

Patterns and Severity of Alcohol Consumption in Patients with Alcoholic Liver Disease: A Cross-Sectional Study

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ABSTRACT

Introduction: Alcohol is a known cause of liver cirrhosis, with its incidence increasing in relation to the total amount and duration of intake. Excessive consumption of alcohol remains the main cause of alcohol-related liver disease and associated complications and deaths. **Aims:** To delineate the drinking patterns and severity of alcohol consumption in alcoholic liver disease patients. **Methods:** A descriptive cross sectional study was conducted among 95 patients of both sexes with the diagnosis of alcoholic liver disease (ALD), who were admitted in Medicine ward at Nepalgunj Medical College, Nepalgunj. The diagnosis of ALD was confirmed by the criteria of the ICD-10-CM. The severity of alcohol drinking screened and categorized as “low-risk drinkers,” “hazardous drinkers,” and “harmful drinkers” were based on the AUDIT score. **Results:** Among a total of 95 ALD patients, the mean age was 45.10 ± 7.60 years, the mean duration of alcohol use was 22.6 ± 7.65 years and the average amount of alcohol consumed in grams/day was 240 ± 35 . Majority of the patients consumed locally brewed alcohol, Raksi 46.3% followed by Jaad 22.1% and Others 11.6%. Very few patients consumed commercially available Spirits 6.3% or Beer 13.7%. Majority of patients were found to be drinking regular with intermittent bingeing pattern 61%, outside meal times 69.5% and hazardous drinking 53.7%. **Conclusion:** Overall our analyses indicated a precise picture of drinking patterns in ALD patients that are profoundly influenced on several cofactors like alcohol type, duration of exposure, drinking patterns, cultural habits, availability of homemade beverages and individual susceptibility. We recommend screening for alcohol abuse in all adult patients presenting to the hospital as early detection of ALD can decrease its both morbidity and mortality.

Keywords: Alcoholic liver disease, Alcohol use disorder, Alcohol consumption, Hazardous drinking, Home brewed alcohol

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INTRODUCTION

Alcohol use disorder account for a significant cause of preventable disease worldwide, with resultant alcoholic liver disease causing significant liver-related morbidity and mortality among adults with prolonged alcohol abuse.^{1, 2} Approximately 1 in 12 adults have alcohol use disorder defined as consumption of >3 drinks per day in males and >2 drinks per day in females, or binge drinking >5 drinks in males and >4 drinks in females, consumed over 2 h period.³

The three most widely recognized forms of ALD are alcoholic fatty liver (steatosis), acute alcoholic hepatitis, and alcoholic cirrhosis. Steatosis will develop in 90%-95%,⁴ 10%–35% develop alcoholic hepatitis, and approximately 10% will develop alcoholic cirrhosis.⁵ South Asian race and female sex are more prone to develop liver disease with lesser alcohol consumption.⁶ A prospective Italian study showed the risk threshold for developing ALD is 30g ethanol/day and this risk

increases with increasing daily intake.⁷ ALD is increased in those who drink without accompanying food and also in those who drink multiple different alcoholic beverages.⁷ Subjects who consumed more than 120 g/day had the highest risk of cirrhosis, with a prevalence of 13.5%.⁷ Women had greater susceptibility to ALD at any given level of intake.^{8,9}

Drinking problems occur over a broad continuum, ranging from heavy or hazardous drinking to harmful drinking. Prevalence estimates range from 4% to 29% for hazardous drinking and from less than 1% to 10% for harmful drinking.¹⁰ While it is the ethanol in spirits that is primarily responsible for liver damage, other aliphatic alcohols have even more pronounced hepatotoxic effects.^{11, 12}

METHODS

A cross-sectional study was conducted to evaluate the patterns of alcohol consumption and severity of drinking behavior of patients with alcoholic liver disease, who were admitted in

Medicine ward at Nepalgunj Medical College, Nepalgunj, Banke, Nepal, from August 2019 to August 2020 following all appropriate institutional ethics committee clearances. For inclusion, patients admitted in medicine ward with the established diagnosis of alcoholic liver disease by the criteria of the ICD-10-CM¹³ entered into the study. The patients were excluded from the study, if they showed hepatic encephalopathy, were inebriated at the time of the interview, or had any other condition that prohibited them to properly answer the questionnaire. The amount of alcohol consumed per day was calculated in grams (one unit equals 10ml or 8g of pure alcohol) and the concentration of locally brewed alcohol was taken as raksi, 25%; chhang, 12%; Jaad, 5.2%; and tongba, 5.5% obtained from the previous study done in the laboratory of the Hôpitaux Universitaires de Genève (Geneva University Hospitals, Geneva, Switzerland).¹¹ In our study, the health risk of drinking alcohol was graded using AUDIT¹² as low risk drinkers defined as those having AUDIT score of <8, while hazardous drinkers were defined as those with AUDIT score between 8 and 15. However, those with AUDIT score of ≥ 16 were classified as harmful drinkers. Developed by the World Health Organization (WHO), AUDIT incorporates questions about the quantity and frequency of alcohol use in adults to identify persons whose alcohol consumption has become hazardous or harmful. The sample size was calculated by using the formula $4pq/d^2$ (where; p=prevalence, 38.5%¹⁴; q= 100-p, 95%; d= margin of error, 10%). The sample size according to this formula was 95. A self-designed semi structured questionnaire was used to obtain the socio-demographic characteristics of the study population. Information about drinking pattern, frequencies, and other factors were also collected from a reliable informant as persons with alcohol dependence may underestimate their alcohol consumption, which is inherent in studies of this population. Data were analyzed using SPSS version 16 and descriptive analysis was performed.

RESULTS

A total of 95 patients were analyzed, of which 72 (76%) were male and 23 (24%) were females. The mean age of ALD patients was 45.10 ± 7.60 years, mean age of first drink was 20.4 years and that of alcohol abuse/dependence was 27.07 years. The mean duration of alcohol use was 22.6 ± 7.65 years. The amount of alcohol consumed in grams/day was 240 ± 35 . (Table-I). Majority of the patients consumed homemade, locally brewed alcohol, like Raksi 46.3% followed by Jaad 22.1%. and Others (Aila/Chhang/Tungba) 11.6%. Very few patients consumed commercially available spirits (whiskey, vodka, gin) 6.3% or beer 13.7% as presented in Table-I. Majority of patients were found to be drinking with regular with intermittent bingeing pattern 61% and outside meal times 69.5%. The severity of alcohol drinking was screened using AUDIT scale as summarized in Table-I, which shows that the most prevalent pattern was hazardous drinking 53.7%.

		n=95
Variables	Category	Mean \pm SD/ n (%)
Age	Mean age of ALD patients (years)	45.10 \pm 7.60
	Mean age at first alcohol use (years)	20.4 \pm 3.6
	Mean age at alcohol abuse or dependence (years)	27.07 \pm 3.14
Sex	Male	72 (76)
	Female	23 (24)
Duration of drinking	Mean duration of alcohol intake (years)	22.6 \pm 7.65
Amount of drinking	Amount of alcohol consumed (g/day)	240 \pm 35
Frequency of drinking	Regular	26 (27.4)
	Regular with intermittent bingeing	58 (61.0)
	Bingeing	11 (11.6)
Relation to meals	With meals	29 (30.5)
	Outside meal times	66 (69.5)
Types of alcohol	Raksi	44 (46.3)
	Jaad	21 (22.1)
	Beer	13 (13.7)
	Spirit (whiskey, rum, vodka, gin)	6 (6.3)
	Other (Aila/Chhang/Tungba)	11 (11.6)
Severity of Alcohol Use (AUDIT)	0-7 (Low risk drinking)	11 (11.5)
	8-15 (Hazardous drinking)	51 (53.7)
	≥ 16 (Harmful drinking)	33 (34.8)

Table I: Alcohol consumption characteristics of the study participants.

DISCUSSION

The demographic variables of our study revealed that the prevalence of ALD was higher in men 76% than in women 24%, which is consistent with previous studies.^{15, 16} The male predominance over female is most probably due to high incidence of ethanol intake among men compared to women. In the case of alcohol, social stigma may also lead to delay in seeking health care in females and it is possible that this could specifically have led to underreporting of ALD in women.

The mean age of first drink was 20.4 ± 3.6 years and that of alcohol abuse/dependence was 27.07 ± 3.14 years in our study. In a study by Johnson *et al.*¹⁷, the mean age of first drink

was 21.39 ± 5.34 years, and the mean age of alcohol abuse/dependence was 27.8 ± 5.7 years which is similar to our study. The mean duration of drinking in ALD patients in our study was 22.6 ± 7.65 years. Narawane *et al*¹⁸ and Kamper-Jorgensen *et al*.¹⁹ found that drinking for more than 14 and 20 years, respectively, was significantly more common in ALD. The average amount of alcohol consumed was 240 grams or 30 units in our study. This is similar to a study conducted by Becker *et al* in which ALD is associated with higher alcohol intake 345 g of alcohol consumption per day.⁹ Around 400 g of alcohol per day was associated with death due to liver cirrhosis related to alcohol. However, the relationship between alcohol and liver injury depends on several cofactors like alcohol type, duration of exposure, drinking patterns, and individual susceptibility. Specifically, patients with moderate alcohol drinking may still be predisposed to ALD, if they have other metabolic risk factors.¹⁹ Majority of patients were found to have a regular with intermittent bingeing pattern 61% and drinking outside meal times 69.5% in the present study. Food has an attenuating effect on alcohol.²⁰ It was observed in a study by Bellentani *et al* that persons who drink without accompanying food and also who drink multiple different alcoholic beverages have a higher risk of ALD.⁷ Their progression also depends on the pattern of alcohol intake—drinking alcohol at mealtimes results in a lower risk of liver disease than consumption at other times; intermittent drinking is more sparing for the liver than a continuous supply of alcohol.²¹ Health risk of drinking alcohol graded using AUDIT scale in our study showed that the most prevalent pattern in ALD patients was “hazardous drinking” 53.7% followed by “harmful drinking” 34.8%, which was consistent with the studies of Hilton *et al*, where the hazardous drinking and harmful drinking pattern was more associated with the development of ALD.²²

In our study, we found that most of the patients developing ALD showed increase in the consumption of locally-made alcoholic beverages like Raksi 46.3% followed by Jaad 22.1%. An increase in the risk for the development of ALD with increasing alcohol consumption was seen in patients consuming 240 grams or ≥ 30 units per day in our study. In Nepal, locally brewed alcohol is available at much cheaper rates and it is also more widely available whereas wine is consumed very rarely, mainly because it is expensive by local standards.¹¹ There are conflicting data regarding the type of alcohol consumed and the risk for developing liver disease. In a study performed in India, ALD occurred more commonly with the consumption of illicit liquor, despite its lower alcohol content.¹⁸ In yet another study, researchers found that when the alcohol intake is high, the risk for developing alcoholic cirrhosis is equal, irrespective of the type of alcoholic beverage.²³ Free radical formation after alcohol intake and a reduced level of antioxidants has been implicated in the pathogenesis of alcohol-induced liver disease.^{24,25} Locally brewed alcoholic beverages frequently

contain aliphatic alcohols as by products, and the amount of these contaminants in spirits varies considerably depending on the raw materials and production methods used.²⁶ We do not know the cause of toxicity of locally brewed alcohol, like Raksi. The toxicity of raksi may be related to the manufacturing process, the fermentation process, and the additives used. It may also be possible that raksi drinkers are more exposed to other known cofactors for liver disease than other beverages drinkers, which were not recorded in this study.

LIMITATIONS

The study only included hospitalized patients and does not reflect distribution of alcohol-related diseases in the population. In the case of alcohol, social stigma may also lead to delay in seeking health care. It is possible that this could specifically have led to underreporting of ALD in women. This knowledge is imperative to plan and develop specific alcohol prevention programs.

CONCLUSION

The findings of this study provide a precise picture of drinking patterns in ALD patients that are profoundly influenced by several cofactors like alcohol type, duration of exposure, drinking patterns, cultural habits, availability of homemade beverages and individual susceptibility. In addition, the increased risk of ALD in rakshi consumers indicates the possibility of specific toxicity for some homemade alcoholic beverages. Thus, it is imperative to devise new strategies to raise public awareness about the harmful effects of alcohol, screen alcohol drinking, and conduct brief intervention sessions in the outpatient department. Thus, a test such as the AUDIT providing data on the drinking pattern should be used for screening for alcoholism, as laboratory parameters do not help in distinguishing frequent heavy drinkers. It would also be helpful to set up abstinence clinics or organizations, with intent to convince patients with liver disease to stay away from alcohol consumption.

REFERENCES

1. Gao B, Bataller R. Alcoholic liver disease: pathogenesis and new therapeutic targets. *Gastroenterology* 2011; 141: 1572–85.
2. Bruha R, Dvorak K, Petrtyl J. Alcoholic liver disease. *World J Hepatol* 2012;4:81-90.
3. Alcohol Facts and Statistics. In: Alcoholism NIOAAa, editor. 2017
4. European Association for the Study of Liver. EASL clinical practical guidelines: management of alcoholic liver disease. *J Hepatol* 2012;57:399–420.
5. Grant BF, Dufour MC, Harford TC. Epidemiology of alcoholic liver disease. *Semin Liv Dis* 1988;8:12–25.
6. Sato N, Lindros KO, Baraona E, Ikejima K, Mezey E, et al. Sex difference in alcohol-related organ injury. *Alcohol Clin Exp Res*. 2001;25:405–455.

7. Bellentani S, Saccoccio G, Costa G, et al. Drinking habits as cofactors of risk for alcohol induced liver damage. The Dionysos Study Group. *Gut* 1997;41:845–50.
8. Becker U, Deis A, Sorensen TIA, et al. Prediction of risk of liver disease by alcohol intake, sex and age: a prospective population study. *Hepatology* 1996;23:1025–9.
9. Corrao G, Bagnardi V, Zambon A, Torchio P. Meta-analysis of alcohol intake in relation to risk of liver cirrhosis. *Alcohol Alcohol* 1998;33:381–92.
10. Saunders JB, Aasland OG, Babor TF. Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. II. *Addiction*. 1993;88:791-804.
11. Pradhan B, Hadengue A, Chappuis F, Chaudhary S, Baral D, et al. Alcoholic liver disease in Nepal: identifying homemade alcohol as a culprit. *Clinical and Experimental Gastroenterology*; 2015;183.
12. McKarns SC, Hansch C, Caldwell WS, Morgan WT, Moore SK, et al. Correlation between hydrophobicity of short-chain aliphatic alcohols and their ability to alter plasma membrane integrity. *Fundam Appl Toxicol*. 1997;36(1):62–70.
13. World Health Organization. The ICD-10-CM- International Classification of Diseases, Tenth Revision, Clinical Modification. Geneva: World Health Organization; 2019.
14. Mishra A, Shrestha P, Bista N, Bhurtel P, Bhattarai S, et al. Pattern of Liver Diseases. *JNHRC* 2009;7(1):14–8.
15. Wei H, Derson Y, Shuiyuan X, Lingjiang L, Yalin Z. Alcohol consumption and alcohol-related problems: Chinese experience from six area samples, 1994. *Addiction*. Wiley; 1999; 94(10):1467–76.
16. Maskey R, Karki P, Ahmed SV, Manandhar DN. Clinical profile of patients with cirrhosis of liver in a tertiary care hospital, Dharan, Nepal. *Nepal Med Coll J*. 2011; 13(2):115-8.
17. Johnson PR, Banu S, Ashok MV. Severity of alcoholism in Indian males: Correlation with age of onset and family history of alcoholism. *Indian J Psychiatry* 2010; 52:243-9.
18. Narawane NM, Bhatia S, Abraham P, Sanghani S, Sawant SS. Consumption of ‘country liquor’ and its relation to alcoholic liver disease in Mumbai. *J Assoc Physicians India* 1998;46:510-3.
19. Kamper-Jorgensen M, Gronbaek M, Tolstrup J, Becker U. Alcohol and cirrhosis: Dose – Response or threshold effect? *J Hepatol* 2004;41:25-30.
20. Pikaar NA, Wedel M, Hermus RJ. Influence of several factors on blood alcohol concentrations after drinking alcohol. *Alcohol Alcohol* 1988;23:289-97.
21. Marugame T, Yamamoto S, Yoshimi I, Sobue T, Inoue M, et al. Patterns of alcohol drinking and all-cause mortality: Results from a large-scale population-based cohort study in Japan. *Am J Epidemiol* 2007;165:1039-46.
22. Hilton ME Drinking patterns and drinking problems in 1984: results from a general population survey. *Alcohol Clin Exp Res*. 1987;11:167-175.
23. Pelletier S, Vaucher E, Aider R, et al. Wine consumption is not associated with a decreased risk of alcoholic cirrhosis in heavy drinkers. *Alcohol Alcohol*. 2002;37(6):618–21.
24. Reinke LA, Moore DR, McCay PB. Free radical formation in livers of rats treated acutely and chronically with alcohol. *Alcohol Clin Exp Res*. 1997;21(4):642–46.
25. Nordmann R. Alcohol and antioxidant systems. *Alcohol Alcohol*. 1994;29(5):513–22.
26. Leon DA, Chenet L, Shkolnikov VM, et al. Huge variation in Russian mortality rates 1984–94: artefact, alcohol, or what? *Lancet*. 1997;350(9075):383–88.