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# Hepatic Hazards of Quantity and Duration of Alcohol Consumption Patients Attending De-Addiction Tertiary Level Teaching Hospital Jharkhand

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**KEYWORDS**

**ABSTRACT**

**Objective**: To study the correlation between duration and quantity of alcohol consumption with biochemical markers and radiological liver function tests in patients coming to tertiary level psychiatric h.

**Methods**: The study included 1030 patients with a history of alcohol consumption for more than 3 years, presenting to the Central Institute Psychiatry, Radiology Department, Ranchi, Jharkhand, from January 2023 to December 2023. All the male patients in the adult age group with more than 3 years of alcohol consumption were included in this study. A complete hemogram, liver function, and lipid profile were done in each patient. Liver ultrasonography was also conducted. Alcohol Use Disorders Identification Test Results (AUDIT) assessment of excessive alcohol use was done.

Results: 14.56 % of patients had hemoglobin < 12 gm., with only 50% of patients having TLC count within the normal range. MCV < 100 was seen in 72.9%, biochemical jaundice in 45.72%, and raised serum GGT in 83%, SGOT in 78.05%, and SGPT in 47.08% was seen. Fatty liver in 67.08% and hepatomegaly in 74.07% were seen in this study. A significant correlation of > 80gm alcohol per day with De arranged liver function test, lipid profile, and radiological findings was observed in this study.

**Conclusion**: A significant correlation between the quantity and duration of alcohol with liver function tests, biochemical blood markers, and lipid derangements was found in this study. Primary and secondary prevention of alcohol-induced hepatic injury can be done with early detection of the same with blood markers.

## INTRODUCTION

Chronic and excessive alcohol consumption has a hazardous effect on human health and society1. The pattern of alcohol consumption differs with the amount and type consumed. In Jharkhand, Orissa, and West Bengal, the most common country-made alcohols are hadiya and mahua. They are the most widely consumed alcoholic beverages and are accepted socially in various tribal societies; however, Alcohol consumption is a public health problem with significant socioeconomic

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consequences. Hadiya is a fermented rice beer (Karahani and Gora rice as substrate and fermentation is done using ranu, a herbal root mixture that is an amalgamation of 21 herbs mixed with polished rice and made in pebble-shaped and dried for 4 days; mahua comes from flowers of Mahua tree(Madhuca longifolia), an Indian tropical tree2. Alcohol in any form causes direct hepatocyte injury, affecting the storage, synthesis, and metabolization capacity of the liver; it leads to alcoholic liver disease (ALD), which includes a spectrum of diseases from hepatic steatosis, hepatitis-necrosis, fibrosis, and cirrhosis.3,4. Clinicians can identify a minimal amount of patients who have alcohol use disorder and its hazards, as only 20-50% of patients with alcoholism present to medical care facilities5. Patients self-reporting of alcohol consumption are often used for diagnosis of alcoholism are mostly relaible6. Although alcohol is considered a direct hepatotoxic, only 10-20% progress to alcoholic hepatitis. The factors predisposing to this development may include the amount, type, and duration of alcohol consumed along with specific less obvious facts like a person's genetic predisposition, race, sex, and other co-morbid conditions. South Asian Population is more prone to develop ALD due to genetic polymorphism of the genes responsible for alcohol metabolism7

It has been seen that patients mostly hide the issue of alcohol dependence or addiction in front of doctors and other healthcare providers. As a result, most of the patients present late with end-organ damage. A clinician can stop the development and progression of alcohol-induced mental and physical ill health if problems are identified at an early age8.

One alcoholic drink has approximately 14 g (0.6 fl Oz) of pure ethanol, which is equal to 1.5 fluid ounces of distilled spirits (40% alcohol),5 fluid ounces of wine (12% alcoholic beverage), or 12 fluid ounces of regular beer (5% alcoholic beverage). Individuals with alcoholic habits mostly have deranged liver function. The biochemical markers for chronic alcohol consumption that have been most commonly studied are serum GGT, AST, ALT, mean corpuscular volume (MCV), and carbohydrate-deficient transferrin (CDT) [82-84]. An AST to ALT ratio over 2 highly suggests ALD [85, 86]. Most patients with non-ALD have AST to ALT ratios below one. Specific IgA antibodies directed towards acetaldehyde-derived protein modifications are frequently seen in alcoholics, and thus, IgA levels are increased in chronic ALD. An increased ratio of IgA to IgG is highly suggestive of ALD.

METHOD

This study was conducted in the Department of Radiology, Central Institute of Psychiatry, Kanke, Ranchi, Jharkhand, India, as a prospective analysis of retrospectively collected data from 1 year. The purpose of the study was clearly explained, informed consent were taken and the participants were assured of confidentiality

Inclusion criteria

Adult age group male patients admitted to the hospital.

Alcohol consumption with two units (drinks)/day or at least 16 g of alcohol/day for more than three years.

Exclusion criteria

Patients with non-alcoholic liver disease

A recent history of taking hepatotoxic drugs, or fever due to infective etiology,

Patients diagnosed with cardiovascular issues

Females

**STUDY INSTRUMENTS**

Fundamental questions regarding the patient's complaints, histories, and habit of alcohol and other substance use, including frequency and abstinence and medical history, were collected in a format.

To assess alcohol consumption, alcohol-related problems, and drinking manner, the Alcohol Use Disorders Identification Test (AUDIT) 8 was applied.

BIOCHEMICAL MARKERS AND LIVER FUNCTION TESTS

All the blood samples were collected on the first day of hospital admission. They underwent various pathological and biochemical tests, including a complete hemogram, liver function tests, prothrombin time, and lipid profile. The sonological evaluation of the liver included echo texture, size, fatty concentration, portal vein diameter, and blood flow in the portal vein. The blood test was done at the hospital's central laboratory, while the ultrasound was done by the radiology consultants.

DATA ANALYSIS

The SPSS version 19 (IBM Corp, Armonk, New York) was used for statistical analysis of collected data.

RESULTS

A total of 1030 patients were included in the study.

Journal of Chemical Health Risks
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Table 1-TYPE OF ALCOHOL AND NUMBER OF DRINKS PER DAY

| VARIABLE | N | PERCENTAGE |
|---|---|---|
| TYPE OF ALCOHOL | | |
| BEER | 13 | 12.6 |
| WHISKY, RUM, GIN | 195 | 18.9 |
| LOCAL SPIRITS | 520 | 50.48 |
| MIXED DRINKS | 302 | 29.32 |
| NUMBER OF DRINKS/DAY (1 drink=14gm alcohol) | | |
| </=3 (42gm.) | 82 | 7.9 |
| </= 6 (84 gm.) | 154 | 14.9 |
| </= 9 ( 126gm ) | 256 | 24.8 |
| >9 (168 gm.) | 538 | 52.23 |

In this study, local spirit alcohol, the countryside liquor of the Indian subcontinent, was consumed by one-half (50.48%) of the patients. Moreover, 52.23% were heavy drinkers (> 10 drinks/day) (Table 1).

TABLE 2- BIOCHEMICAL MARKER AND LIVER FUNCTION TEST

| variable | cases | percentage |
|---|---|---|
| Haemoglobin | | |
| <12 | 150 | 14.56 |
| 12-18 | 880 | 85.43 |
| TLC | | |
| <4500 | 110 | 10.67 |
| 4500-11500 | 520 | 50 |
| >11500 | 400 | 38.83 |
| PLATELETS | | |
| MCV | | |
| <100 | 751 | 72.9 |
| >100 | 279 | 26.99 |
| TOTAL BILLIRUBIN | | |
| 0.2-1 | 559 | 54.27 |
| >1 | 471 | 45.72 |
| SGGT | | |
| 0-47 | 175 | 16.99 |
| 48-94 | 330 | 32.03 |
| 95-141 | 123 | 11.9 |
| 142-188 | 134 | 13.0 |
| 189-235 | 103 | 10 |
| >235 | 165 | 16.01 |
| SGOT(AST) | | |
| <37 | 226 | 21.94 |
| 37-74 | 298 | 28.9 |
| 75-111 | 158 | 15.33 |
| 112-148 | 145 | 14.07 |

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| | | |
|---|---|---|
| 189-185 | 82 | 7.9 |
| >185 | 121 | 11.74 |
| SGPT (ALT) | | |
| <65 | 545 | 52.9 |
| 65-130 | 330 | 32.03 |
| 131-195 | 92 | 8.9 |
| 196-260 | 41 | 3.98 |
| >260 | 22 | 2.13 |
| SGOT/ SGPT | | |
| <1 | 216 | 20.9 |
| 1-2 | 576 | 55.9 |
| >2 | 238 | 23.10 |
| SERUM ALBUMIN | | |
| <3.4 | 149 | 14.4 |
| 3.4-5 | 844 | 81.94 |
| >5 | 37 | 2.84 |
| PT (PER SEC) | | |
| 9.5-13.5 | 577 | 56.01 |
| >13.5 | 453 | 43.98 |
| SERUM CHOLESTROL | | |
| <200 | 299 | 29.09 |
| >200 | 731 | 70.9 |
| SERUM TRIGLYCERIDES | | |
| <100 | 185 | 17.96 |
| >100 | 845 | 82.03 |
| HDL | | |
| <66 | 690 | 66.9 |
| >66 | 340 | 33 |
| LDL | | |
| <100 | 248 | 24.07 |
| >100 | 782 | 75.92 |
| VLDL | | |
| <35 | 114 | 10.75 |
| >35 | 916 | 88.91 |

TABLE 3- ULTRASOUND ADBDOMEN FINDINGS OF THE PATIENTS

| VARIABLE | N | PERCENTAGE |
|---|---|---|
| LIVER TEXTURE | | |
| HOMOGENOUS | 927 | 90 |
| HETEROGENOUS with coarse | 103 | 10 |
| LIVER SIZE | | |
| NORMAL | 267 | 25.92 |
| ENLARGED | 660 | 64.07 |
| SHRUNKEN (CHIRRHOTIC) | 103 | 10 |
| FATTY CHANGES | | |
| NONE | 236 | 22.91 |
| YES | 691 | 67.08 |

Journal of Chemical Health Risks
www.jchr.org
JCHR (2024) 14(2), 1643-1649 | ISSN:2251-6727

| | | |
|---|---|---|
| GRADES OF FATTY CHANGES | | |
| GRADE 1 | 197 | 19.1 |
| GRADE 2 | 422 | 40.97 |
| GRADE 3 | 72 | 6.99 |
| PORTAL VEIN | | |
| A)DIAMETER | | |
| <13MM | 927 | 90 |
| >13MM | 103 | 10 |
| Periportal & perisplenic collaterals | 63 | 6.11 |
| B) FLOW (CMS PER SEC) | | |
| >15 | | |
| <15 | 927 | 90 |
| | 103 | 10 |

The mean (SD) Hb level was 14.14 (2.34) g/dL; only 14.56% had a level < 12 g/dL. Half of the patients had tlc levels in the normal range of 4500-11500, and 26.99% had MCV >100.45.72% of patients had a mean total bilirubin level > 1 mg/dL. A high percentage of participants (83.01%) had an SGGT level > 47 U/L; 29.01% had an SGGT level more than 3 times the normal value. Less than one-fourth of patients (21.94%) had a mean SGOT value <37 U/L, and one-third (33.71%) had a level more than 3 times the normal value. Similarly, normal SGOT level (< 65 U/L) was seen in 52.9% of the patients and 15.01% had SGOT levels 2 times greater than normal. An SGOT/SGPT ratio > 2 was found in 23.10% of participants; the mean ± SD albumin level was 3.78 ± 0.59 g/dL; hypoalbuminemia (<3.4 gms/dl) was seen in 14.4% of participants. The mean (SD) PT value was 13.21 (1.2)/second. This study saw PT level > 13.5/second in 43.98% of patients. 70.09 % of patients had hypercholesterolemia (> 200 m/dl), and hypertriglyceridemia (>100mg/dl) was seen in 82.03% of patients. HDL below 66mg/dl was seen in 66.9% of patients. Nearly one-fourth (24.07%) of patients had LDL below 100mg/dl, and over three-fourths (88.91%) had VLDL of over 35mg/dl.

Ultrasound findings in Table 3 show that only 10% of patients had coarse echo texture of the liver, and more than half 64.07% had hepatomegaly; shrunken liver (cirrhosis) was seen in 10% of patients only. 67.08% of patients had fatty changes in the liver, and nearly half (40.97%) had grade 2 fatty liver. Normal Portal vein diameter (< 13mm) with normal flow rate (>15 cms/sec) was seen in 90% of patients.

DISCUSSION

In the present study, we have assessed the correlation of alcohol consumption, more than 80gm of alcohol, for more than 3 years with variables with biochemical markers and radiological findings.

Country-made spirits were the most common liquor consumed by 50.48% of participants, followed by whisky, rum, and gin in 29.32%. Mixed drinks were consumed by 18.9 % of patients, and a similar pattern was observed by Nand et al. 13. In the present study, only 7.9% of patients consumed less than 42 gms alcohol per day, while the rest, 92.1%, consumed more than 42 gms alcohol per day.

It was observed that 52.23% of patients had 168 gm. (9 drinks) of alcohol per day, and it was very similar to the findings of 44% of patients consuming > 9 drinks/day by Walter and Ashraf14.

Our study found anemia (Hb levels < 12 g/dL) in 14.56% of participants. In patients having > 3 drinks per day, anemia was seen in only 8.1 % of patients; in the rest, 83.3 patients Hb > 12, which was statistically significant. Normal TLC was seen in 50.4% of patients, and 47.47% of patients consuming > 3 drinks daily had statistically significant leucocytosis. Anemia was seen in 87% of patients who consumed more than 16 g or 2 units of alcohol a day and 36% had leucocytosis.13, 14.

Most of the studies have reported hyperbillirubinemia with alcohol consumption15. In our study, hyperbillirubinemia was seen in 45.72% of patients out of 44.56% of patients who had > 3 drinks per day for more than 3 years, and only 4.6% of patients had < 3 drinks per day, which was statistically significant.

In the study of Walter and Ashraf and Nand et al., Hyperbillirubinemia was seen in 40% and 80 % of

Journal of Chemical Health Risks
www.jchr.org
JCHR (2024) 14(2), 1643-1649 | ISSN:2251-6727

patients. A statistically significant correlation between the quantity and duration of alcohol with serum bilirubin level was also seen in the study of Walter and Ashraf13, 14.

In our study, 16.99% of patients had normal serum GGT levels, and the remaining 83.1 % had elevated GGT levels.

Raised serum GGT levels were seen in 81.4 % of patients with > 3 drinks per day for more than 3 years, with only 1.23% of patients with < 3 drinks per day, which was statistically significant and is similar to other studies16, 17.

Our study found raised spot and sgpt levels in 78.05 % and 47.08% of patients, respectively. A ratio of SGOT/ SGPT > 2 was seen in 23% of patients.

In many studies, a ratio of SGOT/SGPT > 2 is indicative of advanced alcoholic liver disease 18 Patients with alcohol consumption > 3 drinks per day per year had raised SGOT, SGPT, and PT as 76.4%, 45.82%, and 42.5%, respectively, however those consuming <3 Daily drinks had the above marker as 1.6%, 1.2 %, and 1.4 % respectively.

This finding was significant in our study. A positive correlation is also seen in the study of Walter and Ashraf with quantity and duration of alcohol and deranged liver profile13, 14

Our study showed a deranged lipid profile with elevated cholesterol, triglycerides, LDL, VLDL, and reduced HDL levels. Increased cholesterol, triglycerides, HDL, LDL, and VLDL patients consuming > 3 drinks per day for more than three years were seen in 68.7%, 80.1%, 30.97%, 74.85%, 87.1% wrt patients consuming <3 drinks per day 2.2%, 1.8%, 2.0%, 1.06%, 1.7% which was significant.

However, this result was different from other studies19. In our study, homogenous hepatic echo texture was present in 90.5% of patients; 67.08% had hepatic steatosis, 64% had hepatomegaly, and 10 % had shrunken heterogeneous coarse echo texture of the liver. Studies have shown that hepatomegaly in 42% of cases with shrunken liver seen in 13 % of cases13. It has been seen that ethnic and geographical factors influence the effect of alcohol along with sex, obesity, level of consumption, etc.

A Japanese study showed no significant risk in Japanese females with a history of alcohol consumption20. Studies have shown that heavy alcohol consumers had a significant adverse risk factor for hepatic steatosis in obese males17

## CONCLUSION

Alcohol is a hepatotoxic element that degrades the liver in a stage-wise manner. Alcohol-related liver diseases have a high socioeconomic impact and have significant morbidity and mortality. The alcohol-related hepatotoxicity can be assessed with biochemical markers, liver function tests, and ultrasonography. Amount and duration of alcohol intake are significant in predicting liver injury based on various biochemical and radiological markers, along with disorders in lipid profile due to alcohol. However, no single test or marker is good enough to predict liver injury on a consistent basis. Preventive care and treatment of these patients can be done by early detection of liver injuries with the help of these markers.

## REFERENCES

1. Bitton R. The global status report on alcohol and health. World Health Organisation 2011.
2. Jeewan Kumar Mitra, Praneet Ansal Mundu,Bindey Kumar, Rajendra Kumar Satapathy, Rashmi Sinha, Manish Kumar. Profile of alcoholic liver disease in population of jharkhand: an insight into the realm of alcoholism from profligacy to burden.International Journal of Contemporary Medical Research 2017;4(3):770-773.
3. OECD. Tackling Harmful Alcohol Use: Economics and Public Health Policy. Paris 2015.
4. WHO. GLOBAL STATUS REPORT on noncommunicable diseases 2014. WHO Library Cataloguing-in-Publication Data; 2014
5. Chisty SJS, Das D. Biomarkers in the treatment of alcohol use disorders. Dysphrenia. 2012;3(1):21-31.
6. Popham RE, Schmidt W. Words and deeds: the validity of self-report data on alcohol consumption. J Stud Alcohol. 1981;42(3):355-368. PubMed CrossRef Show Abstract
7. Shuppan D, Afdhal NH. Liver Cirrhosis. Lancet. 2008;371:838–851.
8. Sharpe PC. Biochemical detection and monitoring of alcohol abuse and abstinence. Ann Clin Biochem. 2001;38(Pt 6):652-664. PubMed CrossRef Show Abstract
9. Mukamal KJ. A safe level of alcohol consumption: the right answer demands the right question. J Intern Med. 2020 Nov;288(5):550-559. doi:

10.1111/joim.13129. Epub 2020 Jun 29. PMID: 32529652

10. Torruellas C, French SW, Medici V. Diagnosis of alcoholic liver disease. World J Gastroenterol. 2014 Sep 7;20(33):11684-99. doi: 10.3748/wjg.v20.i33.11684. PMID: 25206273; PMCID: PMC4155359.
11. Walter A, Ashraf M. A study correlating the quantity and duration of alcohol consumption with liver function test. IOSR Journal of Dental and Medical Sciences. 2014;13(3):70-75. CrossRef
12. ??
13. Nand N, Malhotra P, Dhoot DK. Clinical profile of alcoholic liver disease in a tertiary care centre and its correlation with type, amount and duration of alcohol consumption. J Assoc Physicians India. 2015;63(6):14-20. PubMed Show Abstract
14. Walter A, Ashraf M. A study correlating the quantity and duration of alcohol consumption with liver function test. IOSR Journal of Dental and Medical Sciences. 2014;13(3):70-75. CrossRef
15. Das SK, Nayak P, Vasudevan DM. Biochemical markers for alcohol consumption. Indian J Clin Biochem. 2003;18(2):111-118. PubMed CrossRef Show Abstract
16. 14.Gandhi PA, Sendhav SS, Sanghani HI, et al. A study on changes in serum GGT and magnesium level in alcoholic liver disease. Int J Med Res health Sci. 2014;3(1):12-15. CrossRef
17. Takahashi H, Ono M, Hyogo H, et al. Biphasic effect of alcohol intake on the development of fatty liver disease. J Gastroenterol. 2015;50(11):1114-1123. PubMed CrossRef Show Abstract
18. Nyblom H, Berggren U, Balldin J, et al. High AST/ALT ratio may indicate advanced alcoholic liver disease rather than heavy drinking. Alcohol Alcohol. 2004;39(4):336-339. PubMed CrossRef Show Abstract
19. Takahashi H, Ono M, Hyogo H, et al. Biphasic effect of alcohol intake on the development of fatty liver disease. J Gastroenterol. 2015;50(11):1114-1123. PubMed CrossRef Show Abstract
20. Cao G, Yi T, Liu Q, et al. Alcohol consumption and risk of fatty liver disease: a meta-analysis. PeerJ. 2016;4:e2633. PubMed CrossRef Show Abstract w