Original Research Article

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A study of clinical profile of patients presenting with abnormalities detected by upper gastrointestinal endoscopy

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ABSTRACT

Background: Alcoholic liver disease (ALD) continues to be a cause of disability and death throughout the world. According to two recent studies from the United States, 50-67% of the adult population drinks alcohol. The objective of the study was to study the upper gastrointestinal endoscopic abnormalities of patients with alcoholic Liver Disease. **Methods:** We conducted an observational, cross-sectional study to study the upper gastrointestinal endoscopic abnormalities in patients with alcoholic liver disease. A total of 97 patients, who satisfied the inclusion and exclusion criteria have been enrolled into our study.

Results: On upper gastrointestinal endoscopy, 94.73% cirrhotic patients were found to have esophageal varices (P<0.001), 23.68% cirrhotic patients were found to have esophagogastroduodenal PHG (P = 0.04) and 15.78% cirrhotic patients were found to have GAVE. None of the patients with USG findings of fatty liver / acute hepatitis had the above endoscopic abnormalities.

Conclusions: Most significant clinical association's esophageal varices and portal hypertensive gastropathy. Early detection and management of the above conditions may be helpful in lowering the disease burden and its morbidity.

Keywords: Acute hepatitis, Esophageal varices, Fatty liver

INTRODUCTION

Alcoholic liver disease (ALD) continues to be a cause of disability and death throughout the world. According to two recent studies from the United States, 50-67% of the adult population drinks alcohol. In recent years, clinicians have drawn attention to the alarming epidemic of alcohol-related liver disorders in India.

Two decades ago, it was estimated that a majority of cases of chronic liver diseases (60%) and hepatocellular carcinoma (80%) in India were due to hepatitis B virus. Approximately 10-20% cases of portal hypertension were attributed to non-cirrhotic causes, including non-cirrhotic

portal fibrosis and extra-hepatic portal venous obstruction. The earliest reports of alcohol as a significant contributor to liver diseases in India emerged from its metro cities. Even in early 1990s, alcohol has been the cause of 60% cases with cirrhosis. Simultaneously, there has been a steady decline in the tropical entity of NCPF, with a recent symposium characterizing it as a "vanishing" disease.

Upper GI endoscopy is the most commonly used method to detect oesophageal varices. All patients with suspected liver cirrhosis should be screened for oesophageal varices by endoscopy. In patients with no varices detected on initial endoscopy, endoscopy should be repeated in 2 to 3

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years. None of the various non-invasive methods of determining which patients benefit from endoscopic screening are accurate enough to recommend for routine use in clinical practice.

Upper gastrointestinal haemorrhage is one of the more important complications of cirrhosis and a major cause of death in such patients. The main sites of bleeding are oesophageal varices, gastritis, and peptic ulcers.³ Recently, characteristic lesions have been showed in the gastric mucosa in portal hypertension (congestive gastropathy). They observed variceal haemorrhage in 52.2% of cases; by gastro duodenal mucosal lesions in 13.8%; by gastric and duodenal ulcers in 13.8%; undetermined origin in 14.8% (due to coexistence of two or more lesions, without active bleeding).⁴ Alcoholic cirrhosis is by far the most common cause of bleeding oesophageal varices (BEV) in the Western world.⁵

Present study was conducted to study the upper gastrointestinal endoscopic abnormalities (esophageal varices, Gastric antral vascular ectasia- GAVE, esophagogastroduodenal Portal hypertensive gastropathy - PHG) of patients with A.L.D.

METHODS

Study site

The present study was conducted in patients admitted in the medical wards of Ispat general hospital, Rourkela.

Time frame

Over a period of one year and six months from 01.01.2013 to 30.06.2014, data on patients admitted to this hospital in the medical wards with suspected alcoholic liver disease was collected.

Study population

Data on patients admitted to this hospital in the medical wards with suspected alcoholic liver disease was collected. Among them who gave consent for the study and fulfilling all the inclusion criteria irrespective of sex discrimination were included in my study population. Approval from the ethical committee and written consent from scientific committee were obtained for the study. This is an observational cross sectional study.

Inclusion criteria

- Patients admitted with history of alcohol intake greater than 60 gms per day if male and 20 gms per day if female for > 10 years
- Physical signs suggestive of alcoholic liver diseasehepatomegaly, jaundice, ascites, hepatic encephalopathy, splenomegaly, with or without peripheral stigmata of CLD (gynaecomastia, parotid

- enlargement, dupuytren's contracture, spider angiomas)
- Patients (or relatives-when patient is incapable) giving consent before enrolment.

Exclusion criteria

- Patients of ALD not qualifying inclusion criteria.
- History of intake of hepatotoxic drugs (phenytoin, warfarin, rifampicin, acetaminophen, nicotinicacid, valproate, isoniazid, cotrimoxazole, sulfonamides, disulfiram, ketoconazole, nitrofurantoin, allopurinol, carbamazepine, hydralazine, oral contraceptive pills and androgens, tricyclic antidepressantss, chlorpromazine, flucloxacillin, amiodarone, methyl dopa)
- Patients refusing to give consent for the study.

A proforma of detailed clinical history, risk factors, physical examination, investigations is prepared. Clinical history was taken from patient (preferred) or his attendants/relatives in case patient incapable to give history (altered sensorium etc.). All patients satisfying inclusion criteria and not excluded by exclusion criteria will be followed up till date of discharge with special reference to their condition on the day of admission. A complete physical examination was done to evaluate the signs and symptoms of liver disease, presence of any respiratory, cardiovascular or any associated disease.

Investigations performed

- Upper gastrointestinal endoscopy
- Ultrasonography of abdomen

Procedure for investigations

Upper gastrointestinal endoscopy was done using video endoscope (Olympus, GIF-V-70, Tokyo, Japan).

Ultrasound examination was done in the department of radiology, by using 6-12 MHz linear array transducer with HDI 5000 Sono, ATL, USA, Ultrasound system.

Statistical methods

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD (Min-Max) and results on categorical measurements are presented in number (%). Significance is assessed at 5 % level of significance. The following assumptions on data are made.

Assumptions

- Dependent variables should be normally distributed
- Samples drawn from the population should be random; cases of the samples should be independent.

Chi-square and Fisher exact test has been used to find the significance of study parameters on categorical scale between two or more sets of variables. The results were taken to be statistically significant if P value <0.05 and strongly significant if P value ≤0.01 .

RESULTS

Table 1: Age distribution of patients studied according to gender.

Age in	Gender		Total
years	Female	Male	Total
31-40	1 (9.1%)	9 (10.5%)	10 (10.3%)
41-50	2 (18.2%)	21 (24.4%)	23 (23.7%)
51-60	6 (54.5%)	41 (47.7%)	47 (48.5%)
61-70	1 (9.1%)	14 (16.3%)	15 (15.5%)
>70	1 (9.1%)	1 (1.2%)	2 (2.1%)
Total	11 (100%)	86 (100%)	97 (100%)

The patients in this study varied between the age groups of 30-80 years. The mean±SD of age was 52.81±9.56 years. Most of the patients (48.5%) enrolled belong to the age group of 51-60 years which falls in the age range of the disease. In our study, 86 (88.7%) were males and 11 (11.3%) were females.

Table 2: Esophageal varices in patients studied according to gender.

Esophageal	Gender		Total
Varices	Female	Male	Total
Negative	1 (9.1%)	24 (27.9%)	Negative
Positive	10 (90.9%)	62 (72.1%)	Positive
Total	11 (100%)	86 (100%)	

P = 0.279, Not significant; Fisher exact test.

Esophageal varices were present in 62 male patients (72.1%) and 10 female patients (90.9%) and 72 (74.2%) of the total study population. None of the patients with USG findings of fatty liver/acute hepatitis had esophageal varices upon endoscopy. 72 patients (94.73 %) patients with USG findings of liver cirrhosis had esophageal varices upon endoscopy.

Table 3: Esophageal varices studied with respect to USG findings.

	Fatty liver	Acute hepatitis	Liver cirrhosis
Esophageal varices	0	0	72
No esophageal varices	6	15	4

The p-value for the chi-square test for this table is < 0.001** (value of the statistic is 77.1916 on 2 degrees of freedom).

Table 4: GAVE findings in patients studied according to gender.

GAVE	Gender		Total
GAVE	Female	Male	Total
Negative	10 (90.9%)	75 (87.2%)	Negative
Positive	1 (9.1%)	11 (12.8%)	Positive
Total	11 (100%)	86 (100%)	Total

P>0.999; Not significant; Fisher exact test

GAVE was present in 11 male patients (12.8%) and 1 female patient (9.1%) and 12 (12.4%) of the total study population. None of the patients with USG findings of fatty liver/acute hepatitis had GAVE upon endoscopy. 12 patients (15.78 %) with USG findings of liver cirrhosis had gastric antral vascular ectasia (GAVE) upon endoscopy.

Table 5: GAVE studied with respect to USG findings.

	Fatty liver	Acute hepatitis	Liver cirrhosis
GAVE	0	0	12
No GAVE	6	15	64

The p-value for the chi-square test for this second table is 0.1508 (value of the statistic is 3.7839 on 2 degrees of freedom. degrees of freedom).

Table 6: Esophagogastroduodenal PHG in patients studied according to gender.

Esophago-	Gender		
gastroduodenal PHG	Female	Male	Total
Negative	8 (72.7%)	71 (82.6%)	Negative
Positive	3 (27.3%)	15 (17.4%)	Positive
Total	11 (100%)	86 (100%)	Total

P=0.423; Not significant; Fisher exact test.

Esophagegastroduodenal PHG was present in 15 male patients (17.4%) and 3 female patients (27.3%) and 18 (18.6%) of the total study population. None of the patients with USG findings of fatty liver/acute hepatitis had esophagogastroduodenal PHG upon endoscopy. 18 patients (23.68%) with USG findings of liver cirrhosis had esophagogastroduodenal portal hypertensive gastropathy (PHG) upon endoscopy.

Table 7: Esophagogastroduodenal PHG studied with respect to USG findings.

Endoscopic	USG findings		
findings	Fatty liver	Fatty liver	Fatty liver
PHG	0	0	0
No PHG	6	6	6

The p-value for the chi-square test for this third table is 0.0472** (value of the statistic is 6.1069 on 2 degrees of freedom).

DISCUSSION

Patients in this study were mostly in the 6th decade (48.5%) and the mean age of presentation was 52.81 years. In another study from India the mean age of presentation was 46.97 years.⁶ The late age of presentation could possibly be related to lack of awareness and education regarding alcohol abuse and alcoholic liver disease in this part of the country. In this study, 86 (88.7%) were males and 11 (11.3%) were females. This is in contrast to many western reports. It has been shown in a number of studies that females are more susceptible to alcoholic liver damage, as alcoholic liver injury is seen in females at lower alcohol consumption. However a similar lack of female patients was reported in a study from Punjab.⁷

This could possibly be related to social and cultural background leading to hesitation on part of the alcoholic females in seeking medical help. The average alcohol intake in this study was 69.4 gms of pure alcohol. In one study the average reported daily alcohol intake was 131 gms. This could possibly be due to yet unknown genetic variations in the population of this region which might be leading to liver damage at lower amounts of average daily alcohol intake.

Also, males had a higher average daily intake of alcohol than females (P<0.001). Similar observations were made previously in another study.⁹

In this study, on upper gastrointestinal endoscopy, 94.73 % cirrhotic patients were found to have esophageal varices (p<0.001), 23.68% cirrhotic patients were found to have esophagogastroduodenal PHG (p< 0.04) and 15.78 % cirrhotic patients were found to have GAVE.

In a previous study it was mentioned that esophageal varices are present in approximately 40% of patients with cirrhosis and as many as 60% patients with cirrhosis and ascites. ¹⁰ In another study it was mentioned that esophageal varices are the most significant complication of portal hypertension, which may be associated with 20% to 80% of cirrhotic patients. ¹¹

The prevalence of PHG in patients with portal hypertension has been reported to vary between 20% and 80%. ^{12,13} The prevalence of GAVE in cirrhotics this study (15.78%) is higher when compared to previous studies in which they have observed that the prevalence of GAVE in cirrhosis is very low. In a recent study performed in patients awaiting liver transplantation, GAVE was observed in only 8/345 (2%) of the patients. ¹⁴

The absence of life-threatening endoscopic abnormalities in patients in the early stages of A.L.D (fatty liver and acute alcoholic hepatitis) explains the higher morbidity and mortality in patients who have developed liver cirrhosis and complications of portal hypertension.

CONCLUSION

Alcoholic liver disease is a cause of significant morbidity in the present study. A. L. D patients in this study were found to have a variety of abnormalities. Most significant clinical association's esophageal varices and portal hypertensive gastropathy. Early detection and management of the above conditions may be helpful in lowering the disease burden and its morbidity.

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institutional ethics committee

REFERENCES

- 1. Kim WR, Brown RS, Terrault Na. Burden of liver disease in the United States: Summary of a workshop. Hepatology. 2002;36:227-42.
- Foster SE, Vaughan RD, Foster WH. Alcohol consumption and expenditures for underage drinking and adult excessive drinking. JAMA. 2003;289:989-95.
- 3. Rabinovitz M, Yoo YK, Schade RR. Prevalence of endoscopic findings in 510 consecutive individuals with cirrhosis evaluated prospectively. Dig Dis Sci. 1990;35:705-10.
- 4. Fassio E, Viudez P, Landeria G. Upper digestive hemorrhage in liver cirrhosis: clinical and endoscopic findings. Acta Gastroenterol Latinoam. 1992;22:181-6.
- 5. Orloff MJ, Isenberg JI, Wheeler HO. Alcoholic versus nonalcoholic cirrhosis in a randomized controlled trial of emergency therapy of bleeding varices. J Surg Res. 2012;174:98-105.
- Mishra P, Desai N, Alexander J. Applicability of MELD as a short-term prognostic indicator in patients with chronic liver disease: an Indian experience. J Gastroenterol Hepatol. 2007;22(8):1232-5.
- 7. Roy K, Chawla LS, Sabharwal BD. Alcoholic liver disease in Punjab. Indian J Gastroenterol. 1985;4(3):161-2.
- 8. Hourigan KJ, Bowling FG. Alcoholic liver disease: a clinical series in an Australian private practice. J Gastroenterol Hepatol. 2001;16(10):1138-43.
- 9. Arico S, Galatola G, Tabone M. The measure of life-time alcohol consumption in patients with cirrhosis: reproducibility and clinical relevance. Liver. 1995;15(4):202-8.
- 10. Bosch J, Abraldes JG, Groszmann R. Current management of portal hypertension. J Hepatol. 2003;38(1):S54-68.
- 11. Suzuki A, Mendes F, Lindor K. Diagnostic model of esophageal varices in alcoholic liver disease. Eur J Gastroenterol Hepatol. 2005;(17):307-9.
- 12. Primignani M, Carpinelli L, Preatoni P. Natural history of portal hypertensive gastropathy in patients with liver cirrhosis. The New Italian Endoscopic

- club for the study and treatment of esophageal varices (NIEC). Gastroenterology. 2000;119:181-7.
- 13. Sarin SK, Shahi HM, Jain M. The natural history of portal hypertensive gastropathy: influence of variceal eradication. Am J Gastroenterol. 2000;95(10):2888-93.
- Ward EM, Raimondo M, Rosser BG. Prevalence and natural history of gastric antral vascular ectasia in patients undergoing orthotopic liver transplantation. J Clin Gastroenterol. 2004;38(10):898-900.

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