ORIGINAL RESEARCH PAPER

General Medicine

A STUDY OF CLINICAL PROFILE OF ALCOHOLIC LIVER DISEASES IN 50 PATIENTS AT TERIARY CARE CENTER, BELAGAVI

KEY WORDS: Alcohol; Alcoholic Liver Disease; Clinical Profile

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ABSTRACT

Introduction: Alcoholic liver cirrhosis is a major cause of morbidity and mortality all over the world. Alcohol is most common substance abused. South Asian populations are prone to develop ALD, and majority of population consuming country made alcoholic beverages. 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 access 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 54 % belonged to age group 40-49 years. All were male. 60 % of patients from urban areas, with 62 % belonging to middle class.60 % of patients have chief complaint of abdominal distension and jaundice and malena 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 in22% cases, while 36% had fatty liver and 42 % had alcoholic cirrhosis. Overall mortality rate was 24 %. , most common cause is encephalopathy (41.66%), hepatorenal syndrome (33.33%) and cougulopathy leading to DIC(25%).

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

INTRODUCTION:

Alcoholic consumption is a major risk factor for development of chronic diseases worldwide, and remains 3rd major cause for chronic liver disease [1]. The spectrum of liver injury varies from simple steatosis to cirrhosis, which is linked to amount of alcohol consumption. Chronic liver disease is the end result of cirrhosis characterized by nodular regeneration and extensive fibrosis. It is estimated that about 30% of heavy alcohol consumers develop cirrhosis, other factors contributed to development of alcoholic cirrhosis include sex, obesity, duration of alcohol intake, non sex linked genetic factors and cigarette smoking [2]. Other causes of cirrhosis of liver include chronic viral hepatitis, autoimmune hepatitis, bilary cirrhosis, non-alcoholic steatohepatitis, Inherent metabolic disorders such as Wilson's disease, 1 anti-trypsin deficiency and cystic fibrosis [3].

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 [4].

Complications of cirrhosis in ALD patients include development of ascites- portal hypertension, encephalopathy, Upeer GI bleed, Renal failure and spontaneous bacterial peritonitis. Abstinence from alcohol is the cornerstone of the therapy along with good nutrition and medical supervision [3]. Various studies have shown relation with drinking pattern including amount and duration, however there are uncertain results about the type of alcohol intake and development of cirrhosis [5].

Alcohol is consumed at some time in their life by 80% of the population[7]. 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[6,8].

Alcoholic liver disease encompasses a spectrum of injury, ranging from simple steatosis to frank cirrhosis[9,10]. 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)[11]. Chronic and excessive alcohol ingestion is one of the major causes of liver disease in western world[12,13]. 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[14]. 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[15].

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

MATERIALS AND METHODS:

A study of clinical profile of alcoholic liver disease was carried out on total Fifty patients with diagnosis of alcoholic liver disease presented to dept.of General Medicine from jan 2018 to dec 2018. Patient's details including occupation, socio economic status, risk factors, clinical features, complications, laboratory and radiological investigations were carried out. Informed written consent and institutional ethical clearance taken.

INCLUSION CRITERIA:

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.

EXCLUSION CRITERIA:

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. 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.

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 (gynaecomestia 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 and upper G.I. endoscopy were done.

RESULTS:

In 5th decade of life, incidence of alcoholic liver disease was 54 % in our study. Unfortunately, our study also showed 40 % of incidence in 4th 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 42years, the youngest patient being 31 years old and oldest patient being 56 years old. All patients were male. 60 % patients were from urban areas and 40%from rural areas. 62% belonged to middle class, while 22% patients were belonged to lower class and 16 % belonged to higher class. The mean duration of alcohol consumption was 15.25 years, with a minimum of 5 years and a maximum of 32 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 and jaundice .While melena25 (50%), anorexia 22 (44%), encephalopathy 19 (38%), haematemesis17(34%),pedal edema12(24%) 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 (32%), dilated veins over abdomen (15%), pedal edema (24%), and spider naevi (6%).

The mean haemoglobin was 10.2 gm%. 32% patients have haemoglobin less than 9 gm%, correlating with history of hematemesis (34%). The mean total leukocyte count (TLC in mm3), platelet count (×10 3 /μL), and MCV were 9432,141, 97.3, 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 3.15 ± 1.71. S. bilirubin is more than 5mg./dl in 38 % of patients. The mean S. albumin was 2.48 g/dl. Hypoalbuminemia (S. albumin < 3.5 gm/dl) was found in 83% 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 1.91. International normalized ratio (INR) was 1-1.5 in 40%, >1.5-2 in 24%, >2-2.5 in 10%, >2.5-3 in 10%, >3-3.5 in 8% and >3.5 in 8%patientsThe mean serum sodium was 131.2 mEq/L. The mean serum creatinine was 1.72 mg%. S. creatinine >1.5 mg/dl was found in 34 % of patients out of which 8 % of patients had hepatorenal syndrome. Hyponatremia (S. sodium < 130 meq/dL) found in 33 % of patients, while hypokalaemia was found in 21% of patients. USG abdomen showed shrunken liver with coarse echo texture in 42% patients which is suggestive of cirrhosis, while increased echo texture was seen in 36 % patients which is s/o fatty liver and enlarged liver in 22 % of patients s/o hepatitis. All patients were subjected to upper GI scopy and 64 % patients showed varices. 10 patients have grade I varices, while 13 and 9 patients have grade II and grade III varices respectively. 18% patients had gastritis; rest 20 % had normal upper GI scopy findings. Final diagnosis was alcoholic cirrhosis in 42% cases while 36% had fatty liver and 22 % cases had alcoholic hepatitis. Portal hypertension were most common complications found in 60 % of patients. While other complications were melena(50%), hematemesis (34%), hepatic encephalopathy (38%), hepatorenal syndrome (8%), coagulopathy (10 %), hypovolemic shock (4%). 18 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. 11 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. 22 patients who presented with alcoholic cirrhosis were treated with Diuretic (potassium sparing and Loop), 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 18 patients who had fatty liver, 10 patients continue to imbibe alcohol after discharge. 4 patients abstained from alcohol, while 4 were lost in 6 months follow up. Out of 11 patients who had alcoholic hepatitis, in 6 month follow up; 5 presented with decompensated liver disease in form of ascites, 3 patients has no complain, while 3 were lost in 6 months follow up. Out of 22 cirrhotic patients, 5 patients died of hepatic encephalopathy , while 4 patients died of hepatorenal syndrome. 3 patients who were died of coagulopathy leading to DIC. 6 patients had regular followed up with chronic encephalopathy and ascites. 4 patients were lost during 6 months follow up.

In present study 10 patients died during hospital stay. 5 patients died of hepatic encephalopathy. 3 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 24 % in our study.

DISCUSSION :

In our study average age of the patients was 42 years. While in Chacko and Chacko et al study and Sarin et al study average age of patients were 48 ± 11 years and 43 ± 8.7 years respectively[15,16]. 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[17,18]. 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)[20].

In present study, 62 % patients were from middle class population, 16 % were higher class while the rest of 22 % 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[16]. Jaundice was found in 60 % of patients which is comparable with Pathak et al in which it is 57.5%[20]. Ascites was observed in 60 % of patients in our study, while in Mendenhall et al study it is 50.9 %[21]. Hepatic encephalopathy was found in38 % of patients which is comparable with Antio Chedid et al study (32%)[22]. In our study 60 % of patients had splenomegaly which is comparable with Sarin et al study (55%)[16]. Hepatomegaly was found in 50 % of cases which is comparable with Pathak et al (48.6%)[20]. The mean haemoglobin level in our study was 10.2 gm % which is comparable with Sarin et al which has 10.2 gm% of mean haemoglobin level[16]. The mean total leukocyte count was 9422 mm3 which is comparable with Pathak et al in which it is 9303.89 mm3.[20] The mean value of MCV was 97.3 in out study which is comparable with Mendenhall et al (99.8-102.8) and pathak et al (96.42 ± 9)[19,20,]. The absolute value of SGOT and SGPT is usually 2 is suggestive of alcoholic hepatitis[23]. A high AST/ALT ratio suggests advanced alcoholic liver disease[24]. The mean SGPT level in present study was 56.1 IU/L which is comparable with Mendel hall et al (47-50 IU/L)[21]. The mean SGOT level is 134.6 which is comparable with pathak et al (142.95 ± 159.85)[20]. The AST: ALT ratio are 2.11 which are comparable with pathak et al (2.27 ± 1.33)[20]. Average prolongation in PT in our study was 5.6 sec. which is comparable with pathak et al (6.29 ± 6.16). [15] Serum AlPO4 in present study was 208 IU/L; while in Antonio Chedid study is 163-219 IU/L[22]. Average Serum billirubin in present study was between 3.15 ± 1.71 mg/dl, which is comparable with Chacko and Chacko et al (3.3-4.5)[15]. 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[16]. Average serum albumin in present study was 2.48 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[15,21]. Thus the result of our study establishes most of the known facts about alcoholic liver disease in Indian population.

CONCLUSION:

54% and 40% of patients belonged to age group 40-49 years and 30-39 years respectively. In our study, 24 % patients died and hepatic encephalopathy (41.66%) was most common cause of death. Total 90 % 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.

REFERENCES:

1. Suk KT, Kim MY, Baik Sk. Alcoholic Liver Disease: Treatment; World J Gastroenterol. 2014;28-20(36):12934–12944.
2. Phukan JP, Sinha A, Deka JP. Serum lipid profile in alcoholic cirrhosis: A study in a teaching hospital of northeastern India; Niger Med J. 2013;54(1):5-9.
3. Bacon BR. Cirrhosis and its complications. In: Kasper et al., Harrison's principles of internal medicine, 19th edition. Part 14, chapter 365, page no 2058.
4. Shuppan D, Afdhal NH. Liver Cirrhosis. Lancet 2008(8); 371(9615):838–851.
5. Nand N, Malhotra P, Dhoot DK. Clinical profile of alcoholic liver disease in a tertiary care center and its correlation with type, amount and duration of alcohol consumption; Journal of the association of physicians of India. 2015; 63:14-20.
6. Black JM, Hawks JH. Medical-Surgical Nursing: Clinical Management for Positive Outcomes. 8th ed. St Louis, Mo: Saunders; 2005. (Vol. 1) p. 1336- 1352.
7. Schuckit MA. Alcohol and Alcoholism. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. (eds.). Harrison s Principles Of Internal Medicine 18 th Edi. New York: McGraw-Hill Professional. 2012. P. 3546-52.
8. World Health Organization. Evidence-based strategies and interventions to reduce alcoholrelated harm. Sixtieth world health assembly, Provisional agenda item 12.7. WHO. 2007. Available from: URL: http:// apps.who. int/gb/ebwha/pdf_files/WHA60/ A60_14-en.pdf
9. O'Shea RS, Dasarathy S, McCullough AJ & Practice Guideline Committee of the AASLD & Practice Parameters Committee of ACG. AASLD practice guidelines - Alcoholic Liver Disease. AASLD. 2010. Available from: URL: http://www.aasld.org/practiceguidelines/Docume nts/ Bookmarked % 20 Practice%20Guidelines/Alco holicLiverDisease1-2010.pdf
10. Bruha R, Dvorak K, Petrtyl J. Alcoholic liver disease. World Journal of Hepatology 2012;4(3):81-90.
11. Patrick CH. Alcohol, Culture, and Society. Durham, NC: Duke University Press; 1952.
12. Mailliard ME, Sorrell MF. Alcoholic liver Disease. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. (eds.). Harrison s Principles Of Internal Medicine 18 th Edi. New York: McGrawHill Professional. 2012. P. 2589-91.
13. Stewart S, Day C. Alcohol and the liver. In: Sherlock S, Dooley J (eds.). Diseases of the Liver and Biliary Sys¬tem. 12th ed. Oxford: Blackwell Publishing 2011. P. 507-520
14. McCullough AJ, O Connor JF. Alcoholic liver disease: Proposed recommendations for the American College of Gastroenterology. AM J Gastroenterol 1998;93:2022-36.
15. Chacko RT, Chacko A. Serum and muscle magnesium in Indians with cirrhosis of liver. Indian Journal of medical Research 1997;106:469- 474.
16. Sarin SK, Dhingra N, Bansal A, Malhotra S, Guptan RC. Dietary and nutritional abnormalities in alcoholic liver disease: a comparison with chronic alcoholics without liver disease. Am J Gastroenterol 1997;92(5):777-783.
17. Thun MJ, Peto R, Lopez AD, Monaco JH, Henley SJ, Heath CW, et al. Alcohol Consumption and Mortality among Middle-Aged and Elderly U.S. Adults. The New England Journal of Medicine 1997;337:1705-1714.
18. Itoh S, Youngel T, Kawagoe K. Comparison between nonalcoholic steatohepatitis and alcoholic hepatitis. Am J Gastroenterol 1987;82:650–654.
19. Friedman SL, McQuaid KR, Grendell JH (eds). Current Diagnosis and Treatment in Gastroenterology. 2nd edi. USA: McGraw-Hill. 2003.
20. Pathak OK, Paudel R, Panta OB, Giri BR, Adhikari B. Restospective study of clinical profile and prognostic indicators in patients of alcoholic liver disease admitted to a tertiary care teaching hospital in western Nepal. Saudi J Gastroenterology 2009;15(3):172-175.
21. Mendenhall CL, Anderson S, Weesner RE, Goldberg SJ, Crolic KA. Protein-calorie malnutrition associated with alcoholic hepatitis. Veterans Administration Cooperative Study Group on Alcoholic Hepatitis. Am J Med. 1984;76:211–222.
22. Chedid A, Mendenhall CL, Gartside P, French SW, Chen T, Rabin L. Prognostic factors in alcoholicliver disease. VA Cooperative Study Group. Am J Gastroenterol. 1991;86:210–216.
23. Fairbanks K D. Alcoholic liver disease. Availble from: URL: http://www.clevelandclinicmeded.com/medicalpu bs/ disease management/hepatology/alcoholic - liver-disease/
24. Cohen JA, Kaplan MM. The SGOT/SGPT ratio- an indicator of alcoholic liver disease. Dig. Dis. Sci. 1979;24:835-8. 20. Shen Z, Li YM, Yu CH, Shen Y, Xu L, Xu CF, et al. Risk factors for alcohol-related liver injury in the island population of China: A population-based casecontrol study. World J Gastroenterol. 2008;14(14):2255-61.