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TO STUDY THE CLINICAL SPECTRUM OF ALCOHOL LIVER DISEASE IN TERTIARY CARE CENTER

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Abstract

Background: Alcoholic liver disease (ALD) is a major contributor to global morbidity and mortality, encompassing a spectrum of conditions from steatosis to cirrhosis.

AIM: To evaluate the demographic profile, alcohol intake patterns, and clinical spectrum of ALD in patients at a tertiary care hospital in India.

Material and Methods: A single-center, hospital-based cross-sectional study was conducted over 18 months, including 100 adult patients with confirmed ALD. Data on demographics, alcohol consumption patterns, and clinical features were collected through structured interviews and clinical assessments. Laboratory parameters and ultrasound findings were also analyzed.

Results: Participants were predominantly male (92%), with a mean age of 48 years. The prevalence of alcoholic cirrhosis was 70% (including 28 patients with decompensated cirrhosis) and 30% had hepatic steatosis (fatty liver) and alcoholic hepatitis. Heavy alcohol consumption (≥720 units/month) was strongly associated with advanced ALD, including cirrhosis (86%) and decompensated cirrhosis (38%) ($p < 0.001$). Chronic alcohol consumption (>20 years) correlated with higher prevalence of cirrhosis (89%). Common clinical manifestations included jaundice (55%), ascites (35%), and hepatic encephalopathy (15%). Laboratory findings revealed elevated SGOT/SGPT ratios (>2), hypoalbuminemia, and anemia. Ultrasound abnormalities such as coarse liver echotexture were prevalent in 70%.

Conclusion: This study highlights the role of heavy and prolonged alcohol consumption in disease progression. The findings underscore the need for targeted interventions to reduce alcohol-related liver damage and improve clinical outcomes.

Keywords: Jaundice, ascitis, decompensated cirrhosis, chronic Alcoholic consumption

Introduction

Alcoholic liver disease (ALD) represents a major global health challenge, contributing significantly to morbidity 4.4% and mortality 3.7 %[1]. ALD encompasses a spectrum of liver pathologies directly attributable to excessive alcohol consumption, ranging from simple steatosis (fatty liver) to more severe conditions such as alcoholic hepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma (HCC)[2]. The progression of ALD is influenced by a complex interplay of factors, including the

duration and quantity of alcohol intake, genetic predispositions, comorbid conditions, and lifestyle factors[2]. Chronic alcohol consumption leads to hepatic injury through various biochemical mechanisms, primarily driven by oxidative stress, acetaldehyde toxicity, and inflammation[3]. Excessive alcohol intake results in the accumulation of reactive oxygen species (ROS), overwhelming the liver's antioxidant defenses and inducing lipid peroxidation[3]. The toxic effects of acetaldehyde, a byproduct of ethanol metabolism, further compound this damage by impairing cellular repair mechanisms and promoting fibrosis[4].

India study. According to NFHS-5 (National family health survey) 18.8 % men and 1.3 %women consume alcohol [5,6].
The clinical presentation of ALD varies widely[7,8]. Many patients are asymptomatic in the early stages, with steatosis often detected incidentally. As the disease progresses, symptoms can range from non-specific manifestations, such as fatigue and nausea, to severe complications like jaundice, ascites, hepatic encephalopathy, and variceal bleeding in cases of advanced cirrhosis[7,8].
Understanding the demographic profile, alcohol consumption patterns, and clinical spectrum of ALD is essential to provide timely interventions, improve patient outcomes, and reduce the healthcare burden associated with advanced liver disease.
Hence we tried to study the spectrum of alcohol liver disease, including factors such as age ,gender, alcohol intake patterns in tertiary care center .

Material and Methods

- **Study Design:** A single centre, hospital-based, cross-sectional study design . In Department of General Medicine at Sri Aurobindo Medical College and PG Institute, Indore. The total duration of the present study was 18 months. after the **Ethical Clearance.**
- **Aim of the study-** demographic profile, alcohol intake patterns, and clinical spectrum of ALD in patients at a tertiary care hospital in India among study participants. Measurements included physical examination findings, laboratory values (SGPT, SGOT, serum proteins, ammonia levels), and ultrasound results, documented at the time of enrollment. And correlating alcohol intake parameters (type, frequency, and duration) with the severity of liver disease.
- **Independent Variables**: Alcohol intake history (type, amount, frequency, duration), demographic factors (age, gender,).

Wine	**100 mL**	**1 unit (10 gram)**
Beer	250 mL	1 unit (10 gram)
Country liquor	30 mL	1 unit (10 gram)
Whiskey	40 mL	1 unit (10 gram)

- **Definition of the Exposure**: In this study, the exposure was defined as the participants' alcohol consumption history, including type, amount, frequency, and duration of alcohol intake. This was documented based on self-reported data collected through structured interviews during the data collection phase. parameters: type of alcohol, duration of drinking (in years), amount of alcohol consumed (units per month), Each type of alcoholic beverage was converted into a standard unit measure, with one unit of alcohol defined as approximately 10 grams of pure alcohol. This corresponds to around 30 mL of spirits (with a concentration of 40% alcohol by volume),

➢ Light Consumption: < 360 units per month.
➢ Moderate Consumption: 360-719 units per month.
➢ Heavy Consumption: ≥ 720 units per month.

These categories were determined by aggregating the daily units consumed and adjusting for the frequency of drinking days each month.

- **Inclusion Criteria:**

i. Patients aged 18 years and above.
ii. Patients with a confirmed diagnosis of alcoholic liver disease.
iii. Patients willing to provide written informed consent.

- **Exclusion Criteria:**

i. Patients diagnosed with other hepatic diseases, including infectious, autoimmune, malignant, post-hepatic, and congenital liver disorders.
ii. Patients under 18 years of age.
iii. Patients on hepatotoxic medications.

- **Sample Size:**, a total of 100 participants were enrolled in the present study. After **Obtaining Informed Consent.**

- **Data Collection Procedure:** After obtaining written informed consent, each participant underwent a structured interview to document their demographic details, alcohol consumption history. Subsequently, a physical examination was performed, and laboratory tests (SGPT, SGOT, CBC, serum protein, and ammonia levels) were conducted to assess liver function. Ultrasound imaging was utilized to evaluate liver morphology and confirm the diagnosis of alcoholic liver disease. The outcomes were recorded in the data collection form immediately after each assessment.

- **Statistical Analysis:** The data from paper-based data collection was initially entered into MS Excel and was imported into Stata 17.0. Descriptive statistics, such as mean, standard deviation, and frequency distribution, were used to summarize demographic and clinical data. Inferential statistics, including t-tests and chi-square tests, were applied to assess associations between alcohol intake variables and clinical outcomes. Statistical significance was determined with a p-value threshold of <0.05.

- **Funding:** There was no external funding for this study. Participants were not paid any compensation to participate in this study.

- **Conflict of Interest:** The authors declare no conflict of interest in the design, implementation, and interpretation of the findings of this study.

Results

The participants' ages ranged from 29 to 68 years, with a mean age of 48 years. The study population was predominantly male, with 92% being male and 8% female. Table 1 illustrates the sociodemographic characteristics of the participants.

Table 1: Characteristics of Participants

Variable	Category	n	%
Age	<35 years	15	15
	40-50 years	45	45
	>50 years	30	30
Gender	Male	92	92

	Female	8	8
Education	Primary or Illiterate	50	50
	Higher Secondary	30	30
	Senior Secondary or Above	20	20

Table 2: Clinical and Laboratory Parameters

Category	**n**	**%**
Clinical category		
Jaundice	65	65
Ascites	30	30
Splenomegaly	40	40
Investigation		
SGOT/SGPT Ratio >2	70	70
Hypoalbuminemia	65	65
Hyperbilirubinemia	55	55
Hyperammonemia	20	20
Mean Hemoglobin (g/dL)	10.5	1.2
Mean SGPT (U/L)	85	15
Mean Serum Albumin (g/dL)	50	50
Thrombocytopenia	40	40
USG finding		
Ascitis	30	30
Coarse Echotexture	70	70

The most common symptoms of presenting included jaundice (65%), and loss of appetite (62%). Ascites was observed in 30% of participants, particularly in those with decompensated cirrhosis. Hepatic encephalopathy was noted in 15% of cases, primarily among those with advanced disease. while splenomegaly was observed in 40% of cases. The mean SGPT level among participants was elevated, with an average value of 85 U/L, indicating hepatocellular injury. Higher SGPT levels were observed in participants with moderate to heavy alcohol consumption. The mean SGOT level was 112 U/L, with a notable SGOT/SGPT ratio >2 in cases of advanced liver disease, particularly in those with cirrhosis. Hypoalbuminemia (serum albumin <3.5 g/dL) was prevalent, with an average serum albumin level of 2.8 g/dL among participants. Mean total bilirubin levels were elevated at 3.2 mg/dL,

with higher values observed in participants presenting with jaundice and decompensated disease. Hyperammonemia was recorded in 20% of participants, correlating with instances of hepatic encephalopathy. Low levels of serum protein were recorded, with mean total protein levels of 5.9 g/dL, indicating compromised liver function. Anemia was common, with 65% of participants showing hemoglobin levels below 12 g/dL. The average hemoglobin level was 10.5 g/dL, with the severity of anemia more pronounced in participants with heavy alcohol consumption. Thrombocytopenia was observed in 40% of cases, with a mean platelet count of 125,000/mm³, particularly in those with portal hypertension and splenomegaly.

Ultrasound imaging revealed changes consistent with chronic liver disease in 70% of participants, including coarse echotexture and nodularity, particularly in those with cirrhosis. Signs of portal hypertension (e.g., splenomegaly, portal vein dilation) were evident in 45% of participants.

Table 3: Child-Pugh Score and MELD-Score

Score Type	Number of Participants (n)	Percentage (%)
Child-Pugh		
Child-Pugh Class A (Mild)	20	**20**
Child-Pugh Class B (Moderate)	45	**45**
Child-Pugh Class C (Severe)	35	**35**
MELD- Score		
MELD- < 15	25	**25**
MELD- 15-20	40	**40**
MELD-> 20	35	**35**

The results from Table 3 indicate the severity of liver disease among participants based on the Child-Pugh and MELD- scores. For the Child-Pugh classification, 20% of participants had mild liver disease (Class A), 45% had moderate liver disease (Class B), and 35% had severe liver disease (Class C). Regarding the MELD- scores, 25% of participants scored below 15, indicating milder disease, 40% scored between 15 and 20, representing moderate severity, and 35% scored above 20, reflecting severe liver disease.

Based on the intensity of alcohol consumption, participants were divided into three categories:
- Light Consumption: 27 % participants
- Moderate Consumption: 44 % participants
- Heavy Consumption: 29 % participants

Table 4: Association between alcohol intake and Liver disease

Alcohol Intake Category	Cirrhosis (%)	Decompensated Cirrhosis (%)	Non-Cirrhotic Alcoholic Liver Disease (%)
Light Consumption (<360 units/month)	10 (37%)	2 (7%)	17 (63%)
Moderate Consumption (360-719 units/month)	35 (80%)	15 (34%)	9 (20%)
Heavy Consumption (≥720 units/month)	25 (86%)	11 (38%)	4 (14%)
Total	**70 (70%)**	**28 (28%)**	**30 (30%)**
Pearson chi2 = 20.63 P-value < 0.001			

Cirrhosis was observed in 70 participants (70%), among these, 28 patients (40% of those with cirrhosis) had decompensated cirrhosis, characterized by complications such as ascites, hepatic encephalopathy, and variceal bleeding. The remaining 30 participants were diagnosed with alcoholic liver disease but had not yet progressed to cirrhosis. This group primarily exhibited hepatic steatosis (fatty liver) and alcoholic hepatitis

Among participants with light alcohol consumption (<360 units/month), 37% had cirrhosis, 7% had decompensated cirrhosis, and 63% had non-cirrhotic ALD. For those with moderate consumption (360-719 units/month), 80% had cirrhosis, 34% had decompensated cirrhosis, and 20% had non-cirrhotic ALD. Participants with heavy consumption (≥720 units/month) showed the highest prevalence of severe disease, with 86% having cirrhosis, 38% having decompensated cirrhosis, and only 14% presenting with non-cirrhotic ALD. These findings underscore the dose-dependent impact of alcohol intake on disease progression ($p < 0.001$).

Table 5: Association of Duration of Alcohol consumption and Type of ALD

Duration of Alcohol Consumption (years)	Number of Participants	Cirrhosis (%)	Decompensated Cirrhosis (%)	Non-Cirrhotic Alcoholic Liver Disease (%)
0-5	**12**	**3 (25%)**	**1 (8%)**	**9 (75%)**
6-10	**20**	**10 (50%)**	**3 (15%)**	**10 (50%)**
11-15	**22**	**15 (68%)**	**5 (23%)**	**7 (32%)**
16-20	**28**	**22 (79%)**	**8 (29%)**	**6 (21%)**
>20	**18**	**16 (89%)**	**11 (61%)**	**2 (11%)**
Total	**100**	**70 (70%)**	**28 (28%)**	**30 (30%)**
Pearson chi2 = 14.48; P-value = 0.008				

The results from Table 5 illustrate the association between the duration of alcohol consumption and the severity of alcoholic liver disease (ALD). Among participants with

alcohol use of 0–5 years, 25% had cirrhosis, 8% had decompensated cirrhosis, and 75% had non-cirrhotic ALD. For 6–10 years of consumption, 50% had cirrhosis, 15% had decompensated cirrhosis, and 50% had non-cirrhotic ALD. In those with 11–15 years of consumption, 68% had cirrhosis, 23% had decompensated cirrhosis, and 32% had non-cirrhotic ALD. For 16–20 years of drinking, 79% had cirrhosis, 29% had decompensated cirrhosis, and 21% had non-cirrhotic ALD. In participants consuming alcohol for more than 20 years, 89% had cirrhosis, 61% had decompensated cirrhosis, and only 11% had non-cirrhotic ALD. These results highlight a significant correlation between longer alcohol consumption and more severe liver disease (p = 0.008).

Table 6: Association of Type of Alcohol consumed and Type of ALD

Type of Alcohol Consumed	**Number of Participants**	**Cirrhosis (%)**	**Decompensated Cirrhosis (%)**	**Non-Cirrhotic Alcoholic Liver Disease (%)**
Country-made Alcohol (64%)	64	50 (78%)	20 (31%)	14 (22%)
Beer (17%)	17	8 (47%)	3 (18%)	9 (53%)
Hard Liquor (19%)	19	12 (63%)	5 (26%)	7 (37%)
Total	**100**	**70 (70%)**	**28 (28%)**	**30 (30%)**
Pearson chi2 = 17.44; P-value = 0.004				

The results from Table 6 demonstrate a significant association between the type of alcohol consumed and the severity of alcoholic liver disease (ALD). Among participants consuming country-made alcohol, 78% had cirrhosis, 31% had decompensated cirrhosis, and 22% had non-cirrhotic ALD. Those consuming beer had a lower prevalence of severe disease, with 47% having cirrhosis, 18% having decompensated cirrhosis, and 53% presenting with non-cirrhotic ALD. Participants consuming hard liquor showed intermediate severity, with 63% having cirrhosis, 26% having decompensated cirrhosis, and 37% presenting with non-cirrhotic ALD. These findings indicate that country-made alcohol is associated with the most severe liver damage, likely due to its impurities and higher ethanol content (p = 0.004).

Discussion:

This study provides a comprehensive analysis of the demographic, clinical, and alcohol consumption patterns among patients with alcoholic liver disease (ALD) in a tertiary care setting.

In our studyThe participants' ages ranged from 29 to 68 years, with a mean age of 48 years. The study population was predominantly male, with 92% being male and 8% female. Similar study by P.thivakar et al 2019 ,with male preponderance.

common symptoms of presenting included jaundice (65%), and loss of appetite (62%). Ascites was observed in 30% of participants, particularly in those with decompensated cirrhosis. Hepatic encephalopathy was noted in 15% of cases.The mean SGOT level was 112 U/L, with a notable SGOT/SGPT ratio >2 in cases of advanced liver disease, particularly in those with cirrhosis. Hypoalbuminemia (serum albumin <3.5 g/dL) was prevalent, with an average serum albumin level of 2.8 g/dL among participants. (50%)

Study done by tilottama parade et al 2022 .alcohol consumption effects on liver disease sample size 83 ,show jaundice in 90% ,loss of appetite (85%) ,haematemesis in 27% ,hepatic encephalopathy in 7% .mean .
A dose-dependent relationship between alcohol consumption and the severity of alcoholic liver disease (ALD), Heavy drinkers (≥720 units/month) demonstrated the highest prevalence of cirrhosis and decompensated cirrhosis]. For instance, long-term alcohol consumption exceeding 80 grams per day significantly increases the risk of advanced liver damage, including fibrosis and cirrhosis, compared to lower levels of intake[12]. A study by Simpson et al. (2019) analyzed alcohol consumption patterns in the UK and found that individuals consuming >80 grams of alcohol per day had a markedly higher risk of advanced liver disease (80%) [11]. This parallels the high prevalence of cirrhosis (86%) observed in heavy drinkers in our study.
Åberg et al. (2020), which noted that even moderate intake, when sustained over time, poses a substantial risk for progression to advanced liver disease .[13].
Roerecke M et al., (2020) conducted a meta-analysis and reported that drinking ≥5 drinks per day was associated with a substantially increased risk in both women (Relative Risk = 12.44) and men (RR = 3.80)[15].
Moon SY et al., (2023) reported that drinking more than (92 ± 26.4 g/week) increases the risk of developing liver-related diseases by 74%[18].
We observed a significant statistical association between longer durations of drinking correlate with more advanced liver disease, such as cirrhosis and decompensated [19,20]. Chronic alcohol consumption over many years exacerbates liver injury.
In the present study shorter durations of alcohol use (e.g., 0–5 years) are predominantly associated with non-cirrhotic ALD, such as fatty liver and alcoholic hepatitis 75 % This is consistent with findings from Kondili et al. (2005), which showed that earlier stages of ALD in 70% , including steatosis, occur with recent drinkers (< 5 years) [14].

Åberg et al. (2019) emphasized that > 10 years exposure to alcohol exacerbates the progression to cirrhosis [13]. The type of alcoholic beverage consumed and the severity of ALD, revealing significant differences in disease progression based on the type of alcohol. individuals consuming country-made alcohol exhibit the highest rates of cirrhosis and decompensated cirrhosis, compared to those consuming beer or hard liquor. This aligns with evidence suggesting that both the type and quality of alcohol influence the progression of liver disease[21,22]. The high prevalence of advanced liver disease among consumers of country-made alcohol (78% with cirrhosis, 31% with decompensated cirrhosis) is likely attributable to its unregulated production, which may result in higher levels of impurities, toxic byproducts, or higher ethanol content.

Studies have shown that spirits and country-made alcohol are more commonly associated with binge drinking patterns,]. In contrast, beer consumption is often associated with lower cumulative ethanol intake, potentially explaining the lower rates of cirrhosis (47%) and decompensated cirrhosis (18%). The impact of different types of alcohol may also be linked to their biochemical effects[12,23]. Hard liquor and spirits tend to deliver ethanol more rapidly, potentially overwhelming hepatic metabolism and leading to more significant hepatocyte injury.

This is consistent with findings from Kondili et al. (2005), where higher alcohol concentrations correlated with advanced liver damage[14]. The consumption of country-made alcohol may also be linked to lower socioeconomic status, as highlighted in studies, where financial limitations and limited access to regulated alcohol increased reliance on cheaper, unregulated beverages.

Conclusion:

This study provides critical insights into the demographic, clinical, and alcohol consumption patterns associated with alcoholic liver disease (ALD). It highlights the dose-dependent relationship between alcohol intake and liver disease severity, the significant role of prolonged alcohol use in the progression to advanced disease, and the differential impact of alcohol types, particularly country-made alcohol, on liver health. These findings underscore the multifactorial nature of ALD and the need for targeted prevention strategies, including public awareness campaigns, regulation of alcohol quality, and early interventions to reduce alcohol consumption. Addressing socioeconomic and lifestyle factors will be essential to mitigate the growing burden of ALD and improve clinical outcomes for affected individuals.

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