

**A study of Clinical profile of Alcoholic liver disease**Smita S Mansingh<sup>1</sup>, Yogesh Kumar Chahar<sup>2\*</sup>, Rahul Prem Mr<sup>3</sup><sup>1</sup>Assistant Professor, Department of Medicine, Military Hospital, Agra, Uttar Pradesh, India<sup>2</sup>Assistant Professor, Department of Medicine, World College of Medical Science & Research, Jhajjar, Haryana, India<sup>3</sup>Senior Resident, Department of Medicine, Military Hospital, Agra, Uttar Pradesh, India

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**Abstract**

**Background:** Alcoholic liver disease (ALD) is the one of leading causes of non-communicable diseases. The morbidity and mortality associated with ALD is immense in present context. However, ALD develops with myriad of complex and interlinked etiologies. Its clinical presentation and prognosis depends on amount and duration of alcohol, age of drinking etc. on the background of genetic predisposition. **Material and methods:** Study was conducted on 50 patients with diagnosis of Alcoholic liver disease at Military Hospital, Agra. **Result:** There were 46 males and 4 females. Average age of study group was 38.6 years, indicating trend towards younger onset of ALD. There were 22% of Fatty liver, 34% of Hepatitis and 44% of Cirrhosis patients. Most frequent presentation was nausea and vomiting. Higher % of patients were consuming alcohol at night, majority of them consumed > 3 – 6 times / week, majority of them started consuming at young age of 20s and symptoms of alcoholic liver disease started at 20-40 years. Majority of them consumed alcohol in groups. Prognostic scoring in cirrhotic patients was: DF Score – 37.15, MELD score – 19.27, Child pugh score – 11.65. Pearson correlation for DF score MELD score, CP score and was 0.76, 0.67, 0.82 respectively. **Conclusion :** Duration of alcohol and quantity of alcohol results in early onset ALD. GI symptoms predominate presentation of ALD. Ascites was the commonest complication. CP scoring was best most realistic prognostic scoring. Mortality was 20%.

**Keywords:** Alcoholic liver disease, Prognostic scores, Alcoholic hepatitis, fatty liver.

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**Introduction**

Alcohol is an organic substance, which has pharmacological effects on central nervous system causing euphoria, tolerance and addiction. Alcohol is a major threat to public health in both developed and developing countries. However, in a country like India where poverty is pervasive and nutritional status is already compromised, effect of alcohol on general health is more deleterious.

World Health Organization (WHO), report quotes that among the people addicted, the total per capita consumption of alcohol by individuals above 16 years of age is 6-7 L of pure alcohol per year, which equals 13.5 g of pure alcohol per day. However, there is a variable prevalence in different regions of the world. Nearly 5.1% of the global burden of disease is attributable to alcohol consumption, and it causes nearly 3.3 million deaths every year[1].

Over the last decade, alcohol consumption of alcohol has increased both in quantity and frequency world over. India is no exception to this. This has led to alcohol abuse becoming a worldwide social and medical problem. With the rapid economic and socio cultural changes the alcohol is being viewed as a symbol of social stature. Problem is compounded by

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the youth getting influenced by their role models. This has led to age at which people start alcohol getting declined[2]. An alcoholic is a person who progressively consumes an amount of ethanol capable of producing pathological changes and exhibit a cumulative pattern of social behavior associated with drinking including physical injuries, problem with family, job, and accidents while driving[3]. The liver digests small amount of alcohol consumption. If the consumption of alcohol increases – in frequency and quantity, it leads to pathological metabolic dysfunctioning of the liver. Initially, this involves fat accumulation in the hepatocytes, leading to fatty liver or steatosis. With continuous consumption of alcohol it will progress to alcoholic hepatitis. With further continued alcohol consumption, the alcoholic liver disease progresses to severe damage to hepatocytes causing cirrhosis. At this stage, progressive hepatic fibrosis and nodules ensues[4].

#### Pathophysiology

Normally alcohol dehydrogenase converts that alcohol into the acetaldehyde. Aldehyde dehydrogenase further converts acetaldehyde into the substance of acetate. Alcohol metabolism increases the production of NADH by reduction of NAD in the body. This change of metabolic balance toward the production of NADH leads to the formation of glycerol phosphate[5]. This combines with the fatty acids deposited in hepatocytes and becomes triglycerides and gets deposited in the liver. When lipid lipolysis stops due to consumption of alcohol, fatty acids accumulate in the liver cells leading to fatty liver disease. Further alcohol consumption brings down the immune system. The complex interaction leads to alcoholic hepatitis takes . Continuous liver injury causes irreversible liver damage, the cirrhosis of the liver.

#### Aims & objectives

- To study the clinical features of alcoholic liver disease
- To assess the prognostic scores of patients with Alcoholic liver disease

#### Material and methods

**Study area:** Out patient Department, Department of General Medicine, Military Hospital, Agra.

**Study population:** All persons with alcoholic liver disease

#### Inclusion criteria

- Patients in age group within 18 – 70 years
- Patients with clinical or investigational evidence of liver dysfunction with chronic significant alcohol intake

#### Exclusion criteria

- Patients with clinical or investigational evidence of liver dysfunction without chronic significant alcohol intake
- Patients admitted in Intensive care unit
- Patients who have not consented
- Patients suffering from Psychosis

**Sample size: 50**

#### Study design

A retrospective observational study

#### Methodology

After recruitment, detailed history was taken followed by clinical examination. Relevant laboratory reports were collected. Patients suffering from alcohol-related liver disease attending the outpatient liver clinic or admitted to hospital were collected prospectively till death or last follow-up and analyzed for delineation of factors affecting the outcome. Patients with clinical and/or investigational evidence of liver dysfunction and significant alcohol intake (i.e., daily intake of more than 45 grams for a prolonged period) were included. Active alcoholism was defined as per DSM-V criteria and its frequency, amount, and type were elicited. Alcoholic hepatitis was defined as recent onset of jaundice with SGOT/SGPT >1.5–2 without clinical/ultrasonological evidence of cirrhosis or liver decompensation with recent history of heavy consumption of alcohol. Cirrhosis was diagnosed based on clinical (presence of jaundice, ascites, encephalopathy, variceal bleed, splenomegaly or history of previous such complaints), biochemical, radiological, endoscopic criteria considered together or if documented on previous liver biopsy.

Definition of organ failure : AKI - Creatinine >1.5 mg/dL in the absence of other causes of kidney disease), Hepatic encephalopathy: As per West Haven criteria in the absence of other acute neurological disease. Severity of liver disease was calculated through prognostic scoring : Maddrey's discriminant function, Child-Turcotte-Pugh (CTP), Model for End-Stage Liver Disease (MELD)[6].

**Result****Table 1: Characteristics of Alcohol consumption pattern**

Characteristics	Sub categories	No of Subjects (%)
Time of alcohol consumption	Night	62%
	Both	38%
Frequency of alcohol consumption	1-2/W	28%
	3-6/W	40%
	Daily	32%
Age at start of alcohol consumption	< 20 yrs	60%
	20-30 yrs	16%
	> 30 yrs	24%
Age at start of alcohol liver disease	20-40 yrs	66%
	40-60 yrs	20%
	60-80 yrs	14%
Occasion of drinking pattern	Single	14%
	Group	58%
	Both	28%
Cause of Alcoholism	Environmental	12%
	Social	52%
	Psychosocial	36%

**Table 2: Symptoms of alcoholic liver diseases**

Symptoms	% of Patients
Jaundice	66 %
Nausea and vomiting	72%
Abdominal pain or discomfort	56 %
Loss of appetite	62 %
Weakness	80 %
Weight loss	52 %
Fever	21 %
Altered mental status	14 %
Alteration of the sleep-wake cycle	26 %
Mood swings	18 %
Fainting	12 %

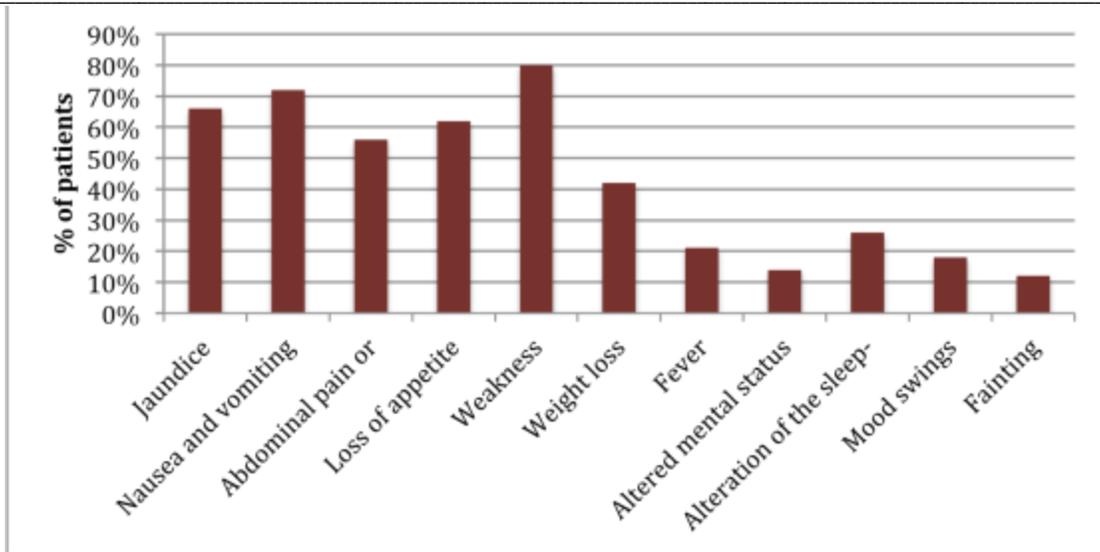


Fig 1:Symptoms of alcoholic liver diseases

Table 3: Classification of alcoholic liver diseases

Classification	% of patients
Fatty liver	22%
Hepatitis	34%
Cirrhosis	44%

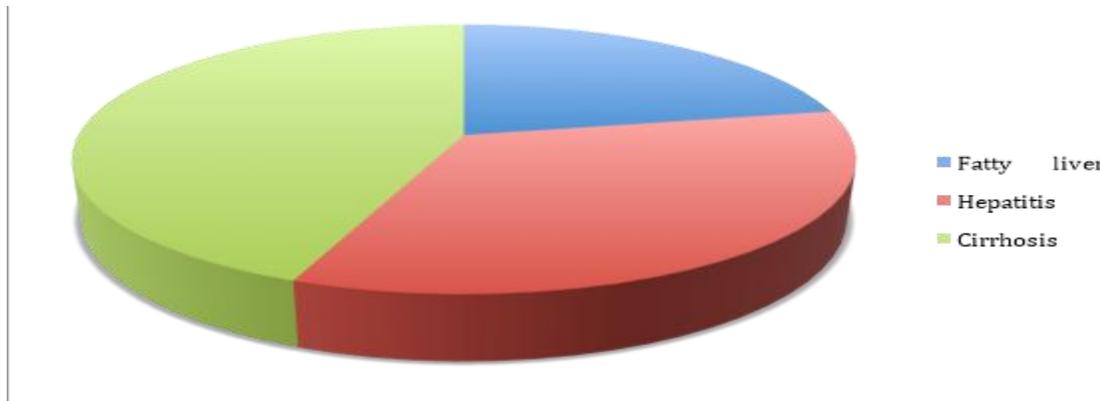
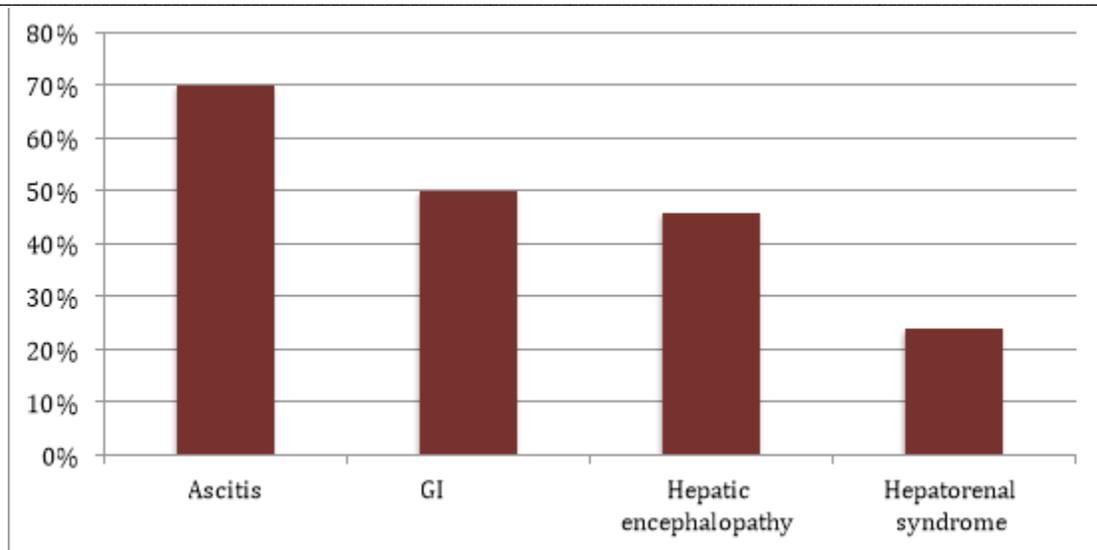


Fig 2:Classification of alcoholic liver disease

Table 4:Comparison of complications of Alcoholic beverages

Complications	% of Patients
Ascitis	70 %
GI bleed	50 %
Hepatic encephalopathy	46 %
Hepatorenal syndrome	24 %



**Fig 3: Complications of ALD**

**Table 5: Prognostic scoring (n=22)**

Prognostic scores	Scoring
DF Score	37.15
MELD score	19.27
Child pugh score	11.65
Mortality rate	22%

## Discussion

In our study, there were 46 males and 4 females. Average age of study group was 38.6 years, indicating trend towards younger onset of ALD. We had 22% of Fatty liver, 34% of Hepatitis and 44% of Cirrhosis patients.

We observed that higher % of patients were consuming alcohol at night, majority of them consumed > 3 – 6 times / week, majority of them started consuming at young age of 20s and symptoms of alcoholic liver disease started at 20-40 years. Majority of them consumed alcohol in groups. Similar observations were made by Nitya et al, where average duration of alcohol intake was 17 years, which was not significantly different among various liquor groups [7]. Narawane et al observed that average duration of drinking >14 years were significantly more common in those with liver disease. In both studies mean age of ALD was 41-45 years highlighting early onset ALD similar to our observations[8]. Major complaints

observed in our study were nausea and vomiting, abdominal pain or discomfort, loss of appetite, weakness and weightloss > 50% of patients. Similar observations were made by Sachdeva et al and Nayaka et al where nausea and vomiting predominated the presentation of ALD. (9,10). In contrast, various western literature points toward weight loss as predominant symptom in ALD[4]. Difference can be reasoned by the probable genetic disposition of Asian population. Among complications, we noted ascites followed by GI bleed and Hepatic encephalopathy. Hepatorenal syndrome was least represented. Nitya et al observed complications were ascites (72%), hepatorenal syndrome (35%), hepatic encephalopathy (59%) and gastrointestinal bleeding (59%). In a study by Mitra et al, the most complications observed among the patients were ascites (53.7%), followed by hepatic encephalopathy (20.7%), upper gastrointestinal bleed (18.86%), sub-acute bacterial peritonitis (3.7%) and

hepatorenal syndrome (2.8%) [11]. Prognostic scoring in cirrhotic patients was: DF Score – 37.15, MELD score – 19.27, Child pugh score – 11.65. Mortality rate was 22%. Nagamani et al made similar observation, where prognostic outcome measures correlated well with age at which patients started drinking alcohol. Mortality rate was 18.6 % in their study, indicating worse the prognostic score, worse is the mortality. Pearson correlation in our study, for DF score MELD score, CP score and was 0.76, 0.67, 0.82 respectively emphasizing better prognostication with CP scoring system in our study. Similar observations were made by Nagamani et al

### Conclusion

Duration of alcohol and quantity of alcohol results in early onset ALD. GI symptoms predominate presentation of ALD. Ascites was the commonest complication. CP scoring was best most realistic prognostic scoring. Mortality was 20%.

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