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Evaluating Alcohol Use Disorder Identification Test (AUDIT) Scores as a Predictor of Alcoholic Liver Disease Severity in a Coastal Tertiary Care Centre: An Observational Study

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Abstract

Alcohol consumption is a significant public health concern due to its link to various health problems, including alcoholic liver disease (ALD). Despite its long-standing presence in cultures worldwide, alcohol addiction remains a complex issue affecting individuals of all ages. This study aims to explore the correlation between alcohol abuse and the severity of ALD. Sixty adult patients with a history of significant alcohol consumption were selected for the study. Patients were divided into three groups based on alcohol abuse severity. Various clinical and biochemical parameters were assessed, including liver function tests and ultrasound findings. Statistical analysis was conducted to determine the relationship between alcohol abuse and ALD severity. Patients presented with varying degrees of ALD, with complications such as ascites, hepatic encephalopathy, and gastrointestinal bleeding. The study found a significant correlation between alcohol abuse and manifestations of portal hypertension, namely ascites and gastrointestinal bleeding. Mortality was observed only in the group with possible addiction. The study highlights the importance of addressing alcohol abuse as a risk factor for ALD complications, particularly portal hypertension-related manifestations. Further research with larger sample sizes and longitudinal studies is recommended to better understand the impact of alcohol abuse disorder on the severity of ALD.

Keywords: *Alcohol abuse, alcoholic liver disease, portal hypertension, ascites, gastrointestinal bleeding, mortality..*

Introduction:

Ethanol stands the most common preventable leading cause of hepatocyte damage throughout the globe. In the International Statistical Classification of Diseases and Related Health Problems –10th Revision (ICD-10) (rehm

et al), alcohol consumption is associated with around 230 3-digit disease and injury codes, including infectious diseases, non-communicable diseases (NCDs), and injuries. This makes ethanol consumption an inimitable risk factor for population health. Health Target 3.5, which reads, "Strengthen the prevention and treatment of substance use, including narcotic drug abuse and harmful use of alcohol," expressly mentions ethanol. One example of the greater variety of the new global development agenda and its identification of detrimental alcohol use as a development issue in and of itself is the inclusion of a separate health target under Sustainable Developmental Goals (SDG)3 to strengthen the prevention and treatment of substance use disorders, with first priority to alcohol related abuse (2).

Although drinking alcohol is ingrained in many cultures across the world, its link to a number of health problems, notably alcoholic liver disease (ALD), makes it a significant public health concern. Ethanol related liver diseases include a broad continuum of diseases, ranging from mild steatosis (fatty liver) to more serious types such cirrhosis, fibrosis, alcoholic hepatitis, and hepatocellular cancer (3). Numerous factors, including the amount and length of alcohol consumption, concurrent liver disorders, medication with hepatotoxic potential, nutritional health, and crucially, genetic predispositions, affect the seriousness and clinical course of ethanol related liver disease (ALD). For millennia, alcohol addiction has been a widespread and complex problem that affects people of all ages. Alcohol has permeated every aspect of our life, from customs from antiquity to contemporary social events. Yet, behind the veneer of conviviality lies a darker reality—a struggle that transcends cultures, socioeconomic backgrounds, and age groups. We delve into the intricate layers of alcohol addiction, exploring its causes, consequences, and potential solutions.

Ethanol, the active ingredient in alcoholic beverages, affects neurotransmitters like dopamine and Gamma Amino Butyric Acid (GABA). The euphoria and relaxation it induces reinforce consumption. Some individuals inherit a genetic susceptibility to alcohol dependence. Variations in genes related to alcohol metabolism influence tolerance and cravings (4). Occasional indulgence can escalate into overindulgent drinking—defined as consuming excessive alcohol in a short period. Binge drinking damages organs, impairs judgment, and increases the risk of accidents. Regular alcohol use leads to tolerance, withdrawal symptoms, and physical dependence. The body craves alcohol to maintain equilibrium. Beyond physical dependence, psychological cravings drive addiction. Alcohol becomes a coping mechanism for stress, anxiety, or loneliness. Broken relationships, lost jobs, and legal troubles haunt those ensnared by addiction. Alcohol exacerbates depression, anxiety, and suicidal tendencies (5). Acetaldehyde and excess hydrogen are the main causes of complications from excessive alcohol use. Low blood sugar, elevated blood lactic acid, fatty liver, and hyperlipidaemia are all caused by hydrogen. Alcoholic hepatitis is caused by fat accumulation, the impact of acetaldehyde on liver cells, and maybe other unidentified causes (6,7,8). Cirrhosis is the following stage. Drinking alcohol increases the likelihood of developing epithelial malignancies, such as HCC. Cirrhosis, end stage liver disease is the prime risk factor for Hepato cellular carcinoma (HCC), even though DNA-adducts containing aldehydes produced by ethanol oxidation have mutagenic & carcinogenic potential (9). Complex factors, such as activation of pathways that form the backbone for tumour cell survival, growth of the mutant cells, loss of cell cycle regulation checkpoints, activation of oncogenes, and telomere shortening, influence the advance of HCC in patients with cirrhosis (10).

Due to the ensuing disruption of blood chemistry brought on by the impaired liver function, there is a risk of a high ammonia level, which can cause a coma or even death (11). Additionally, excessive blood pressure in the arteries supplying the liver can lead to ruptured varices and an increase of fluid in the abdominal cavity. Cirrhosis also alters the configuration of liver, impairing blood flow. Individuals react to alcohol differently; for

example, not all heavy drinkers acquire cirrhosis and hepatitis (12).

Materials & methods:

Sixty patients with a noteworthy history of alcohol intake which is an average of 80 gms/day for more than 10 years were selected for study. Patients with comorbidities contributing to hepatocyte damage like Wilsons disease, hemochromatosis, hepatitis(viral) & patients on long term hepatotoxic drugs like anti tubercular therapy & antiepileptic therapy were not included in the study population to limit the cases with multifactorial hepatocyte damage. A properly structured informed written consent was taken from every participant or attender where the patient is conscious & oriented to understand the study design. A detailed history of ethanol consumption and clinical history were obtained from every patient. Complete physical examination including the assessment for signs of liver failure was done. Biochemical parameters effected by liver cell function like Serum Albumin, Total Bilirubin, International Normalized Ratio (INR) & renal parameters to assess the hepatorenal syndrome noted in a pre-structured proforma. Ultrasound imaging findings i.e., ascites, liver echotexture noted in a pre-structured proforma. Alcohol abuse and addiction were calculated using standard questionnaire – NoVo Psych AUDIT questionnaire. Study population is divided into 3 groups (Figure1), group with low risk – hazardous group (score of 0-15), group with harmful drinking (score of 16-19), group with possible addiction (score > 20).

All the data obtained was compared & correlation between alcohol abuse & severity of alcoholic liver disease are predicted as mean± standard deviation, while qualitative parameters like ascites, hepatic encephalopathy, hepatorenal syndrome, history and evidence of Gastrointestinal bleeding & mortality as percentage. P value was assessed by means of one-way anova calculator. P-value less than 0.05 was considered to be significant.

Results:

The study comprised of 60 patients, with mean age of 53.20±13.09, mean weight of 52±6 kgs. The study group is divided into 3 groups. Patients presented with varying presentations, jaundice (70%), melena (33%), oliguria (25%), altered senses (15 %), abdominal distension (35%). Clinical examination revealed signs of liver failure; alopecia (13.3%), fetor hepaticus (8.3%), spider naevi (26.6%), gynaecomastia (40%), jaundice (70%), ascites (35%), testicular atrophy (5%), bleeding tendency (21.6%), ankle oedema (30%). Ultrasound abdomen revealed fatty liver (21.6%), hepatomegaly (33.3%), shrunken liver (45%). Biochemical parameters revealed anaemia in 45% patients with mean HB of 10.82±3.01, thrombocytopenia n 16 % patients with mean Platelet count 177±83* 10^{3}. Liver function tests revealed hyperbilirubinemia in 38.3%, with mean bilirubin level 3.75±6.48 mg/dl. Hypoalbuminemia is noted in 61.6% with mean albumin 3.17± 0.75 g/dl. Elevated INR values noted in 26% with mean INR 1.47±0.51. Complications of liver disease observed are ascites (33.3%), hepatic encephalopathy (30%), hepatorenal syndrome (25%), Gastrointestinal bleed (38.3%). 4 out of the admitted patients expired, with mortality (6.6%).

Figure 1: Study design

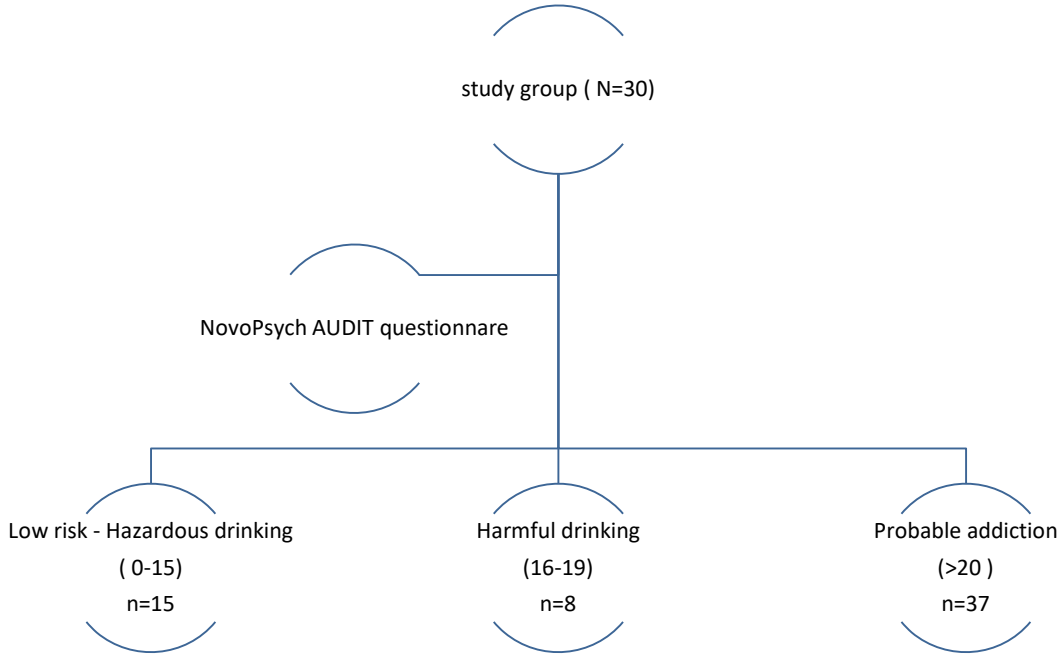
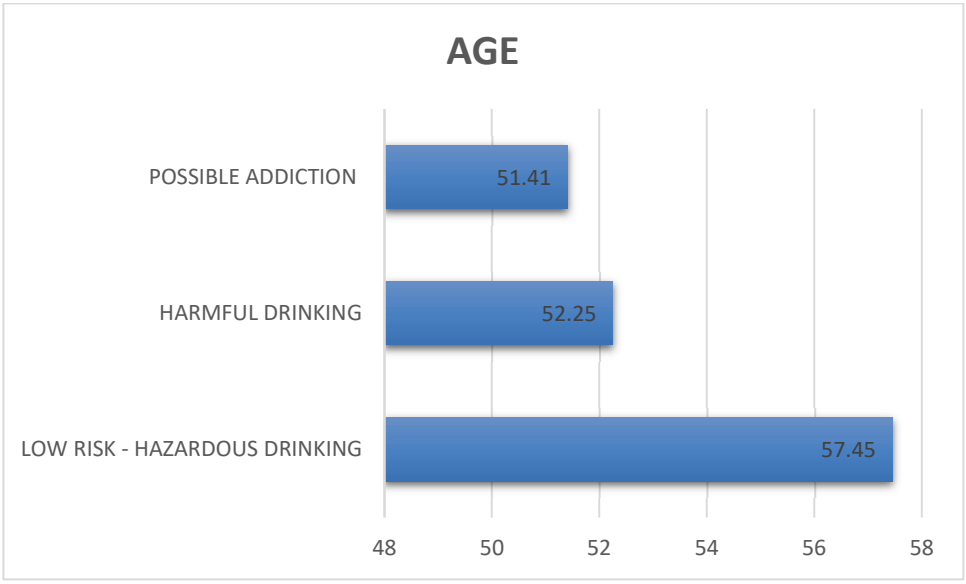
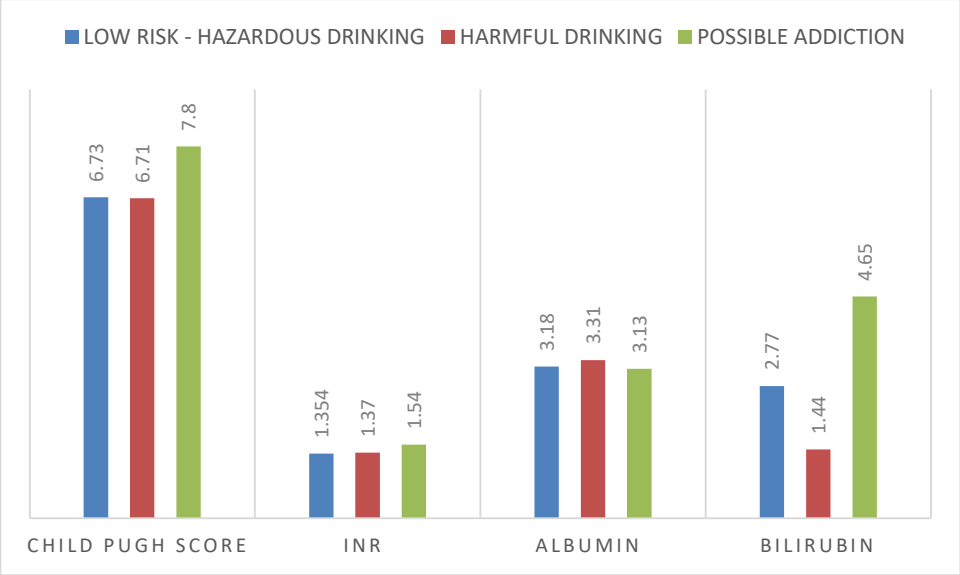


Figure 2 Age distribution in the study population



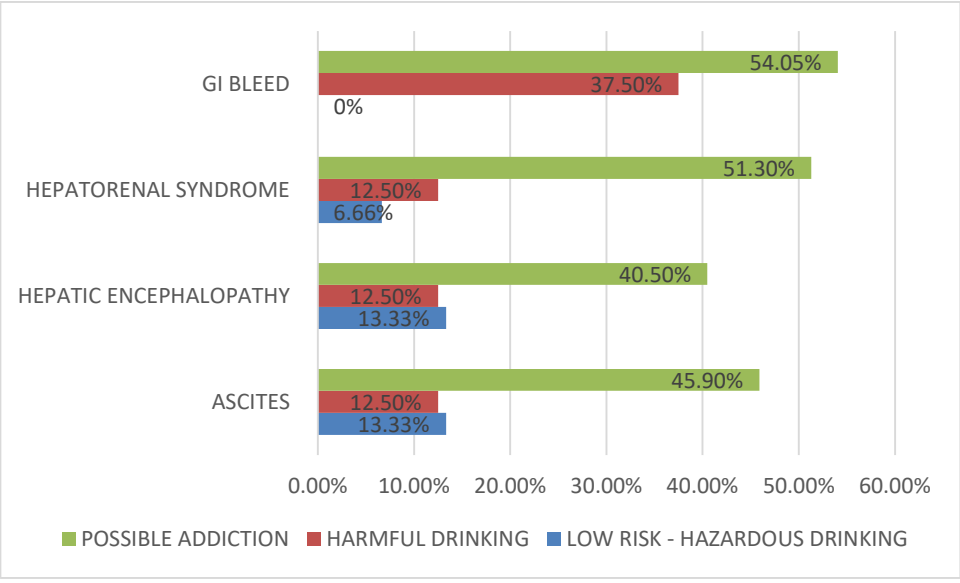
As depicted in Figure 2, The mean age of presentation in low risk – hazardous drinking is 57.45 which is almost a half-decade later when compared to groups with higher AUDIT scores (harmful drinking & possible addiction groups).

Figure 3 Comparison of parameters of liver cell failure based on AUDIT classification



As depicted in Figure 3, The mean child Pugh score was found to be comparatively lower in harmful drinking with mean of 6.71, mean child Pugh score in low risk – hazardous drinking group is 6.73, mean child Pugh score in possible addiction is 7.8 which is high in comparison to other groups. The mean INR is highest in possible addiction group of 1.54. The mean albumin is highest in harmful drinking group. the mean bilirubin is highest in possible addiction group.

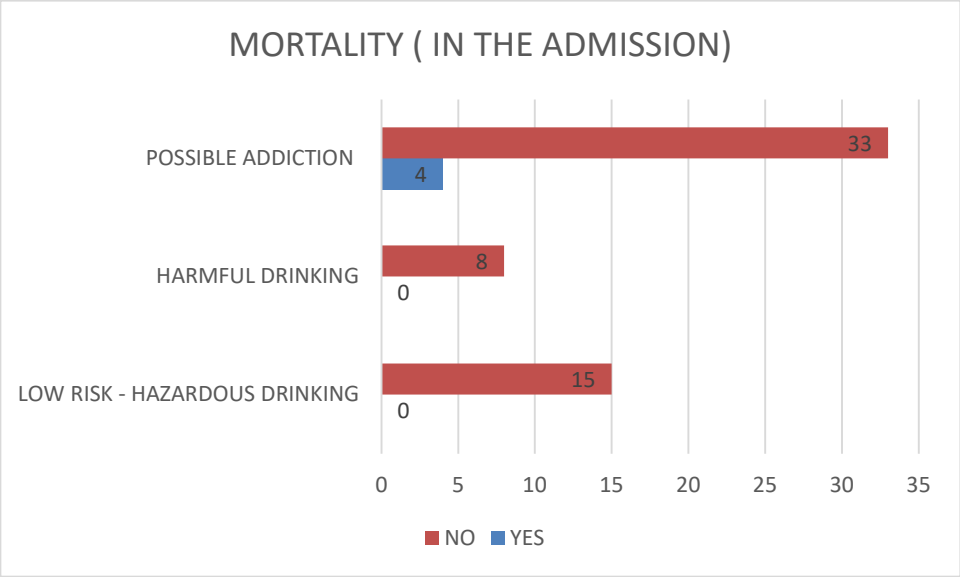
Figure 4 Comparison of complications of liver disease based on AUDIT classification



As depicted in Figure 4, in Ascites was noted in 33.3 % of study population, of which majority of the cases with ascites belong to possible addiction group (45% of group). Hepatic encephalopathy was noticed in 30 % of the study population, of which majority of cases with hepatic encephalopathy belong to possible addiction group (40.5% of the group). Hepatorenal syndrome was diagnosed in 25% of study population, however the fraction is higher in possible addiction group with 51.3%. History & evidence of gastrointestinal bleed was

traced to be in 38.3 % of study population, 54% of the possible addiction group has the history / evidence of gastrointestinal bleed

Figure 5 IN PATIENT mortality comparison based on AUDIT classification



As depicted in Figure 5, Death occurred in 6% of study population in the same admission period, with all the 4 patients belonging to possible addiction group. Figure 6 child Pugh score comparison based on AUDIT classification

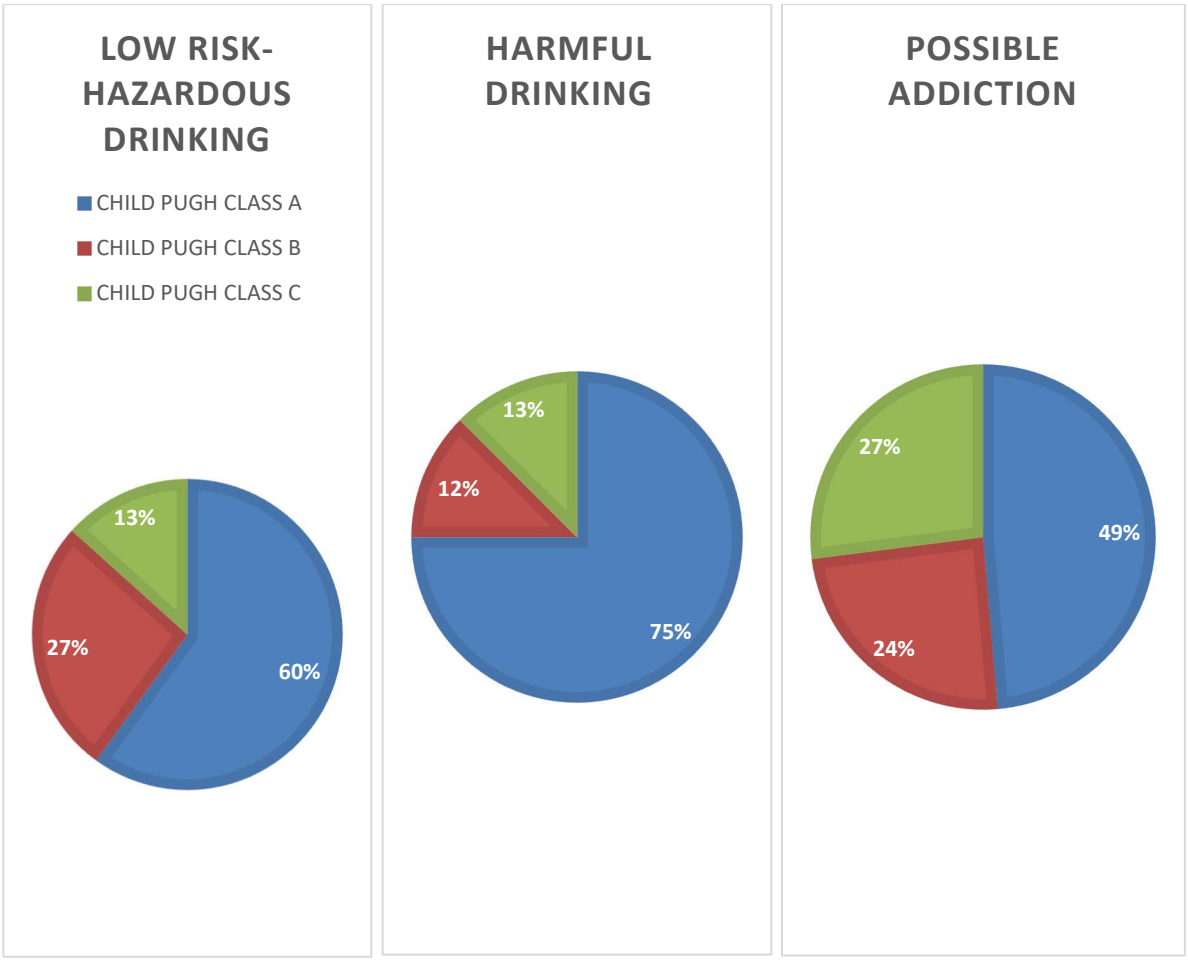


Figure 6 Child Pugh Score classification in study population

As depicted in Figure 6, the study population falls into Child pugh class A – 61.3%, Child pugh class B – 21%, Child pugh class C- 17.6%, majority of the study population fell in child pugh class A , the percentage of study group belonging to class C increased with increase in AUDIT score .

Table 1 summary of parameters among the groups based on AUDIT classification

AUDIT CLASSIFICATION	LOW RISK HAZARDOUS DRINKING (0-15)	HARMFUL DRINKING (16-19)	POSSIBLE ADDICTION (> 20)	
PARAMETERS	MEAN (±SD)	MEAN (±SD)	MEAN (±SD)	P VALUE
AUDIT SCORE	11.85±2.50	17.37±1.06	30.47±7.41	
AGE	57.45 ±14.53	52.25±15.72	51.41±11.73	0.3402
CHILD PUGH SCORE	6.73± 2.12	6.71±2.56	7.80±2.80	0.3
INR	1.354±0.38	1.37±0.43	1.54±0.57	0.4814
ALBUMIN	3.18±0.54	3.31±0.78	3.13±0.83	0.8415
BILIRUBIN	2.77±6.75	1.44±1.52	4.65±6.9	0.3612
QUALITATIVE PARAMETERS	[YES, NO]	[YES, NO]	[YES, NO]	
ASCITES	[2,13]	[1,7]	[17,20]	0.0305
HEPATIC ENCEPHALOPATHY	[2,13]	[1,7]	[15,22]	0.0791
HEPATORENAL SYNDROME	[1,14]	[1,7]	[13,24]	0.0686
GASTROINTESTINAL BLEED	[0,15]	[3,5]	[20,17]	0.0008
MORTALITY (IN THE ADMISSION)	[0,15]	[0,8]	[4,33]	0.2751

Discussion:

Matthew Baillie was the first to identify the link between alcohol consumption and cirrhosis in 1793. There have been numerous significant aspects of this illness that remain unresolved, even after extensive investigation since the 1950s. Among these imperative objections are: despite of heavy drinking why only limited candidates are prone to hepatocyte damage & land up in advanced stages of disease- cirrhosis? What else factors are contributing for liver disease development in an individual? Although recent research is focussed on the role of genetic factors, affecting the metabolism of alcohol` has attempted to explain these differences between population developing liver disease & alcoholics free of liver disease, the complete molecular pathways causing the disease remain to be established. The rationalization for the apparent inclination of certain individuals to develop ethanol related liver pathologies is undetermined. What is thought to be the most substantial risk factor for the development of ethanol related liver pathologies is the amount and duration of alcohol consumption. A prospective study design that monitors alcohol intake and records the rate at which cirrhosis develops over time is ideal, but impractical for estimating the risk function.

Studies have revealed there is relation between the severity of disease & type, duration of alcohol consumption (13,14). type of alcohol was presumed to have the effect on severity of the disease (15); however, the current study could not elicit any data on the type of alcohol being consumed as most of the participants consumed a variety of alcohol & few patients consumed local toddy where the standardization of drink could not be assessed. studies have suggested low level of alcohol consumption caries low predisposition of complications of alcoholic liver disease, reducing the presentation of decompensated alcoholic liver disease, however it remains

controversial as different studies have different norms for safe drinking (16). A similar study done in south India, has elicited that correlation between duration of alcohol consumption and the spectrum of disease , incidence of complications of liver cell damage insignificant (17). Our study group has mean age of presentation 53.20±13.09 years, similar to other studies (12,17), suggesting a proportionate relation between duration, degree of alcoholism has a role in the clinical presentation, severity of the disease. No significant relation was established in parameters of child Pugh score (table 1), except for ascites. ascites & gastrointestinal bleed – clinical signs of portal hypertension, have a significant difference among the three groups with a p- value 0.0305 & 0.0008 respectively. Mortality however did not show up to be significant, but observed only in group with possible addiction.

Conclusion

The study concludes there is significant relation of complications predominantly manifestations of portal hypertension – ascites & Gastrointestinal bleed with alcohol abuse measured by AUDIT score. However other parameters haven't proven to have statistically significant relation to the alcohol abuse & dependence. The small size of study population & nature of the study being an observational study are the study limitations. Further studies with age-matched larger sample & longitudinal studies can be more informative on the effect of alcohol abuse disorder and its correlation with severity of alcoholic liver disease.

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Declaration of interest statement

We declare no known competing financial interests or personal relationships have appeared to influence the work reported in this article.

APPENDIX

NoVo Psych AUDIT Questionnaire

1. How often do you have a drink containing alcohol?
   0 Never
   1 Monthly or less
   2 2-4 times a month
   3 2-3 times a week
   4 4 or more times a week
2. How many drinks containing alcohol do you have on a typical day when you are drinking?
   0 0 or 2
   1 3 or 4
   2 5 or 6
   3 7 to 9
   4 10 or more

Q.no		Never	Less than monthly	Monthly	weekly	Daily or almost daily
3	How often do you have six or more	0	1	2	3	4

	drinks on one occasion?					
4	How often during the last year have you found that you were not able to stop drinking once you had started?	0	1	2	3	4
5	How often during the last year have you failed to do what was normally expected of you because of drinking?	0	1	2	3	4
6	How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	0	1	2	3	4
7	How often during the last year have you had a feeling of guilt or remorse after drinking?	0	1	2	3	4
8	How often during the last year have you been unable to remember what happened the night before because of your drinking?	0	1	2	3	4

9. Have you or someone else been injured because of your drinking?

0 No
2 Yes, but not in the last year
4 Yes, during the last year

10 Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down?

0 No
2 Yes, but not in the last year
4 Yes, during the last year

11 Do you think you presently have a problem with drinking?

0 No
1 Possibly not
2 Unsure
3 Possibly
4 Definitely

12 In the next 3 months, how difficult would you find it to cut down or stop drinking?

0 No
1 Possibly not
2 Unsure
3 Possibly
4 Definitely

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