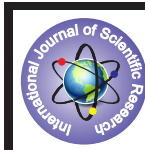


## Retrospective Study of the Clinical Profile and Prognostic Indicators in Patients of Alcoholic Liver Disease Admitted to a tertiary Care teaching Hospital



### Medical Science

**KEYWORDS :** Alcoholic liver disease, prognostic indicators

**Dr. Cecil Parmar**

Assistant Professor, Department of Medicine, B.J. Medical college Ahmedabad-16

**Dr. Monark Vyas**

Tutor, Dept of Community Medicine, B.J. Medical College, Ahmedabad-16

### ABSTRACT

*Background : Alcohol is the most common substance abused. Liver disease caused by alcohol abuse, including its end stage, cirrhosis, is a major health care problem, which is difficult to treat.*

*Objectives: To study the demographic profile, laboratory parameters, complications and their prognostic implications among patients of alcoholic liver disease (ALD).*

*Materials and Methods: Records of all patients of ALD admitted from January 1, 2011 to December 31, 2012 were studied and followed up to December 31, 2013. A total of 100 patients were analyzed. Their clinical profile and laboratory parameters were noted and analyzed.*

*Results: Among the 100 patients, 98% were male. The mean age of presentation was 48 years. Jaundice (60%) was the most common presentation followed by hepatomegaly (40 %). Hypoalbuminemia (70%) followed by ascites (30%) were common complications. Death occurred in 24 % of the patients, the most common cause being hepatic encephalopathy (70%) followed by variceal bleeding and hepatorenal syndrome. Patients had significantly increased mortality along with high aspartate aminotransferase, Alanine aminotransferase  $\geq 2$ , ultrasonography - proven cirrhosis, rise in prothrombin time  $\geq 6$  s, total bilirubin  $\geq 10$  mg/dL.*

*Conclusion: ALD was predominantly seen among the middle age group with a high morbidity and mortality. Jaundice, ascites, hepatic encephalopathy at presentation and , AST:ALT  $\geq 2$ , USG-proven cirrhosis, coagulopathy, hyperbilirubinemia are poor prognostic indicators.*

### Introduction

Alcohol is the most common substance abused.. Alcohol is associated with high morbidity and mortality: 3.7% of the global deaths. Chronic and excessive alcohol ingestion is one of the major causes of liver disease. Alcoholic liver disease (ALD) is the most common cause of cirrhosis in the Western world. Although some of the physical findings, especially of alcohol abuse and feminization, are more commonly observed in ALD patients than in other liver diseases, no single physical finding or constellation of findings is 100% specific or sensitive for ALD. Moreover, both laboratory abnormalities and physical findings may be minimal or absent even in patients with established ALD. Although liver biopsy is the gold standard in the diagnosis of ALD, it is rarely performed in clinical practice. This is because a combination of clinical and laboratory data can make an accurate diagnosis of ALD with the prebiopsy diagnosis of ALD being 98% specific and 79% sensitive. Moreover, coagulopathy in patients of ALD makes liver biopsy difficult and risky.

### Methods:

This retrospective hospital record-based study was conducted in a tertiary care teaching hospital. All ALD patients admitted to the hospital from January 1, 2011 to December 31, 2012 were studied and followed up to December 31, 2013. A total of 100 patients were included in the study. A pilot study was carried out with 5% of the patients and a proforma was designed. Case details with patients' particulars, occupation, risk factors, presenting features, laboratory values, complications, comorbidities and cause of death were entered in the predesigned proforma. A case of ALD is diagnosed in patients with a history of significant alcohol intake, physical signs of liver disease and supportive laboratory data. Ultrasonographic evidence of hepatomegaly and splenomegaly is based on the report of the radiologists. Complications, which were diagnosed clinically with supportive laboratory evidences, were noted till the end of the follow-up period.

A case of ALD who had visited the hospital for an entirely different problem was excluded from the study.

### Results:

A total of 100 patients were analyzed, of which 98% were male. The mean age of presentation in years is  $52.08 \pm 13.11$  and median age is 48 years. The mean duration of stay was  $13.41 \pm 12.57$  days. Jaundice was the most common presentation, which was present in 60% of the patients, followed by hepatomegaly 40% ascites 30% and peripheral edema (36.5%)

The mean hemoglobin, platelet counts ( $\times 103/\mu\text{L}$ ), MCV were  $10.6 \pm 3.45$  g/dL,  $176.8 \pm 89.23$ ,  $100.42 \pm 9.09$  fl respectively. The average value of aspartate aminotransferase (AST), alanine aminotransferase (ALT), AST:ALT, total bilirubin, albumin, A:G ratio and prothrombin time (PT) difference were  $142.95 \pm 158.85$  U/L,  $81.56 \pm 133.7$  U/L,  $2.27 \pm 1.33$ ,  $4.05 \pm 4.5$  mg/dL,  $3.22 \pm 0.86$  g/dL,  $0.97 \pm 0.51$  and  $6.29 \pm 6.16$  s, respectively. The mean Na<sup>+</sup>, K<sup>+</sup>, urea and creatinine were  $130.08 \pm 8.43$  meq/dL,  $3.8 \pm 1.04$  meq/dL,  $70.04 \pm 64.86$  mg/dL and  $1.8 \pm 1.2$  mg/dL, respectively. Hyponatremia ( $<130$  meq/dL), hypokalemia ( $<3.5$  meq/dL), raised urea ( $>40$  mg/dL) and creatinine ( $>1.5$  mg/dL) were present in 20 %, 30%, 52% and 42% of the patients, respectively.

Abdominal sonography was performed, 62% showed fatty change, 37 % had cirrhosis. Splenomegaly and ascites were present in 34% and 30 %, respectively. Upper gastrointestinal endoscopy was performed in 40 patients. Grade I, grade II and grade III varices were present in 33%, 17% and 8% of the patients, respectively. Hypoalbuminemia (serum albumin  $< 3.5$  g/dL) was the most common complication 70% which was closely followed by ascites 55% and portal hypertension 46%. Of the 100 patients 24 patients died during the study period and follow-up. Hepatic encephalopathy was the most common cause of death, accounting for 40 % of the deaths, followed by variceal bleeding 30 and hepatorenal syndrome 30 Sepsis, spontaneous bacterial peritonitis and aspiration pneumonia were less common causes of mortality. Among laboratory and sonographic parameters, variables significantly associated with mortality are PT difference between patient and control of  $\geq 5$  s ( $P < 0.001$ ), AST: ALT  $\geq 2:1$  ( $P = 0.003$ ), total bilirubin  $\geq 4$  mg/dL ( $P = 0.004$ ) and sonographic evidence of cirrhosis ( $P = 0.001$ ) during the study and follow-up period.

### Discussion:

We analyzed 100 patients of ALD, of which the majority (98%) were males. Our the mean age of presentation of 48 years shows a high prevalence of the disease among the middle age group, a result similar to previous studies. The mean number of hospital admission of 1.75 and the mean duration of hospital stay of 13.41 days indicate a significant burden of the disease. The average patient with ALD loses 12 years of productive life. The risk factors for ALD are duration and quantity of alcohol drinking, and nutrition (both malnutrition and obesity). However, they were poorly analyzed during clinical practice as shown in our study. This should be looked at with great concern to further strengthen our understanding of factors accelerating ALD. The

practice of consuming locally available/home-made alcohol of various concentrations in this part of the world has made it difficult to calculate the amount of alcohol consumed. AST/ALT ratio  $>2$  is suggestive of alcoholic hepatitis. Our study showed that AST/ALT  $\geq 2:1$  at presentation is significantly associated with increased mortality. Coagulopathy is common in patients of ALD. Prolonged PT is associated with increased mortality. Our study showed that the mean rise in PT was 6.29. Hyperbilirubinemia was more frequently associated with deaths. Our study also showed that total bilirubin  $>4$  mg/dL at presentation was significantly associated with increased mortality. Cirrhosis was a poor prognostic indicator in patients of ALD. In our study, sonography showed cirrhosis in 37% of the patients and ultrasonography-proven cirrhosis has significantly increased the mortality risk, with . The mean albumin was 3.2 g/dL and the mean A/G ratio was 0.97. Approximately 30% of the patients dying of end-stage liver disease experience significant encephalopathy, approaching coma. Our study had many limitations. The study was retrospective, so some of the information was missing from the records. Quantitative measurement of alcohol intake was not possible in history because of the wide variety of alcohol in use, including home-made alcoholic beverages that vary greatly in concentration. This hospital record-based study

was not able to include the visit of patients to other hospitals for the same disease. Complications and death of patients that occurred outside the study hospital were also not included. The results could have been improved by carrying out a prospective study. The significant association of clinical and laboratory poor prognostic indicators were only binomial and so a multinomial analysis will be required for the generalization of the results of the study.

### Conclusion

The result of this study establishes most of the known facts about ALD in the population of our part of world, reinforcing the clinical and laboratory prognostic indicators of ALD. ALD can have a constellation of clinical presentation and none of them is sensitive and specific for detection of ALD. Not only liver function tests, patients of ALD also have abnormal renal function tests and hematological abnormalities. Mostly affecting the productive age group of the male population, ALD has a high economic burden to the society as well. We recommend screening for alcohol abuse in all adult patients presenting to the hospital as early detection of ALD can decrease both morbidity and mortality due to ALD.

## REFERENCE

1. Dennis L. Kasper, Eugene Braunwald, Anthony S. Fauci, Stephen L. Hauser, Dan L. Longo, J. Larry Jameson. Harrison's Principles Of Internal Medicine 16th Edition. | 2. WHO Sixtieth world health assembly, Provisional agenda item 12.7. April 5 2007. Evidence-based strategies and interventions to reduce alcohol-related harm. | 3. Rehn, N, Room, R, Edwards, G. Alcohol in the European Region – consumption, harm and policies. Copenhagen: World Health Organization Regional Office for Europe; 2001. Available from: <http://mednet3.who.int/prioritymeds/report/FinalRep/Alcohol>. | 4. McCullough AJ, O'Connor JF. Alcoholic liver disease: Proposed recommendations for the American College of Gastroenterology. *AM J Gastroenterol* 1998;93:2022-36. | 5. Ryback RS, Eckardt MJ, Felscher B, Rawlings RR. Biochemical and hematological correlates of alcoholism and liver disease. *JAMA* 1982;248:2261-5. Sanchez GC, Baunsgaard P, Lundborg CJ. A comparison between clinical diagnosis and histopathological findings in liver biopsies. *Scand J Gastroenterol* 1980;15:985-91. | 6. Talley NJ, Roth A, Woods J, Hench V. Diagnostic value of liver biopsy in alcoholic liver disease. *J Clin Gastroenterol* 1988;10:647-50. | 7. Friedman SL, McQuaid KR, Grendell JH. Current Diagnosis and Treatment in Gastroenterology. 2nd edition. | 8. Bell H, Jahnsen J, Kittang E, Raknerud N, Sandvik L. Long-term prognosis of patients with alcoholic liver cirrhosis: A 15-year follow-up study of 100 Norwegian patients admitted to one unit. *Scand J Gastroenterol* 2004;39:858-63. | 9. Fairbanks KD. Alcoholic liver disease. <http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/hepatology/alcoholic-liver-disease/> [Accessed on 24.07.2008]. | 10. Cohen JA, Kaplan MM. The SGOT/SGPT ratio—an indicator of alcoholic liver disease. *Dig Dis Sci* 1979;24:835-8. | 11. Maddrey WC, Boitnott JK, Bedine MS, Weber FL Jr, Mezey E, White RI Jr. Corticosteroid therapy of alcoholic hepatitis. *Gastroenterology* 1978;75:193-9.