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Original Research Article

A STUDY OF CLINICAL PROFILE AND PROGNOSTIC IMPLICATION IN PATIENTS WITH ALCOHOLIC LIVER DISEASE

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ABSTRACT

Background: Consumption of alcohol is directly associated with the morbidity and mortality of liver. The study was conducted to know the various clinical features, laboratory parameters, radiological investigations and to access complications & prognosis via Child Pugh score in patients of Alcoholic. **Material and Methods:** A total of 120 cases of Alcoholic liver disease for this study were obtained from wards and ICU of Medicine and gastroenterology department in Geetanjali Medical College & Hospital, Udaipur, and Rajasthan over a period of 12 months from January 2018 to December 2018. The patients were studied their clinical profile, laboratory parameters and radiological investigations and to correlate development of complications and prognosis via Child Pugh score. **Results:** Out of the 120 individuals, 51.6% had severe hypoalbuminemia; the percentage of patients with elevated AST, ALT, and Total bilirubin and INR were 68.3%, 30.8%, 59.2% and 64.17% respectively. 61.7% had ratio of AST to ALT more than 1 with 30% having more than 2. 51.7% had hyponatremia. 53.3 % of patients had chief complaint of abdominal distension. Splenomegaly was found in 68.3%. Upper GI bleeding (55%) and ascites (53.3%) were commonest complications. USG revealed cirrhosis in 71.7% Pts. 55% oesophageal varices were found in upper GI endoscopic investigation. The P value for association of Child Pugh category with duration & quantity of alcohol consumption was 0.01. **Conclusion:** Amount and duration of alcohol consumption is directly related with severity of liver disease which can be judged by hepatic enzyme elevation, prolonged PT/INR, hypoalbuminemia, ultrasonographic findings, oesophageal varices on UGI endoscopy and child pugh category.

Keywords: Liver Cirrhosis, Child Pugh score, hypoalbuminemia, Splenomegaly, Upper GI bleeding.

INTRODUCTION

Alcohol use is increasing rapidly in developing regions and could be a major concern among autochthonous folks round the world, showing a higher prevalence of the disease.**(1)** The global burden of disease project estimated alcohol to be responsible for 1.5% of all deaths and 3.5% of those who live life with a disability.**(2)** Alcoholism is a condition ensuing from excess drinking of beverages that contain alcohol. The main health risk of alcoholism includes liver disease, heart disease, pancreatitis, central nervous system disorders, and certain forms of cancer.**(3)** Alcohol consumption causes fatty liver, alcoholic hepatitis and alcoholic cirrhosis.**(1,4,5)** In Western countries, alcohol is the major cause of liver cirrhosis, and it is gradually increasing in countries like Japan and India.**(1,6)** According to WHO, 1/3rd of India's population consumes alcohol on daily basis and 11% of Indians

are either moderate drinkers or heavy drinkers.**(7)** Alcoholic liver disease (ALD), includes fatty liver, hepatitis and cirrhosis.(1) Fatty liver is the most common form of ALD, which develops in more than 90% of heavy drinkers. but, only about 10%-20% of heavy drinkers (consuming > 60 gm/day) develop a more severe form of ALD, such as hepatitis and cirrhosis.**(1)** Cirrhosis is the final result of chronic liver damage, which is characterized by parenchymal injury leading to extensive fibrosis and nodular regeneration.**(8,9)** The leading cause of liver cirrhosis in India is excess alcohol consumption. The alcoholic liver cirrhosis is more common in males compare to females.**(10)**

Clinical features of cirrhosis include jaundice, spider angioma, nodular liver, splenomegaly, caput medusa, cruveilhier baumgarten syndrome, palmar erythema, white nails, Hypertrophic osteoarthropathy/ Finger clubbing, dupuytren's contracture, hypogonadism, anorexia, fatigue, weight loss, muscle wasting.**(11)**

Complications of cirrhosis in ALD patients include ascites, encephalopathy, upper GI bleed, renal failure (hepatorenal syndrome) spontaneous bacterial peritonitis and HCC (hepatocellular carcinoma).**(12)**

Since there are very few studies on Indian population on ALD (alcoholic liver disease) regarding amount, duration, type of alcohol consumption and their effect on biochemical parameters and morbidity especially in western part of Rajasthan, we conducted this study to evaluate the demographic, clinical, haematological, biochemical parameters and prognosis in patients with alcoholic liver disease.

MATERIALS AND METHODS

The study was a hospital based prospective study, carried out on 120 patients admitted in wards and ICU of Medicine and gastroenterology department in Geetanjali Medical College & Hospital, Udaipur Rajasthan. The study was carried out for a period of 12 months from January2018 to December 2018.

Adult patients of both the sexes diagnosed with ALD based on a combination of features including History of significant alcohol intake with clinical evidence of liver disease based on sign & symptoms and Supporting laboratory and radiological abnormalities were included in this study.

Patients with viral hepatitis (hepatitis B, hepatitis C), post-necrotic cirrhosis, patients with any other form of chronic liver disease such as Wilson's disease, hemochromatosis NASH etc. ware excluded from the study.

Procedure

A detailed demographic, alcoholism history and clinical history was taken from each patient and detailed medical examination was conducted. All laboratory and radiological investigations were done including LFT(S. albumin, S. bilirubin direct /indirect and transaminases AST/ALT, prothrombin time). complete blood count , serum electrolytes, serum ammonia, ultrasonography, UGI endoscopy ,ascitic fluid biochemical and cytological study and recorded on a specially designed proforma.

Alcoholism history included three measures – type, duration (years), amount (grams per day). The duration was defined as short when it was up to 10 years, moderate when it was 11-20 years and long if it was more than 20 years. Based upon these clinical and biochemical data, Child-Pugh score is calculated in each participant which is used as prognostic marker in this study.

Table:-1 Child-Turcotte-Pugh Grading of Severity of Liver Disease(13)

The study was under taken after obtaining the Ethical clearance from ethical committee of the Geetanjali Medical College & Hospital, Udaipur, Rajasthan.

Parameters	Points		
	1	2	3
Encephalopathy (grade)	None	1 and 2	3 and 4
Ascites	Absent	Slight	Moderate
Serum bilirubin (mg/dL)	1 to 2	2 to 3	>3
Serum albumin (g/dL)	>3.5	2.8 to 3.5	<2.8
Prothrombin time (seconds prolonged)	1 to 4	4 to 6	>6
Total score	CTP class		
5 to 6	A		
7 to 9	B		
10 to 15	C		

Statistical Analysis:

The collected data were analysed and statistically evaluated using SPSS-PC-21 version. Quantitative data was expressed in mean, standard deviation and difference between two comparable groups were tested by student's t-test (unpaired) or Mann Whitney 'U' test. Qualitative data were expressed in percentage. Statistical differences between the proportions were tested by chi square test or Fisher's exact test. 'P' value less than 0.05 was considered statistically significant.

RESULTS

The study included 120 alcoholic liver disease patients of which 116 patients were male (96.7%) and 4 (3.3%) were female with a mean age of 45.58±11.61 years. Majority of the patients 61 patients(50.8%) consumed hard liquor, 24(20.0%) hard liquor and beer both, 10(7.5%) beer, 4(3.3%) wine, 10(8.3%) beer and wine both, 12(10.0%) consumed all type of liquor with mean quantity of alcohol intake was 162.69±72.80 g/day and mean duration of alcohol consumption was 18.88±8.76 years.

The most common presenting symptom in ALD patients were abdominal distension (53.3%), yellow discoloration of sclera (48.3%), hematemesis (35.8%), malena (31.7%), nausea/vomiting (27.5%), sleeping disturbance (15.8%) altered sensorium (15.0%), SOB (15%) and abdominal pain (11.7%). The commonest signs were splenomegaly in 82(68.3%), ascites in 64 (53.3%), icterus in 58 (48.3%), flapping tremors in 37(30.8%) patients and hepatomegaly was present in 28 (23.3%) patients.

The most complications observed in ALD patients were upper GI bleeding in 65(55.0%) patients, ascites in 64 (53.3%), coagulopathy in 61 (50.8%), hepatic encephalopathy in 50 (41.7%), renal failure in 21 (17.5%) and spontaneous bacterial peritonitis in 9 (7.5%) and 1 patient(0.83%) had HCC.

Anaemia was common findings in 87.9% with mean Hb of 9.60±2.47 gm/dl, leukocytosis was seen in 30% of patients, thrombocytopenia was seen in 58.33%, Liver function tests showed mean AST, ALT, S. albumin, S. globulin, A:G Ratio, total Bilirubin, INR were 155.79±304.77 U/L, 87.46±251.04 U/L, 2.69±0.80 g/dl, 4.86±8.96 g/dl, 0.77±0.91, 6.06±4.97 mg/dl and 2.336±1.44 respectively, hyponatremia was seen in 51.7% patients.

Fig: 1. Profile of Complications observed in ALD patients

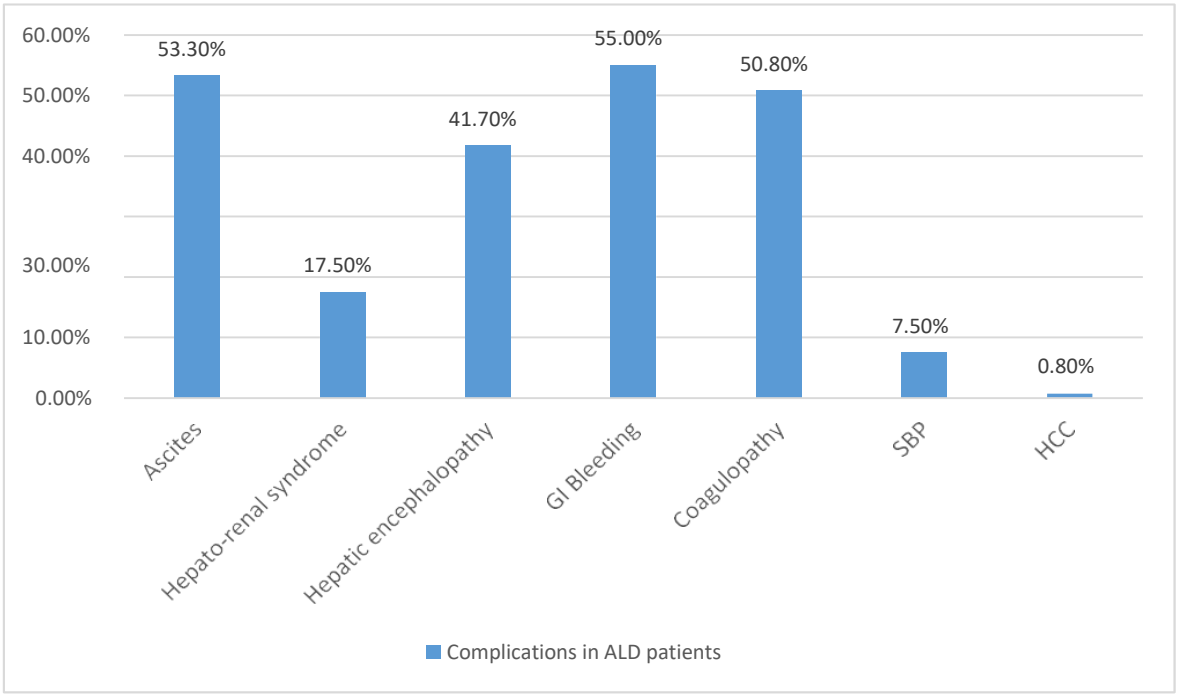

Table 2: Ultrasonography findings in ALD patients (n=120)

USG findings	No.	%
Cirrhosis (normal or shrunken liver)	86	71.7
Fatty liver	6	5
Hepatomegaly + cirrhosis	21	17.5
Hepatomegaly with hepatitis	7	5.8

Table 3: Upper GI endoscopic findings in ALD patients (n=120)

Upper GI endoscopic findings	No.	%
Normal findings	36	30
Gastritis	20	8.3
Oesophageal varices (n= 66) 55%		
Grade I	38	31.7
Grade II	20	16.7
Grade III	8	6.7
Grade II with gastritis	10	8.3

Table 4: ALD spectrum in studied patients (n=120)

ALD spectrum	No.	%
Cirrhosis	107	89.2
Fatty liver	6	5
Hepatitis with hepatomegaly	7	5.8

Table 5: Association of Child Pugh category with duration and quantity of alcohol consumption

	Category A (n=25)	Category B (n=36)	Category C (n=59)	P value
Duration of alcohol consumption (years)	13.26±6.34	17.80±9.01	22.09±8.59	<0.01
Quantity of alcohol consumption (gm/day)	145.37±45.57	158.59±91.65	174.40±54.21	<0.01

Table 5 shows longer the duration and larger the quantity of alcohol consumption develop more child pugh category indicating severe liver damage.

DISCUSSION

The present study was a hospital based prospective study conducted in Department of General Medicine and Gastroenterology of Geetanjali Medical College & Hospital, Udaipur, Rajasthan, enrolling a total of 120 alcoholic liver disease subjects. The study was conducted to assess the demo graphic profile, clinical profile, laboratory parameters and prognostic implications in patients with alcoholic liver disease.

In our study of 120 ALD patients, mean age was 45.58±11.61 years. Similar to our study, the mean age was 48 ± 11 years in a study by Chacko RT et al**(14)**, 43±8.7 years by Sarin SK et al**(15)** and 46.2±9.86 years by Nand N et al**(11)**.

In our study, 96.7% patients were males while only 3.3% were females. Itoh S et al **(16)** & Thun MJ et al **(17)** in their study have shown 95.4% males and 4.6% females; 68% males and 32% females respectively. In India, because of cultural, social, religious and traditional value, females are usually not indulging in alcoholism.

In our study, 50.8% of patients were consuming hard liquor, 20.0% consuming both hard liquor and beer 7.5% consuming beer, 3.3%, wine, 8.3% both beer and wine while 10.0% of patients consumed all type of liquor. While Nand N et al**(11)**, reported 79% of patients were consuming country made spirit, whisky by 6.5%, variable drinking by 14% and 0.5% consumed only beer. Nagmani R et al **(18)** also reported 80 % of patients were consuming country made spirit, 11% whisky and 9% beer.

In our study mean alcohol consumption was 162.69±72.80g/day. While Walter A et al **(19)** in his study observed 60 gm of alcohol/ day by 40% patients, 61-70 gm/day by 16% and 81-90 gm/day by 28% patients.

In our study of alcoholic liver disease patients, mean duration of alcohol consumption was 18.88±8.76 years (ranging from 4 to 42 years). Similar to our observation mean duration of alcohol consumption in development of ALD was 17 years (ranging from 8 to 36 years) by Nand N et al**(11)** and 16.25 years (ranging from 6 to 33 years) by Suthar H et al**(20)**.

In present study, most common presenting symptoms were abdominal distension (53.3%) yellow discoloration of sclera (48.3%), melena (33.3%), nausea/vomiting (27.5%), hematemesis (25%), sleeping disturbance (15.8%) altered sensorium (15.0%), shortness of breath (15%) and abdominal pain (11.7%). In a study by Suthar H et al **(20)**, 60% of patients presented with abdominal

distension, jaundice and melena each while 40% had anorexia and 34% had encephalopathy. In a study by Nand N et al18, abdominal pain (55%), distension (78%) and jaundice (60%) were the most common symptoms.

In our study, splenomegaly (68.3%), ascites (53.33%), icterus (48.3%) and flapping tremors (30.8%) were commonest signs observed. Hepatomegaly was found in 23.3% of patients. Most of the physical examination finding in our study showed similar results as seen in the pioneer study of Mendenhall CL (jaundice in 60%, ascites in 57%).**(21)** Two remarkable differences are higher incidence of splenomegaly and a lower incidence of hepatomegaly in our study as compared to that of Mendenhall C et al (splenomegaly in 26%, and hepatomegaly in 87%).**(22)** This difference was probably because the study group of Mendenhall predominantly contained alcoholic hepatitis patients while our study consists of whole spectrum of alcoholic liver disease predominantly cirrhotics. In a study by Suthar H et al**(20)**, Jaundice was found in 60% of patients, hepatomegaly in 50%, splenomegaly in 60% and ascites in 60% of patients. In study by Pathak OK et al**(23)** jaundice was found in 57.5% of patients, hepatomegaly in 48.6%, ascites in 45.3% and oedema in 36.5% patients.

In our study, the mean haemoglobin, total leukocyte count and platelet counts were 9.60±2.47g/dL, 9277.75±3983.76/mm^3 and 145933.64±100691.6/mm^3 respectively, while in study by Awasthi VR et al(24), the mean haemoglobin, total leukocyte count and platelet counts, were 10.215 ± 3.339 g/dL, 10063 ± 5432.7 mm3, 140627 ± 89899 mm3 respectively.

In our study, 98 males (84.48%) and all 4 females (100%) were anaemic and mean Hb was 9.60±2.47 g/dL. Similar to our study, Awasthi VR et al**(24)** observed anaemia in 75% patients and Nagamani R et al**(18)** observed anaemia in 82% patients. We observed leucocytosis in 30% patients, while Awasthi VR et al **(24)** observed leucocytosis in 62% patients. In our study thrombocytopenia was observed in 58.33% patients, while Awasthi VR et al**(24)** also observed thrombocytopenia in 36% patients.

In our study, hyponatremia was seen in 51.7% of patients while in contrast Awasthi VR et al**(24)** reported hyponatremia in 23% patients Suthar H et al**(20)** in 31% of patients.

In our study, raised bilirubin (> 3gm/dl) was seen in 54.2 % patients and the mean total bilirubin was 6.06±4.97 mg/dl in all studied patients. Similarly, Awasthi VR et al(24) also observed raised bilirubin in 50% of patients and mean bilirubin 6.152 ± 6.35 mg/dL.

SGOT and SGPT are intracellular enzymes which are released on cell injury. Thus these are indicators of liver cell injury even in cases of alcohol mediated liver cell injury. It is observed that these transaminases can increase upto a level of 300 IU/L but levels beyond this are usually indicative of other causes of liver cell injury such as viral hepatitis. In cases of alcohol induced liver damage it is observed that the ratio of SGOT to SGPT is usually more than 1 and if it is more than 2, 90% of the cases are due to alcohol. In our study mean SGOT was 155.79±304.77 IU/L, SGPT was87.46±251.04 IU/L and SGOT: SGPT ratio was 1.45 ± 1.29 while this ratio was >2 in 30% of patients. SGOT was raised in 68.3% and SGPT was raised in 30.8 % patients in our study. In a study by Awasthi VR et al(24), the mean SGOT was 140.84 ±119.66 IU/L, SGPT was 69.645 ± 60.03 IU/L, and SGOT:SGPT ratio was 2.11 while Walter A et al(19) showed raised SGOT in 42% patients, SGPT in 8% patients and SGOT: SGPT ratio was > 2 in 40% patients.

In our study hypoalbuminemia (<3.5gm/dl) was seen in 99.2% and severe hypoalbuminemia(<3gm/dl) in 51.6% of patients with a mean albumin of 2.69±0.80 g/dl (ranging from 1 to 5.5 gm/dl). This was similar to study by Awasthi VR et al(24), in which hypoalbuminemia was seen in 57% of the patients and the mean albumin was 2.82 ± 0.5 gm/dL.

In our study mean A:G Ratio was 0.77±0.91 while in a study by Awasthi VR et al(24) the mean A: G ratio was 0.93 ± 0.64.

INR reflects synthetic function of the liver, particularly clotting factors. Prolonged INR indicates hepatic cell failure or decrease in number of hepatic cells so much so that clotting factors synthesis is hampered.

In our study INR was increased in 64.17% of patients and mean INR was 2.336±1.44. In a study by Awasthi VR et al**(24)**, INR was increased in 68% of patients and mean INR was 1.65 ± 0.69 .

In our study, complications seen due to chronic alcoholic liver disease were upper GI bleeding in 55.0%, ascites in 53.3%, coagulopathy in 50.8%, hepatic encephalopathy in 41.7%, renal failure in 17.5%, spontaneous bacterial peritonitis in 7.5% and

HCC (hepatocellular carcinoma) in 0.83% of patients. Similar to our study Nagmani R et al**(18)** observed ascites in 40%, hepatic encephalopathy in 22%, upper GI bleeding in 18%, renal failure in 15% and spontaneous bacterial peritonitis in 5%.

In our study, on ultrasonography we found cirrhosis in 71.7%, hepatomegaly with hepatitis in 5.8%, hepatomegaly with cirrhosis in 17.5% and fatty liver in 5% of patients.(Table no. 2) Nagmani R et al**(18)** observed, hepatomegaly in 54% cirrhosis in 18%, acute pancreatitis in 38% and chronic pancreatitis in 27% patients of ALD, none of our patients had pancreatitis on USG.

In our study UGI endoscopy was normal in 30% patients while varices grade I, II and III seen in 31.7%, 16.7% and 6.7% of patients respectively. Pathak Ok et al**(23)** also observed normal UGI endoscopy in 42% of patients and varices of Grade I, II and III in 33%, 17% and 8% of the patients respectively.

In our study longer the duration of alcohol and larger the amount of alcohol consumption lead to hepatic damage and ultimately ends with cirrhosis of liver. Jepsen p et al in his study showed the amount and duration of alcohol consumed are closely associated with cirrhosis which is similar to our study.**(25)**

Quantity and duration of alcohol intake are considered the most important risk factors involved in the development of ALD. In our study amount and duration of alcohol intake was significantly directly proportional to child pugh score which indicates more severe will be the liver disease on consumption of large amount of alcohol for a longer time. Nand N et al**(11)** in a study correlated child pugh score well with amount and duration of alcohol consumption similar to our study.

CONCLUSION

Our study concludes that there is a significant correlation in development of alcoholic liver disease with type, amount and duration of alcohol consumed. Alcohol consumption leads to hepatic cell damage and ends with cirrhosis and its complications. Amount and duration of alcohol consumption is directly related with severity of liver disease which can be judged by hepatic enzyme elevation, prolonged PT/INR, hypoalbuminemia, ultrasonographic findings ,oesophageal varices on UGI endoscopy and child pugh category. Other factors like social status, poverty, crowding, nutrition, genetic factors, associated other hepatic infections and simultaneous other addiction may also contribute in addition to alcohol in development of alcoholic liver disease and needs further studies.

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