notable for jaundice and findings suggestive of hepatic dysfunction.

Baseline laboratory investigations demonstrated severe hepatocellular injury accompanied by marked hyperbilirubinemia. Radiological findings showed mild hepatomegaly with diffuse hypoechoic liver parenchyma and periportal edema, resulting in a "starry-sky" appearance. Mild gallbladder wall thickening was noted without gallstones or biliary obstruction. No ascites was observed. Serological testing demonstrated a reactive HBsAg result, with a value of 44.49 S/CO, confirming HBV infection. In conjunction with markedly elevated liver enzymes, hyperbilirubinemia, and ultrasonographic findings suggestive of acute hepatitis, these findings supported probable HBV-associated acute hepatitis as the likely cause of the patient's hepatocellular injury.

The patient remained hospitalized for approximately 10 days and underwent serial laboratory monitoring. The first follow-up laboratory assessment was performed on day 3, with significant biochemical improvement observed by day 10. The patient received routine inpatient medical care with serial laboratory monitoring, along with a structured nutritional support regimen initiated on admission and continued throughout the follow-up period. This regimen comprised a liver support supplement (2 tablets twice daily), a kidney support supplement (1 tablet twice daily), and a gut support supplement (1 tablet once daily), with the stated aims of supporting liver health, digestive function, and overall recovery.

Serial biochemical monitoring demonstrated substantial improvement in biochemical markers of hepatocellular and hepatobiliary injury over the 30-day follow-up period (Table 1) (Figs. 1 and 2). ALT decreased from 2,526 U/L to 58 U/L (97.7% reduction), and AST decreased from 2,086 U/L to 59 U/L (97.2% reduction), indicating substantial resolution of hepatocellular injury. GGT decreased from 140 U/L to 23.3 U/L (83.4% reduction), consistent with improvement in hepatobiliary abnormalities, while ALP decreased from 152 U/L to 74.2 U/L (51.2% reduction); the comparatively slower and more modest decline in ALP is consistent with its longer serum half-life and lower specificity for hepatocellular (as opposed to bone or other extrahepatic) turnover [6]. Total bilirubin decreased from 7.3 mg/dL to 1.3 mg/dL (82.2% reduction), while direct bilirubin decreased from 4.9 mg/dL to 0.59 mg/dL (88.0% reduction), indicating progressive restoration of hepatocellular excretory function and resolution of jaundice. Bilirubin rose slightly between day 1 and day 3 (total bilirubin 7.3 to 7.7 mg/dL) before declining continuously thereafter; this transient early rise despite a falling ALT is a recognized dissociation in acute hepatitis, since bilirubin clearance depends on hepatocyte conjugating and canalicular transport capacity, which may lag behind the fall in aminotransferases that follows cessation of active hepatocyte injury [6].

Albumin is synthesized exclusively by hepatocytes and, because of its long serum half-life (approximately 20 days), is a lagging indicator of hepatic synthetic function rather than a marker of acute injury; a fall in albumin during acute illness more often reflects a negative acute-phase response and redistribution than an acute change in synthesis [10]. Albumin fell transiently at day 3 (4.20–3.70 g/dL) before rising to 4.50 g/dL by day 30, while globulin rose from 3.20 to a peak of 4.13 g/dL at day 10, a pattern compatible with an inflammatory or immune response, before partially declining to 3.60 g/dL by day 30; total globulin alone does not identify a specific immunoglobulin fraction or adaptive mechanism, and immunoglobulin electrophoresis was not performed. The A/G ratio, which reflects the reciprocal changes in albumin and globulin, decreased from 1.30 to 1.00 during the acute phase and recovered to 1.25 by day 30 [10].

Overall, the progressive decline in hepatocellular and hepatobiliary injury markers, resolution of hyperbilirubinemia, and recovery of the serum protein profile indicated substantial biochemical recovery. Following discharge, monitoring continued through day 30, when liver function parameters had approached near-normal levels, accompanied by progressive clinical improvement and resolution of symptoms.

DISCUSSION

This case describes a 26-year-old woman with probable acute HBV-associated severe hepatocellular injury and marked hyperbilirubinemia, followed for 30 days. At presentation, ALT (2,526 U/L) and AST (2,086 U/L) were markedly elevated, with a total bilirubin level of 7.3 mg/dL and a direct bilirubin level of 4.9 mg/dL, consistent with severe hepatocellular injury and clinically evident jaundice. The disproportionate elevation of aminotransferases relative to GGT and ALP supported a predominantly hepatocellular rather than cholestatic

Table 1: Laboratory investigations of the patient

Parameter	Day 1	Day 3	Day 10	Day 30	Percentage change	Reference interval
ALT (SGPT) [U/L]	2,526	1,669	259	58	↓97.7	0–59
AST (SGOT) [U/L]	2,086	767	85	59	↓97.2	0–37
GGT (GGTP) [U/L]	140	123	61	23.3	↓83.4	9–36
ALP [U/L]	152	142	152	74.2	↓51.2	40–150
Total bilirubin (mg/dL)	7.3	7.7	3.7	1.3	↓82.2	0.30–1.20
Direct bilirubin (mg/dL)	4.9	5.1	3.36	0.59	↓88.0	0.0–0.50

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, GGT: Gamma-glutamyl transferase, ALP: Alkaline phosphatase, SGPT: Serum glutamic pyruvic transaminase, SGOT: Serum glutamic oxaloacetic transaminase, GGTP: Gamma-glutamyl transpeptidase

pattern [6]. In acute HBV infection, hepatocellular injury is primarily mediated by the host immune response, particularly cytotoxic T-lymphocyte activity against infected hepatocytes [3].

Other causes of severe hepatocellular injury were considered. Hepatitis A and E typically present similarly but were not specifically excluded serologically in this case; hepatitis C was considered less likely based on the clinical and biochemical presentation. Drug-induced liver injury was considered unlikely in the absence of a temporally associated hepatotoxic medication or supplement exposure before presentation. Ischemic hepatitis was considered unlikely in the absence of a hypotensive or hypoxic precipitant.

Bilirubin increased slightly during the initial days before declining progressively. By day 30, total and direct bilirubin had decreased by more than 80%, accompanied by resolution of jaundice, consistent with recovery of hepatic excretory function. GGT declined by 83.4%, whereas ALP showed a slower decline of 51.2% (Figs. 1 and 2). The differing kinetics may reflect differences in enzyme clearance and tissue sources, with aminotransferases being more reflective of active hepatocellular injury [6]. The absence of biliary dilatation on ultrasonography further argued against an obstructive process. The transient reduction in albumin with increased globulin and a lower A/G ratio was compatible with an acute inflammatory response. Albumin has a relatively long half-life and is therefore not a reliable marker of acute hepatic synthetic dysfunction over a short period [10]. By day 30, the protein profile had improved toward baseline.

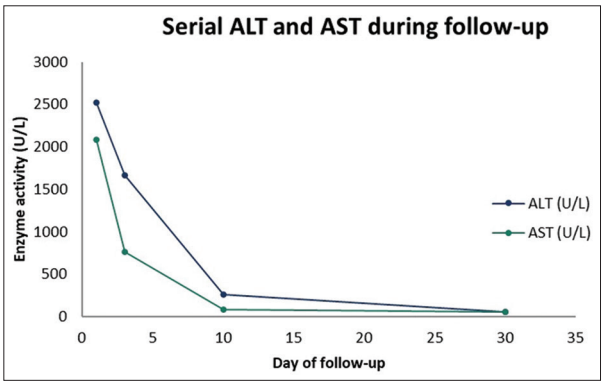


Figure 1: Serial changes in alanine aminotransferase and aspartate aminotransferase from day 1 to day 30

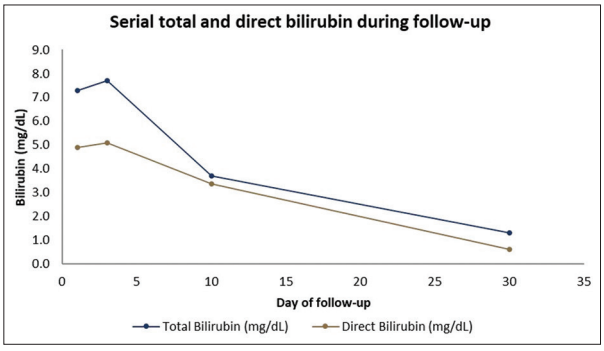


Figure 2: Serial changes in total and direct bilirubin from day 1 to day 30

Ultrasonography demonstrated mild hepatomegaly, periportal edema, and mild gallbladder wall thickening without biliary obstruction. These findings supported acute hepatic inflammation while helping exclude structural causes of jaundice such as cholelithiasis or biliary obstruction [8].

HBsAg was reactive (44.49 S/CO), supporting current HBV infection. However, HBsAg positivity alone cannot distinguish acute infection from previously unrecognized chronic infection with acute exacerbation. IgM anti-HBc, hepatitis B e antigen (HBeAg)/hepatitis B e antibody (anti-HBe), and HBV DNA were not obtained; therefore, acute HBV infection could not be confirmed definitively [5]. The diagnosis should consequently be considered probable rather than confirmed. The substantial biochemical and clinical improvement by day 30 was compatible with recovery from acute HBV-associated hepatocellular injury. In immunocompetent adults, progression from acute to chronic HBV infection occurs in fewer than 5% of cases [4]. However, biochemical recovery does not establish viral clearance; longer-term follow-up with HBsAg and hepatitis B surface antibody testing would be required.

Because this was a single-patient observational report without a comparator group, the improvement cannot be attributed causally to the nutritional support regimen. The observed recovery is compatible with the clinical course of HBV-associated acute hepatocellular injury managed with supportive care; however, the absence of definitive virological markers limits confirmation of the infection phase.

The limitations included the absence of key virological markers (IgM anti-HBc, HBeAg/anti-HBe, HBV DNA), the absence of coagulation parameters (prothrombin time/International Normalized Ratio) and blood glucose, lack of a comparator group, and limited 30-day follow-up. Nevertheless, the case provides a detailed example of the serial clinical and biochemical recovery pattern associated with probable acute HBV-related hepatocellular injury and highlights the importance of comprehensive HBV serological assessment and longitudinal biochemical monitoring.

CONCLUSION

This case report describes marked clinical and biochemical improvement in a 26-year-old woman with probable acute hepatitis B-associated hepatocellular injury. Reactive HBsAg, markedly elevated aminotransferases, hyperbilirubinemia, and ultrasonographic evidence of hepatic inflammation without biliary obstruction supported HBV-associated acute hepatocellular injury; however, absence of IgM anti-HBc, HBeAg/anti-HBe, and HBV DNA testing precluded definitive confirmation of newly acquired infection. During 30 days of follow-up, substantial reductions in ALT, AST, bilirubin, GGT, and ALP were observed, accompanied by resolution of jaundice and clinical symptoms. This case highlights

the importance of integrating serology, imaging, and serial biochemical monitoring in evaluating HBV-associated liver injury, while emphasizing that longer-term serological and virological follow-up is required to confirm viral clearance and establish the infection phase.

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