

# CLINICAL PROFILE OF CASES OF ALCOHOLIC LIVER DISEASE

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## ABSTRACT

**Background:** Alcohol is most common substance abused. Alcoholic liver disease is a major health care problem in India. Alcohol consumption is directly associated with liver disease mortality and accounts for increased social and economic costs. Alcoholic liver disease may take the forms of acute involvement (alcoholic hepatitis) or chronic liver disease (steatosis, steatohepatitis, fibrosis and cirrhosis). The severity and prognosis of alcohol-induced liver disease depends on the amount, pattern and duration of alcohol consumption, as well as on the presence of liver inflammation, diet, nutritional status and genetic predisposition of an individual. While steatosis is complete benign disease, liver cirrhosis is associated with marked morbidity, mortality and life expectancy shortening.

**Aims & Objective:** To study alcoholic liver disease, clinical presentation, to assess the severity of alcoholic liver disease and its complications and their treatment response and overall outcome among patients using laboratory and radiological parameters.

**Material and Methods:** A total of 50 patients were studied and their clinical profile, laboratory parameters and radiological investigations were taken.

**Results:** Among 50 patients 58 % belonged to age group 40-49 years. All were male. 78 % of patients from urban areas, with 60 % belonging to middle class. 60 % of patients have chief complaint of abdominal distension and melena each. Jaundice (60%) and ascites (60%) were commonest finding. All patients had raised SGPT, SGOT, S.ALPO4 and S. bilirubin suggesting liver damage. Prolonged PT and reduced S. albumin suggested reduced protein synthesis because of liver disease. Alcoholic hepatitis was in 24% cases, while 40% had fatty liver and 36 % had alcoholic cirrhosis. Overall mortality rate was 20 %. , most common cause is encephalopathy (40%), coagulopathy leading to DIC (40%) and hepatorenal syndrome (20%).

**Conclusion:** Alcoholic liver disease was seen among the productive age group with high morbidity and mortality. Mortality and morbidity associated with this disease is matter of serious economic loss to the nation and grief for the society

**KEY-WORDS:** Alcohol; Alcoholic Liver Disease; Clinical Profile

## Introduction

Mankind is using alcohol since ages for central nervous system stimulation, aphrodisiac, etc. Even in Hindu mythology, it has been referred as "Somaras". Alcohol itself acts as a behavioural stimulant at lower blood levels but with higher blood levels it acts as a central nervous system depressant. Alcohol affects all systems ranging from central nervous system, cardio-vascular system and genitourinary system. The World Health Organization estimates that there are 140 million people with alcoholism worldwide. Alcoholism is called a "dual disease" since it includes both mental and physical components. The biological mechanisms that cause alcoholism are not well understood. Social environment, stress, mental health, family history, age, ethnic

group and male, all influence the risk for condition.<sup>[1]</sup>

Alcohol is consumed at some time in their life by 80% of the population.<sup>[2]</sup> Alcohol is associated with high morbidity and mortality: 3.7% of the global deaths and 4.4% of the global DALYs lost in the year 2002 could be attributed to this exposure.<sup>[3]</sup>

Alcoholic liver disease encompasses a spectrum of injury, ranging from simple steatosis to frank cirrhosis.<sup>[4,5]</sup> It may well represent the oldest form of liver injury known to mankind. Evidence suggests that fermented beverages existed at least early as the Neolithic period (cir. 10,000 BC).<sup>[6]</sup> Chronic and excessive alcohol ingestion is one of the major causes of liver disease in western world.<sup>[7,8]</sup> Alcohol remains most common cause of

liver disease in India. Alcoholic liver disease encompasses a clinical histological spectrum, including fatty liver, alcoholic hepatitis and alcoholic cirrhosis. Fatty liver is a benign condition but progression to alcoholic hepatitis and cirrhosis is life threatening. Alcoholic hepatitis is diagnosed predominantly on clinical history, physical examination and laboratory findings.

The possible factors that can affect the development of liver injury include the dose, duration and type of alcohol consumption, drinking patterns, gender, ethnicity, and associated risk factors, including obesity, iron overload, nutritional deficiency esp. protein, pregnancy, concomitant infection with viral hepatitis and genetic factors.

In our present study, we have focused on alcoholic liver disease and its various complications using laboratory and radiological investigations.

## Materials and Methods

The clinical survey of alcoholic liver disease was carried out and total one hundred patients with diagnosis of alcoholic liver disease are included in study. Patient's details including occupation, socio economic status, risk factors, clinical features, complications, laboratory and radiological investigations were carried out.

Patients with a history of significant chronic alcohol intake with physical signs of liver disease (jaundice, portal hypertension, complications of portal hypertension) and positive laboratory and radiological finding are included in study.

Patients with viral hepatitis, hepatitis B, hepatitis C, post-necrotic cirrhosis, patients with documented seropositivity for HIV, Patients with any other form of chronic liver disease, Wilson's disease, Hemochromatosis etc.; and patients with other co-morbid illness such as cardiac, respiratory and renal illness are excluded.

A case of Alcoholic liver disease is diagnosed in patients with history of significant alcohol intake, physical signs of liver disease, and supporting laboratory investigations.<sup>[9]</sup> Alcoholic cirrhosis is

diagnosed in a patients with H/o alcohol consumption > 80 g/dl in men and 40 g/dl in female and at least one clinical sign of hepatocellular failure and one of the sign of portal hypertension along with at least three ultrasound finding of cirrhosis of liver.<sup>[7]</sup>

Detailed history was taken and physical examinations were done. Patient is examined for signs of portal hypertension (ascites, splenomegaly, abdominal wall collaterals and a venous hum) and hepatic injury (cutaneous telangiectasia, palmer erythema, finger clubbing, Dupuytren's contracture and peripheral neuropathy) and feminization (gynaecomastia and hypogonadism). All laboratory investigations including a liver chemistry profile (S. albumin, Bilirubin and transaminases, AST/ALT. complete blood count and prothrombin time) are done. Ultra sonogram, barium swallow and upper G.I. endoscopy were done.

## Results

In 5<sup>th</sup> decade of life, incidence of alcoholic liver disease was 58 % in our study. Unfortunately, our study also showed 36 % of incidence in 4<sup>th</sup> decade, this reflects the trend towards alcoholic consumption at a younger age under influence of changing socioeconomic scenario. The mean age of the patients was 41years, the youngest patient being 33 years old and oldest patient being 53 years old. All patients were male. 78 % patients were from urban areas. 60% belonged to middle class, while 24% patients were belonged to lower class and 16 % belonged to higher class. The mean duration of alcohol consumption was 16.25 years, with a minimum of 6 years and a maximum of 33 years. This confirms the impact of duration of alcoholic consumption in alcoholic liver disease. Most of the patients consumed alcohol on a daily basis and most of them consumed country liquor.

30 (60 %) of patients presented with chief complain of abdominal distension, jaundice and melena each. While anorexia 20 (40%), encephalopathy 17 (34%) were other complaints.

Jaundice 30(60%) and ascites 30 (60%) were commonest finding on general examination. Hepatomegaly was found in 50% of patients while

60% of patients had ascites and splenomegaly each on per abdominal examination. Other findings were anaemia (30%), dilated veins over abdomen (16%), pedal edema (16%), and spider naevi (4%).

**Table-1: Clinical Presentation of Alcoholic Liver Disease**

Presentation	No. of Patients (%)
Ascites	30 (60%)
Melena	30 (60%)
Jaundice	30 (60%)
Hepatomegaly	25 (50%)
Anorexia	20 (40%)
Hepatic Encephalopathy	17 (34%)
Hematemesis	17 (34%)
Weight loss	15 (30%)
Fever	15 (30%)
Oliguria	12 (24%)
Pedal oedema	8 (16%)
Spider naevi	2 (4%)

The mean haemoglobin was 10.1 gm%. 30 % patients have haemoglobin less than 8 gm%, correlating with history of hematemesis (34%). The mean total leukocyte count (TLC in mm<sup>3</sup>), platelet count ( $\times 10^3/\mu\text{L}$ ), and MCV were 9521,148, 97.6, respectively. The mean SGOT (AST), SGPT (ALT) and AST: ALT RATIO was 134.6 IU/L, 56.1 IU/L, 2.11 respectively. The mean alkaline phosphatase was 208 IU/L. The mean S. total bilirubin was  $3.17 \pm 1.81$ . S. bilirubin is more than 5mg./dl in 36 % of patients. The mean S. albumin was 2.41 g/dl. Hypoalbuminemia (S. albumin < 3.5 gm/dl) was found in 80% of patients, an indicator of advanced liver disease. The mean prolongation of prothrombin time was 5.6 S 72 % of patients had prothrombin time prolonged by more than 6 sec indicating liver damage altering coagulation profile. The mean International normalized ratio (INR) was 1.92. International normalized ratio (INR) was 1-1.5 in 38%, >1.5-2 in 22%, >2-2.5 in 10%, >2.5-3 in 2%, >3-3.5 in 9% and >3.5 in 11% patients. The mean serum sodium was 132.1 mEq/L. The mean serum creatinine was 1.74 mg%. S. creatinine >1.5 mg/dl was found in 32 % of patients out of which 8 % of patients had hepatorenal syndrome. Hypernatremia (S. sodium <130 meq/dL) found in 31 % of patients, while hypokalaemia was found in 22% of patients.

USG abdomen showed shrunken liver with coarse echo texture in 36% patients which is suggestive

of cirrhosis, while increased echo texture was seen in 40 % patients which is s/o fatty liver and enlarged liver in 24 % of patients s/o hepatitis.

Excluding 5 critically ill patients, 40 patients were subjected to Ba. Swallow of which 48 patients showed presence of oesophageal varices. All patients were subjected to upper GI scopy and 60 % patients showed varices. 9 patients have grade I varices, while 11 and 10 patients have grade II and grade III varices respectively. 20% patients had gastritis; rest 20 % had normal upper GI scopy findings. Final diagnosis was alcoholic hepatitis in 24% cases while 40% had fatty liver and 36 % cases had alcoholic hepatitis.

Melena and portal hypertension were most common complications found in 60 % of patients. While other complications were hematemesis (34%), hepatic encephalopathy (34%), hepatorenal syndrome (8%), coagulopathy (8 %), hypovolemic shock (4%).

20 patients who presented with fatty liver, and symptoms like anorexia, weight loss were prescribed vitamins, especially the B complex, C and K were given. Oral thiamine supplement was prescribed on discharge. 12 patients who presented with alcoholic hepatitis and complain of jaundice, anorexia and fever were treated with intravenous glucose with vitamin B complex, intravenous vitamin K was given after prothrombin time investigated. All patients were advised to abstain from alcohol. 18 patients who presented with alcoholic cirrhosis were treated with Diuretic (potassium sparing and Loop),  $\beta$  blocker and nitrates; Vitamin K. Patients with hepatic encephalopathy were treated with Lactulose, Rifaximin, L-ornithine L-aspartate, Intravenous glucose, intravenous thiamine and vitamin K.

All the patients were followed up for 6 months. Out of 20 patients who had fatty liver, 12 patients continue to imbibe alcohol after discharge. 4 patients abstained from alcohol, while 4 were lost in 6 months follow up.

Out of 12 patients who had alcoholic hepatitis, in 6 month follow up; 6 presented with decompensated liver disease in form of ascites, 2

patients has no complain, while 4 were lost in 6 months follow up.

Out of 18 cirrhotic patients, 4 patients died of hepatic encephalopathy, while 4 patients died of hepatorenal syndrome. 2 patients who were died of coagulopathy leading to DIC. 5 patients had regular followed up with chronic encephalopathy and ascites. 3 patients were lost during 6 months follow up.

In present study 10 patients died during hospital stay. 4 patients died of hepatic encephalopathy. 2 patients died of coagulopathy leading to disseminated intra-vascular coagulation. Death due to hepatorenal syndrome occurred in 4 patients during 6 month follow up. Overall mortality rate was 20 % in our study.

## Discussion

In our study average age of the patients was 41 years. While in Chacko and Chacko et al study and Sarin et al study average age of patients were  $48 \pm 11$  years and  $43 \pm 8.7$  years respectively.<sup>[10,11]</sup> Comparatively lower age in our study is suggestive of growing problem of alcoholic liver disease. Susumu Itoh et al study & Michael J. Thun et al study have shown 21:1 and 17:8 male to female ratio respectively, while in our study all patients were male.<sup>[12,13]</sup> In India, because of cultural and traditional value, females are not indulging in alcoholism. The average patient with alcoholic liver disease loses 12 years of productive life, a much larger than that of heart disease (2 years) and cancer (4 years).<sup>[14]</sup>

In present study, 60 % patients were from middle class population, 16 % were higher class while the rest of 24 % belonged to the lower socio economic class. These is compatible with Sarin et al study were 70 % were middle class, 10 % higher class and 20 % lower socio economic status.<sup>[11]</sup>

Jaundice was found in 60 % of patients which is comparable with Pathak et al in which it is 57.5%.<sup>[15]</sup> Ascites was observed in 60 % of patients in our study, while in Mendenhall et al study it is 50.9 %.<sup>[16]</sup> Hepatic encephalopathy was found in 34 % of patients which is comparable with Antonio Chedid et al study (32%).<sup>[17]</sup> In our

study 60 % of patients had splenomegaly which is comparable with Sarin et al study (55%).<sup>[11]</sup> Hepatomegaly was found in 50 % of cases which is comparable with Pathak et al (48.6%).<sup>[15]</sup>

The mean haemoglobin level in our study was 10.1 gm % which is comparable with Sarin et al which has 10.2 gm% of mean haemoglobin level.<sup>[11]</sup> The mean total leukocyte count was 9521 mm<sup>3</sup> which is comparable with Pathak et al in which it is 9303.89 mm<sup>3</sup>.<sup>[15]</sup> The mean value of MCV was 97.6 in our study which is comparable with Mendenhall et al (99.8-102.8) and pathak et al ( $96.42 \pm 9$ ).<sup>[15,16]</sup> The absolute value of SGOT and SGPT is usually <300 u/L in patients with alcoholic liver disease.<sup>[18]</sup> AST/ALT RATIO > 2 is suggestive of alcoholic hepatitis.<sup>[19]</sup> A high AST/ALT ratio suggests advanced alcoholic liver disease.<sup>[20]</sup> The mean SGPT level in present study was 56.1 IU/L which is comparable with Mendel hall et al (47-50 IU/L).<sup>[16]</sup> The mean SGOT level is 134.6 which is comparable with pathak et al ( $142.95 \pm 159.85$ ).<sup>[15]</sup> The AST: ALT ratio are 2.11 which are comparable with pathak et al ( $2.27 \pm 1.33$ ).<sup>[15]</sup> Average prolongation in PT in our study was 5.6 sec. which is comparable with pathak et al ( $6.29 \pm 6.16$ ).<sup>[15]</sup> Serum AlPO<sub>4</sub> in present study was 208 IU/L; while in Antonio Chedid study is 163-219 IU/L.<sup>[17]</sup> Average Serum bilirubin in present study was between  $3.17 \pm 1.81$  mg/dl, which is comparable with Chacko and Chacko et al (3.3-4.5).<sup>[10]</sup> Average prolongation of prothrombin time in present study was 4-5.9 Second while in Sarin et al study it was 2.0-7.9 S.<sup>[11]</sup> Average serum albumin in present study was 2.4 gm/dl. While in Chacko and Chacko et al study it was 2.5 gm/dl and Mendelhall et al it was 2.3-3.7 gm/dl which is comparable with our study.<sup>[10,16]</sup> Thus the result of our study establishes most of the known facts about alcoholic liver disease in Indian population.

## Conclusion

58% and 36% of patients belonged to age group 40-49 years and 30-39 years respectively. In our study, 20 % patients died and hepatic encephalopathy (40%) was most common cause of death. Total 94 % patients belonged to age group 30-49 years, young and middle age

population, which is active and productive mass of society.

High morbidity of alcoholic liver disease required frequent hospitalization adding to burden for health care system and loss of man-hours at work. Mortality and morbidity associated with this disease is matter of serious economic loss to the nation and grief for the society. We recommended screening for alcohol abuse in all adult patients presenting to the hospital as early detection of alcoholic liver disease can decrease both morbidity and mortality due to alcoholic liver disease.

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