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Liver Function Abnormalities in Viral Hepatitis: A Biochemical and Microbiological Analysis

[1]Dr.P.Sushma,[2]Dr.Madhuri Musunuru,[3] T. Uma Maheswari
[1]*Assistant Professor Department of Biochemistry Maheshwara Medical College and Hospital*
[2]*Associate professor Department of Microbiology Maheshwara medical College*
[3]*Asst professor Department of microbiology Maheshwara medical college*

Correspondence:
Dr. Mohammed Sabiullah

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Abstract

Introduction: Viral hepatitis remains a major global health concern, contributing significantly to liver-related morbidity and mortality. Hepatitis A, B, C, D, and E viruses are responsible for acute and chronic liver disease, with biochemical liver dysfunction serving as a key diagnostic and prognostic indicator. This study aimed to evaluate liver function abnormalities in patients with viral hepatitis and correlate microbiological markers with biochemical derangements. ***Materials and Methods:*** This hospital-based cross-sectional study included 150 patients diagnosed with viral hepatitis. Serological markers (HBsAg, Anti-HCV, Anti-HAV IgM, Anti-HEV IgM) were detected using ELISA. Liver function parameters including serum bilirubin, AST, ALT, ALP, total protein, and albumin were measured using automated biochemical analyzers. Statistical analysis was performed using SPSS software. ***Results:*** Elevated ALT (92%) and AST (88%) were the most common abnormalities. Hyperbilirubinemia was observed in 76% of cases. Hepatitis B was the most prevalent etiology (46%), followed by Hepatitis C (28%). Significant correlation was observed between viral load and transaminase elevation ($p<0.05$). ***Conclusion:*** Liver enzyme derangements are significantly associated with viral hepatitis infections. Combined microbiological and biochemical assessment enhances early diagnosis and disease monitoring.

Keywords: Viral hepatitis, Liver function tests, ALT, AST, HBsAg, ELISA

INTRODUCTION

Viral hepatitis is a systemic infectious disease primarily affecting the liver and remains a substantial global health burden[1]. According to the World Health Organization, approximately 296 million individuals live with chronic hepatitis B and 58 million with chronic hepatitis C worldwide[2]. Acute and chronic viral hepatitis contribute to cirrhosis, hepatocellular carcinoma, and liver failure[3].

The five primary hepatotropic viruses—HAV, HBV, HCV, HDV, and HEV—differ in transmission patterns, clinical progression, and long-term complications[4]. Hepatitis A and E are usually self-limiting, whereas hepatitis B and C frequently progress to chronicity[5]. Chronic viral hepatitis leads to progressive liver inflammation, fibrosis, and eventual cirrhosis[6].

Liver function tests (LFTs) serve as important biomarkers in assessing hepatocellular injury and cholestasis[7]. Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are indicators of hepatocellular damage[8]. Elevated bilirubin reflects impaired hepatic excretory function[9]. Alkaline phosphatase (ALP) elevation may suggest cholestatic involvement[10].

Microbiological diagnosis relies on detection of viral antigens, antibodies, and nucleic acid amplification tests[11]. ELISA remains a cornerstone for serological detection[12]. Early identification of biochemical abnormalities assists in staging disease severity and monitoring therapeutic response[13].

The interplay between viral replication and host immune response determines biochemical injury patterns[14]. Studies have demonstrated that ALT levels correlate with viral activity, especially in HBV and HCV infections[15]. Persistent elevation indicates ongoing necroinflammatory activity[16].

Despite advances in antiviral therapy, liver-related mortality remains high in developing countries due to late diagnosis[17]. Therefore, integrated microbiological

and biochemical evaluation is critical for early intervention[18].

This study aims to analyze liver function abnormalities in viral hepatitis and correlate them with microbiological findings in a tertiary care setting.

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted over a period of 18 months in a tertiary care center to evaluate the microbiological and biochemical profile of patients with viral hepatitis. A total of 150 patients aged between 18 and 65 years who were clinically suspected of viral hepatitis were enrolled in the study.

Patients were included if they were aged 18 years or above, had serologically confirmed viral hepatitis evidenced by positivity for HBsAg, Anti-HCV, Anti-HAV IgM, or Anti-HEV IgM, and demonstrated elevated liver enzyme levels. Patients with alcoholic liver disease, drug-induced hepatitis, autoimmune hepatitis, non-alcoholic fatty liver disease, chronic kidney disease, or pregnancy were excluded to avoid confounding factors that could influence liver function parameters.

Following informed consent, approximately 5 mL of fasting venous blood was collected under strict aseptic precautions from each participant. The samples were allowed to clot, centrifuged, and serum was separated for microbiological and biochemical analysis. Serological markers including HBsAg, Anti-HCV, Anti-HAV IgM, and Anti-HEV IgM were detected using enzyme-linked immunosorbent assay (ELISA) techniques according to the manufacturer's instructions. In selected cases of hepatitis B and hepatitis C infection, viral load quantification was performed using polymerase chain reaction (PCR) methods to assess the degree of viral replication.

Biochemical parameters were analyzed using standardized automated techniques. Total bilirubin was estimated by the Diazo method. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured using the IFCC kinetic method. Alkaline phosphatase (ALP) levels were determined by the p-nitrophenyl phosphate (pNPP) method. Total protein estimation was carried out using the Biuret method, while serum albumin was measured by the Bromocresol green method. All analyses were performed in accordance with standard laboratory protocols with appropriate quality control measures.

Statistical Analysis

The collected data were compiled and statistically analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. The Chi-square test was applied to assess associations between categorical variables, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Distribution

Age Group	Number	Percentage
18–30	40	26.7%
31–45	62	41.3%
46–60	34	22.7%
>60	14	9.3%

Table 1 shows the age-wise distribution of patients with viral hepatitis. The majority of cases (41.3%) were observed in the 31–45 years age group, followed by 26.7% in the 18–30 years group.

Table 2: Gender Distribution of Study Participants

Gender	Number (n=150)	Percentage (%)
Male	94	62.7%
Female	56	37.3%

The study population showed male predominance, with males constituting 62.7% of the cases.

Table 3: Etiological Distribution

Virus	Cases	%
HBV	69	46%
HCV	42	28%
HAV	21	14%
HEV	18	12%

Table 3 demonstrates the distribution of viral hepatitis types among study participants. Hepatitis B virus (46%) was the most common etiological agent, followed by Hepatitis C (28%), Hepatitis A (14%), and Hepatitis E (12%).

Table 4: Serum Bilirubin Levels

Level	Cases
<1 mg/dL	36
1–3 mg/dL	62
>3 mg/dL	52

76% showed hyperbilirubinemia.

Table 5: Transaminase Levels

Parameter	Elevated (%)
ALT	92%
AST	88%

Table 5 shows that ALT was elevated in 92% and AST in 88% of cases.

Table 6: ALP Levels

Normal	Elevated
98	52

34% showed cholestatic pattern.

Table 7: Protein and Albumin Levels

Parameter	Mean ± SD
Total Protein	6.2 ± 0.8
Albumin	3.4 ± 0.6

Mild hypoalbuminemia observed in chronic cases.

Table 8: Serum Ammonia Levels in Viral Hepatitis Patients (n = 150)

Serum Ammonia Level (µmol/L)	Number of Cases	Percentage (%)
≤ 50 (Normal)	82	54.7%
51–100 (Mild Elevation)	44	29.3%
101–150 (Moderate Elevation)	18	12.0%
>150 (Severe Elevation)	6	4.0%

Elevated serum ammonia levels were observed in 45.3% of patients.

Table 9: Prothrombin Time (PT) and INR in Viral Hepatitis Patients (n = 150)

Parameter	Category	Number of Cases	Percentage (%)
Prothrombin Time (seconds)	≤ 14 sec (Normal)	88	58.7%
	15–18 sec (Mild Prolongation)	38	25.3%
	>18 sec (Significant Prolongation)	24	16.0%
INR	≤ 1.2 (Normal)	92	61.3%
	1.3–1.8 (Mild Elevation)	36	24.0%
	>1.8 (Marked Elevation)	22	14.7%

Prolonged Prothrombin Time was observed in 41.3% of patients, indicating impaired hepatic synthetic function. INR elevation was noted in 38.7% of cases, with marked elevation predominantly seen in chronic hepatitis and severe acute liver injury.

Table 10: Serum Lactate Dehydrogenase (LDH) Levels in Viral Hepatitis Patients (n = 150)

Serum LDH Level (U/L)	Number of Cases	Percentage (%)
≤ 250 (Normal)	58	38.7%
251–500 (Mild Elevation)	54	36.0%
501–750 (Moderate Elevation)	26	17.3%
>750 (Marked Elevation)	12	8.0%

Elevated LDH levels were observed in 61.3% of patients, indicating significant hepatocellular injury and ongoing cellular necrosis. Marked elevation (>750 U/L) was noted in 8% of cases, predominantly in severe acute hepatitis and fulminant liver injury.

Table 11: Serum Ferritin Levels in Viral Hepatitis Patients (n = 150)

Serum Ferritin Level (ng/mL)	Number of Cases	Percentage (%)
≤ 300 (Normal)	64	42.7%
301–600 (Mild Elevation)	46	30.7%
601–1000 (Moderate Elevation)	28	18.7%
>1000 (Severe Elevation)	12	8.0%

Elevated serum ferritin levels were seen in 57.3% of patients, reflecting inflammatory activity and hepatic injury. Severe elevation (>1000 ng/mL) was observed in 8% of cases, more commonly associated with chronic hepatitis and severe inflammatory response.

DISCUSSION

The present study evaluated liver function abnormalities in patients with viral hepatitis and demonstrated significant alterations in transaminases, bilirubin, serum ammonia, and coagulation parameters, consistent with contemporary global findings. Elevated ALT (92%) and AST (88%) were the most prominent biochemical abnormalities, indicating predominant hepatocellular injury. This observation aligns with findings from Younossi et al., who reported ALT elevation as the most sensitive marker of active hepatocellular inflammation in viral hepatitis. Similarly, Terrault et al. noted that ALT levels correlate closely with viral replication and immune-mediated hepatocyte damage, particularly in HBV and HCV infections.

Hyperbilirubinemia was observed in 76% of cases in the present study. Comparable findings were reported by Marcellin et al., who demonstrated that elevated bilirubin levels are more pronounced in acute viral hepatitis and during severe necroinflammatory activity. Bilirubin elevation reflects impaired hepatic conjugation and excretory dysfunction, which becomes clinically significant in moderate to severe disease.

The predominance of Hepatitis B virus (46%) in our study mirrors epidemiological trends in developing countries. According to recent WHO and Polaris Observatory reports, HBV remains the leading cause of chronic viral hepatitis globally. Dutta et al. in an Indian cohort similarly reported HBV as the most common etiological agent, reinforcing the regional burden.

Serum ammonia elevation was noted in nearly half of the study population, particularly among patients with advanced disease. Elevated ammonia levels reflect compromised hepatic detoxification and portosystemic shunting. EASL guidelines highlight hyperammonemia as a key contributor to hepatic encephalopathy in chronic liver disease. Our findings are consistent with Rockey et al., who demonstrated that rising ammonia levels correlate with severity of hepatic dysfunction.

Coagulation abnormalities were another significant finding. Prolonged Prothrombin Time and elevated INR were observed in 41.3% and 38.7% of cases, respectively. These abnormalities indicate impaired hepatic synthetic capacity due to reduced clotting factor production. Studies by Kim et al. and Seto et al. have emphasized PT and INR as strong prognostic markers in acute and chronic viral hepatitis, particularly in predicting progression to acute liver failure. The correlation between elevated bilirubin and prolonged PT observed in our study further supports the association between hepatocellular injury and synthetic dysfunction.

Mild hypoalbuminemia observed in chronic hepatitis cases reflects long-standing liver impairment. Similar biochemical patterns have been described in chronic HCV patients by Eddowes et al., where declining albumin levels were associated with advancing fibrosis.

Overall, our findings reinforce the importance of combined microbiological and biochemical evaluation in viral hepatitis. While serological markers confirm etiology, liver function tests provide insight into disease severity, inflammatory activity, and synthetic impairment. The patterns observed in this study are consistent with recent international literature, confirming that transaminases, bilirubin, ammonia, and coagulation parameters remain reliable indicators of hepatic injury and prognosis.

Early identification of these abnormalities allows timely therapeutic intervention and may prevent progression to cirrhosis and hepatocellular carcinoma. Therefore, routine monitoring of liver function parameters should remain integral to the management of viral hepatitis.

CONCLUSION

Liver function abnormalities are strongly associated with viral hepatitis infections. ALT elevation remains the most sensitive indicator of hepatocellular injury. Combined microbiological and biochemical analysis is essential for early diagnosis, disease monitoring, and prevention of long-term complications.

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