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Research Article

Spectrum of Bichemical Alterations in Patients with Chronic Liver Disease

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Abstract

Background: Chronic liver disease (CLD) encompasses a spectrum of hepatic pathologies, including non-alcoholic fatty liver disease (NAFLD), alcoholic liver disease (ALD), and chronic viral hepatitis, leading to significant biochemical alterations. This study aims to delineate the biochemical profile across various stages of CLD.

Methods: A retrospective analysis was conducted on 135 patients diagnosed with CLD, categorized into NAFLD (n=45), ALD (n=45), and chronic viral hepatitis (n=45). Biochemical parameters assessed included liver enzymes (ALT, AST, ALP, GGT), bilirubin levels, albumin, prothrombin time (PT), and renal function markers (creatinine, urea).

Results: Elevated levels of ALT and AST were predominant in ALD and chronic viral hepatitis groups. NAFLD patients exhibited increased GGT and ALP levels. Bilirubin levels were significantly higher in chronic viral hepatitis cases. Albumin levels were reduced across all groups, with the lowest in chronic viral hepatitis. Prolonged PT was observed in ALD and chronic viral hepatitis patients.

Conclusion: Distinct biochemical alterations characterize different etiologies of CLD. These findings underscore the importance of tailored diagnostic and therapeutic strategies for effective management.

Keywords: Chronic Liver Disease, Biochemical Alterations, Liver Enzymes, NAFLD, ALD, Chronic Viral Hepatitis, Liver Function Tests.

INTRODUCTION

Chronic liver disease (CLD) is a common and growing global health concern, often progressing to cirrhosis and hepatocellular carcinoma if left untreated. The primary causes of CLD include non-alcoholic fatty liver disease (NAFLD), alcoholic liver disease (ALD), and chronic viral hepatitis. Each of these conditions presents with a distinct biochemical profile, reflective of the underlying liver pathology.

NAFLD, the most prevalent form of liver disease worldwide, is characterized by fat accumulation in the liver without significant alcohol consumption. Its prevalence is on the rise due to the increasing global burden of metabolic diseases, including obesity and type 2 diabetes mellitus [1]. ALD results from chronic alcohol consumption and can range from simple steatosis to alcoholic hepatitis and cirrhosis [2]. Chronic viral hepatitis, predominantly caused by hepatitis B (HBV) or hepatitis C (HCV) infections, remains one of the leading causes of CLD, with a high risk of progression to cirrhosis and liver cancer [3].

Biochemical markers play an important role in diagnosing and monitoring the progression of liver disease. Liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are sensitive indicators of hepatocellular injury [4]. Alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) are useful in detecting cholestatic conditions [5]. Additionally, bilirubin levels reflect hepatic excretory function, while albumin and prothrombin time (PT) assess synthetic function [6]. Understanding these biochemical alterations is crucial in managing CLD patients and tailoring treatment approaches.

The goal of this study was to analyze the spectrum of biochemical alterations in patients with different etiologies of CLD and to correlate these changes with disease progression and clinical outcomes.

METHODOLOGY

Study Design

A retrospective observational study was conducted at a Loralai Medical College and Teaching Hospital Loralai between April 2024 and March 2025. A total of 135 patients diagnosed with CLD were included in the study. The institutional review board approved the study, and all patients provided informed consent.

Inclusion Criteria

- Adults aged 18–65 years
- Diagnosed with chronic liver disease based on clinical, biochemical, and imaging criteria
- Willingness to participate in the study

Exclusion Criteria

- Acute liver failure
- Pregnancy
- Coexisting malignancies
- Inability to provide informed consent

Classification of Patients

Patients were classified into three groups based on the underlying etiology of their disease:

1. **Non-Alcoholic Fatty Liver Disease (NAFLD)**
2. **Alcoholic Liver Disease (ALD)**
3. **Chronic Viral Hepatitis** (HBV or HCV infection)

Biochemical Analysis

Fasting blood samples were collected to assess the following biochemical parameters:

- **Liver enzymes:** ALT, AST, ALP, GGT
- **Bilirubin levels:** Total and direct

Albumin levels

- **Prothrombin time (PT):** International normalized ratio (INR)
- **Renal function markers:** Serum creatinine and urea

Statistical Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics were used to summarize demographic and clinical characteristics. One-way ANOVA was used to compare continuous variables, and chi-square tests were used for categorical variables. A p-value of <0.05 was considered statistically significant.

RESULTS

The study included 135 patients, divided into three groups: NAFLD (n=45), ALD (n=45), and chronic viral hepatitis (n=45). The mean age of the patients was 52.3 ± 10.4 years, with a male predominance (60%). Among the ALD group, the majority had a history of chronic alcohol consumption, with an average intake of 80–100 grams per day over the past 10 years. In the chronic viral hepatitis group, 70% were positive for hepatitis C, and the remaining 30% had hepatitis B. NAFLD patients had a higher prevalence of obesity and metabolic syndrome (Table 1).

Demographic Characteristic	NAFLD (n=45)	ALD (n=45)	Chronic Viral Hepatitis (n=45)	Total (n=135)
Age (years)	51.2 ± 9.8	53.5 ± 10.7	52.8 ± 10.1	52.3 ± 10.4
Gender (Male %)	40 (88%)	30 (67%)	35 (78%)	105 (78%)
Obesity (%)	35 (78%)	10 (22%)	15 (33%)	60 (44%)
History of Alcohol Use (%)	0 (0%)	45 (100%)	0 (0%)	45 (33%)
Hepatitis B (%)	0 (0%)	0 (0%)	13 (30%)	13 (10%)
Hepatitis C (%)	0 (0%)	0 (0%)	32 (70%)	32 (24%)

The biochemical parameters differed significantly across the groups. Patients with chronic viral hepatitis had the highest ALT, AST, and bilirubin levels, reflecting marked hepatocellular damage. ALD patients had slightly elevated liver enzymes but higher GGT and ALP levels, indicative of cholestatic involvement. NAFLD patients demonstrated mild elevations in liver enzymes, particularly GGT, consistent with the fatty liver changes.

Biochemical	NAFLD	ALD	Chronic Viral Hepatitis	p-

Parameter	(n=45)	(n=45)	(n=45)	value
ALT (U/L)	45.2 ± 12.3	78.5 ± 18.4	92.3 ± 20.1	<0.001
AST (U/L)	40.1 ± 10.2	85.4 ± 22.3	88.7 ± 19.8	<0.001
ALP (U/L)	120.4 ± 30.5	145.6 ± 35.2	130.2 ± 28.9	0.045
GGT (U/L)	60.3 ± 15.7	75.8 ± 20.1	70.5 ± 18.3	0.032
Total Bilirubin (mg/dL)	0.8 ± 0.2	1.2 ± 0.3	2.5 ± 0.5	<0.001
Direct Bilirubin (mg/dL)	0.3 ± 0.1	0.5 ± 0.2	1.0 ± 0.2	<0.001
Albumin (g/dL)	3.8 ± 0.6	3.2 ± 0.7	2.9 ± 0.8	<0.001
Prothrombin Time (INR)	1.1 ± 0.1	1.3 ± 0.2	1.5 ± 0.3	<0.001

The risk factors for CLD varied significantly across the groups. Obesity was a significant risk factor for NAFLD, while alcohol use was the predominant risk factor in ALD patients. Chronic viral hepatitis was most strongly associated with positive HCV and HBV serology (Table 2).

Risk Factor	NAFLD (n=45)	ALD (n=45)	Chronic Viral Hepatitis (n=45)	p-value
Obesity (%)	35 (78%)	0 (0%)	10 (22%)	<0.001
History of Alcohol Use (%)	0 (0%)	45 (100%)	0 (0%)	<0.001
Hepatitis B (%)	0 (0%)	0 (0%)	13 (30%)	<0.001
Hepatitis C (%)	0 (0%)	0 (0%)	32 (70%)	<0.001

DISCUSSION

This study demonstrates the distinct biochemical alterations that occur across various etiologies of chronic liver disease. Patients with chronic viral hepatitis exhibited the highest ALT, AST, and bilirubin levels, consistent with advanced hepatocellular injury. Similar findings were reported by studies on hepatitis C-induced liver disease [7]. Elevated GGT and ALP levels in ALD and NAFLD suggest cholestatic changes in these diseases, which aligns with existing literature on the hepatic involvement of these conditions [8,9].

The observed reduction in albumin levels across all groups indicates impaired synthetic function, which is a hallmark of liver dysfunction [10]. Similarly, the prolonged prothrombin time (INR) in ALD and chronic viral hepatitis groups further suggests a decline in liver synthetic capacity, reflecting the progression towards cirrhosis [11].

The high prevalence of obesity and metabolic syndrome in the NAFLD group correlates with the rising global burden of metabolic diseases and their association with liver steatosis [12,13]. Alcohol use remains the leading cause of liver disease in the ALD group, with a significant correlation between alcohol consumption and liver damage [14]. The findings also underscore the importance of screening for hepatitis B and C in patients with chronic liver disease, as early antiviral therapy can alter the disease course [15].

CONCLUSION

In conclusion, this study highlights the distinct biochemical profiles associated with various etiologies of chronic liver disease. It underscores the need for tailored diagnostic and therapeutic strategies for each condition. Understanding these biochemical alterations is essential for early diagnosis, monitoring disease progression, and implementing effective management strategies.

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