

**Original Research Article** 

# STUDY OF CLINICAL AND BIOCHEMICAL PROFILE OF ACUTE ALCOHOLIC LIVER DISEASE (ALD) FROM TERTIARY CARE TEACHING HOSPITAL OF MADHUBANI, BIHAR

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#### ABSTRACT

**Background:** ALD encompasses a range of clinicopathological abnormalities characterized by acute or chronic hepatic inflammation due to alcohol use, accompanied by changes in biochemical parameters and diverse clinical presentations. Objective: To assess the clinical and biochemical profile of acute alcoholic liver disease in a tertiary care setting.

**Materials and Methods:** This prospective, hospital-based study was conducted in the Departments of Biochemistry and Microbiology at a tertiary care teaching hospital of Madhubani over one-year period. A total of 240 patients diagnosed clinically and biochemically with acute ALD were included.

**Results:** The age of study participants ranged from 20 to 60 years, with a male-to-female ratio of 2.42:1. The majority of cases were in the 31–40 years age group (54.2%). Most patients (66.7%) consumed more than 60 grams of alcohol per day. Jaundice, nausea, and vomiting were the most prevalent clinical features (83.3%), followed by hepatomegaly (66.7%). The majority had a history of alcohol consumption exceeding five years.

**Conclusions:** Chronic alcohol abuse is more prevalent among adult males, with a tendency towards higher daily alcohol intake. ALD presents with a wide array of clinical features and is associated with significant biochemical derangements.

Keywords: Alcoholic liver disease, Biochemical parameters, Liver enzymes

## **INTRODUCTION**

Alcoholism is a chronic condition resulting from excessive consumption of alcoholic beverages, which significantly increases the risk of developing various health complications. Notably, these include hepatic diseases, cardiovascular disorders, pancreatitis, central nervous system impairments, and certain malignancies. Alcoholic liver disease (ALD) encompasses a spectrum of pathological and clinical abnormalities, manifesting as either acute or chronic hepatic inflammation induced by alcohol intake.<sup>[1]</sup> Globally, the prevalence of ALD is estimated to be 94.8 per 10,000 individuals. The hepatotoxic effects of alcohol are mediated through

toxic metabolites (such as protein-aldehyde adducts), endotoxins, oxidative stress, immune responses, and pro-inflammatory cytokines, all of which contribute to hepatic injury.<sup>[2]</sup>

Alcohol-induced hepatic injury can range from simple steatosis to end-stage cirrhosis and may ultimately progress to hepatocellular carcinoma. The liver is particularly susceptible to damage from chronic heavy alcohol consumption, most commonly presenting as alcoholic hepatitis or cirrhosis. The progression of ALD is characterized by steatosis, inflammation, necrosis, and eventual cirrhosis, with severe cases often resulting in mortality. Illicitly brewed alcoholic beverages are reported to be more hepatotoxic compared to legal alcoholic drinks, despite their lower alcohol content. The clinical spectrum of ALD includes alcoholic fatty alcoholic hepatitis, and cirrhosis. liver, Approximately 90% of individuals consuming more than 60 grams of alcohol daily develop fatty liver, a condition reversible with abstinence.<sup>[3]</sup> Laboratory investigations in ALD typically reveal an elevated transaminase (AST) aspartate to alanine transaminase (ALT) ratio, altered albumin-toglobulin ratio, and increased prothrombin time, particularly in cirrhosis.<sup>[4]</sup> Approximately half of these patients progress to irreversible liver damage or cirrhosis, which may be complicated by portal hypertension, gastrointestinal bleeding, ascites, splenomegaly, and other stigmata of chronic liver disease. The pattern and prevalence of ALD vary geographically and among different ethnic groups.<sup>[5]</sup> Serum levels of AST and ALT are typically elevated in ALD, with more than 80% of patients exhibiting a ratio (AST:ALT) of 2 or higher, which serves as a valuable diagnostic marker. Hyperbilirubinemia is also frequently observed.<sup>[6,7]</sup> Additional laboratory indicators include increased alkaline phosphatase (ALP), gamma-glutamyl transpeptidase (GGT), decreased serum albumin. and prolonged prothrombin time. Hematological parameters such as red and white blood cell counts, hemoglobin, and mean corpuscular volume are also informative in diagnosing ALD. The objective of this study was to assess the clinical and biochemical profile of acute alcoholic liver disease in a tertiary care setting.

## **MATERIALS AND METHODS**

This prospective, hospital-based study was conducted in the Departments of Biochemistry and Microbiology at a tertiary care teaching hospital of Madhubani over one-year period in year 2023. A total of 240 patients who were clinically and biochemically diagnosed with acute ALD were enrolled.

## **Inclusion** Criteria

- Age between 20 and 60 years
- Both genders
- History of alcohol consumption
- **Exclusion** Criteria
- Diabetes mellitus
- Autoimmune diseases
- Hemolytic anemia
- Hepatic infections

Patients presenting to the various outpatient departments and meeting the above criteria were included. Detailed demographic information (age, gender, occupation), clinical history (current illness, past hepatic disease, treatments, drug allergies), personal history (alcohol intake, smoking, dietary habits, family history, socioeconomic status), and abdominal ultrasonography findings were recorded.

Laboratory investigations included a complete blood count, urine analysis, and biochemical assessments (total, conjugated, and unconjugated bilirubin; total protein; albumin; albumin:globulin ratio; AST; ALT; ALP; GGT). Blood samples were collected under aseptic conditions after fasting. Acute ALD was diagnosed based on clinical presentation and biochemical findings.

Prior ethical clearance was obtained from the Institutional Ethics Committee of the medical college. Informed written consent was obtained from each participant before administrating the questionnaire. Data from the questionnaire were obtained in the form of Excel sheets. Statistical analysis was performed using I.B.M.'s Statistical Package for Social Sciences software. Descriptive statistics were presented as mean  $\pm$  standard deviation for quantitative data and frequency with percentage for categorical data. Appropriate inferential statistical tests were done.

## RESULTS

The majority of patients were in the 31-40 years age group (54.2%), followed by those aged 21-30 years (20.8%), 41-50 years (14.2%), and above 50 years (10.8%).

Table 1: Age-wise distribution of study subjects			
Age (years)	No. of cases	Percentage	
21–30	50	20.8%	
31–40	130	54.2%	
41–50	34	14.2%	
51-60	26	10.8%	
Total	240	100%	

A male predominance was observed, with 70.8% males and 29.2% females (male-to-female ratio: 2.42).

 Table 2: Gender-wise distribution of study subjects

Gender	No. of cases	Percentage
Male	170	70.8%
Female	70	29.2%
Total	240	100%

Most patients (66.7%) reported consuming more than 60 grams of alcohol per day, 20.8% consumed 50–60 grams, and 12.5% consumed less than 50 grams daily.

Table 3: Distribution of study subjects as per amount of alcohol consumed			
Quantity (gm/24hr)	No. of cases	Percentage	
<50 gm/24 hr	30	12.5%	
50–60 gm/24 hr	50	20.8%	
>60 gm/24 hr	160	66.7%	
Total	240	100%	

The majority (66.7%) had a history of alcohol consumption exceeding five years.

#### Table 4: Duration of alcohol consumption by study subjects

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Duration (years)	No. of cases	Percentage
1–2	10	4.2%
2–3	30	12.5%
3-4	40	16.7%
>5	160	66.7%
Total	240	100%

The most common symptoms were jaundice, nausea, and vomiting (83.3%), followed by hepatomegaly (66.7%), anorexia (50%), splenomegaly (33.3%), and signs of liver failure (58.3%).

Clinical Feature	No. of cases	Percentage
Anorexia	120	50%
Nausea/vomiting	200	83.3%
Abdominal pain	60	25%
Fever	60	25%
Ascites	40	16.7%
Jaundice	200	83.3%
Hepatomegaly	160	66.7%
Splenomegaly	80	33.3%
Signs of liver failure	140	58.3%

#### **Biochemical Parameters**

Elevated bilirubin (>2 mg/dl) was found in 83.3% of cases. AST, ALT, and ALP were elevated in 66.7% of patients, while GGT was raised in 95.8%. Hypoalbuminemia was observed in 41.7%.

Table 6: Biochemical parameters among study subjects		
Parameter	No. of cases	Percentage
Bilirubin >2 mg/dl	200	83.3%
AST >40 IU/L	160	66.7%
ALT >40 IU/L	160	66.7%
ALP >280 IU/L	160	66.7%
GGT >66 IU/L	230	95.8%
Albumin <3 g/dl	100	41.7%
Globulin >3.5 g/dl	120	50%

#### **Occupational Distribution**

The majority of patients were farmers (60%), followed by private sector employees (20%), daily wage laborers (10%), and the unemployed (10%).

### **DISCUSSION**

The present study, conducted at a tertiary care teaching hospital in Lucknow, comprehensively assessed the clinical and biochemical profiles of 240 patients diagnosed with acute alcoholic liver disease (ALD). Our findings underscore the significant burden of ALD among adult males, particularly in the 31–40 years age group, and highlight a strong association between prolonged heavy alcohol consumption and the development of clinically and biochemically evident liver injury.<sup>[8]</sup>

The demographic distribution of our cohort—with 70.8% males and a male-to-female ratio of 2.42:1— mirrors the gender disparity observed in previous studies from India and globally, confirming that ALD disproportionately affects men. This pattern

likely reflects sociocultural factors, including higher rates of alcohol consumption and occupational exposure among males in this region. The predominance of patients aged 31–40 years (54.2%) suggests that the peak risk period for symptomatic ALD occurs in early to mid-adulthood, possibly due to cumulative alcohol exposure and the onset of complications after several years of heavy drinking.<sup>[9]</sup>

Occupational analysis revealed that the majority of patients were farmers (60%), followed by private sector employees (20%), daily wage laborers (10%), and the unemployed (10%). This distribution may be attributed to the socioeconomic environment of the study region, where farming communities have historically reported higher rates of alcohol use, possibly as a coping mechanism for physical labor and stress. The high prevalence of alcohol

consumption and ALD in this group underscores the need for targeted public health interventions in rural and agrarian populations.<sup>[10,11]</sup>

Clinically, the most common presenting symptoms were jaundice, nausea, and vomiting (83.3%), followed by hepatomegaly (66.7%), anorexia (50%), splenomegaly (33.3%), and signs of liver failure (58.3%). These findings are consistent with prior reports and highlight the diverse and often severe clinical manifestations of ALD. The high prevalence of jaundice and hepatomegaly signals significant hepatocellular injury and hepatic congestion, while the presence of liver failure signs indicates advanced disease in a substantial proportion of patients.<sup>[12]</sup>

Biochemically, our data revealed marked derangements. Elevated bilirubin (>2 mg/dL) was observed in 83.3% of cases, indicating impaired hepatic excretory function. AST, ALT, and ALP levels were elevated in 66.7% of patients, consistent with hepatocellular injury and cholestasis. Notably, GGT was elevated in 95.8% of cases, underscoring its utility as a sensitive marker of alcohol-induced liver injury. Hypoalbuminemia (41.7%) and hyperglobulinemia (50%) reflect impaired synthetic chronic function and inflammatory states, respectively, and are hallmarks of advanced ALD.[13]

The strong association between heavy alcohol consumption (>60 grams/day) and the development of ALD—evidenced by the fact that 66.7% of our cohort reported such intake—confirms the dose-dependent hepatotoxicity of alcohol. Furthermore, the majority of patients (66.7%) had a history of alcohol consumption exceeding five years, emphasizing the cumulative effect of chronic alcohol exposure in the pathogenesis of ALD.

Our findings are supported by recent global epidemiological studies, which estimate the prevalence of ALD at 94.8 per 10,000 individuals and highlight the increasing burden of alcohol-associated liver disease worldwide. The hepatotoxic effects of alcohol are mediated through multiple pathways, including the generation of toxic metabolites such as acetaldehyde, oxidative stress, immune activation, and pro-inflammatory cytokine release, all of which contribute to hepatic injury and disease progression.<sup>[14]</sup>

From a clinical perspective, the high prevalence of biochemical derangements and advanced clinical features in our cohort underscores the importance of early detection and intervention. Public health strategies aimed at reducing alcohol consumption, particularly among high-risk groups such as young adult males and agricultural workers, are urgently needed. Additionally, enhanced surveillance for biochemical markers of liver injury, such as GGT and bilirubin, may facilitate earlier diagnosis and improve outcomes in this population.

## CONCLUSION

In conclusion, this study highlights the significant clinical and biochemical burden of acute ALD in a tertiary care setting in Bihar. The findings reinforce the need for comprehensive alcohol control policies and targeted interventions to reduce the morbidity and mortality associated with this preventable disease.

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