Original Research Article

Risk of fatty liver disease in alcoholics

P. Prathiba[1], P. Malarvizhi[2*]

[1]Assistant Professor, [2]Associate Professor
Department of General Medicine, Government Medical College, Omandurar Government Estate, Chennai, Tamil Nadu, India
[*]Corresponding author email: **vpmalar@rediffmail.com**

International Archives of Integrated Medicine, Vol. 5, Issue 7, July, 2018.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 01-07-2018 **Accepted on:** 06-07-2018
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: P. Prathiba, P. Malarvizhi. Risk of fatty liver disease in alcoholics. IAIM, 2018; 5(7): 56-61.

Abstract

Background: Fatty liver (FL) disease is commonly divided into non-alcoholic (NAFLD) and alcoholic (AFLD) FL disease categories. Although it has long been known that long-term heavy drinking is a cause of liver cirrhosis and liver cancer, the findings from recent observational studies have shown that light, moderate, and even heavier alcohol consumption may decrease the risk of FLD. This study aims to elucidate the quantity and pattern of alcohol consumption and fatty liver prevalence. We conducted a cross sectional study on semi urban population at our tertiary care centre
Materials and methods: We performed a cross sectional observational study on male patients admitted for various ailments in medical wards of Government Omandurar Medical college during the period March 2018 to May 2018.
Results: Among 100 cases fatty liver was present in 40% of cases and there is significant association between fatty liver and liver enzymes. The chances of fatty liver increase with duration of alcohol intake also there was significant association between fatty liver and BMI.
Conclusion: In conclusion, this study demonstrates that the major risk factors for FL in alcoholic are factors related to adiposity, alcohol consumption, and that consistent consumption of alcohol for prolonged period may lead to FL. These results suggest that lifestyle modifications aimed at fighting central obesity and metabolic abnormalities should be the most important recommendations for the management of FL.

Key words

Fatty liver, Alcoholic, Heavy drinking.

Introduction

Fatty liver disease (FLD) is caused by excess accumulation of fat in liver cells leading to spectrum of morphological changes ranging from steatosis, steatohepatitis to cirrhosis and hepatocellular carcinoma [1]. Fatty liver (FL) disease is commonly divided into non-alcoholic (NAFLD) and alcoholic (AFLD) FL disease categories.

NAFLD has been considered to be the hepatic manifestation in the patients with metabolic syndrome which in turn is associated with obesity and insulin resistance [2, 3]. Alcohol dehydrogenase-mediated ethanol metabolism generates a reduced form of nicotinamide adenine dinucleotide (NADH), which promotes steatosis by stimulating the synthesis of fatty acids and opposing their oxidation. The hepatic lipogenic pathway is activated after the consumption of 24 g of ethanol per day [4].

Although it has long been known that long-term heavy drinking is a cause of liver cirrhosis and liver cancer, the findings from recent observational studies have shown that light, moderate, and even heavier alcohol consumption may decrease the risk of FLD. The suggested mechanisms of protection by alcohol consumption include decreased insulin resistance, enhanced hepatic blood flow, antioxidant agents in alcoholic beverages, decreased triglyceride content in the liver, and increased circulating adiponectin [5].

This study aims to elucidate the quantity and pattern of alcohol consumption and fatty liver prevalence. We conducted a cross sectional study on semi urban population at our tertiary care centre.

Objective

- To study the role of alcohol consumption in development of fatty liver and analyse the contributory factors causing fatty liver in alcoholics.

Materials and methods

Study Design

We performed a cross sectional observational study on male patients admitted for various ailments in medical wards of Government Omandurar Medical college during the period March 2018 to May 2018.

Sampling Method

Convenient sampling technique was used.

Study Population

Inclusion Criteria

- All male patients of age group > 20 years admitted in medical wards with history of alcohol intake were included in the study.

Exclusion Criteria

- Participants who tested positive for hepatitis B antigen or hepatitis C antibody.
- Those who reported a history of known liver disease, including viral, genetic, autoimmune, drug-induced liver disease.
- Patients taking hepatotoxic drugs like ATT and HARRT.
- Female patients are excluded from the study (because of the decreased prevalence of female alcoholics in our society).

A total of 100 patients who fulfilled the inclusion criteria and did not have any of the exclusion criteria were included in the study.

Definition of fatty liver

The diagnosis of fatty liver was based on the results of abdominal ultrasonography, which was done Radiologists with without reference to any of the participant's other individual data. Of four known criteria (hepatorenal echo contrast, liver brightness, deep attenuation, and vascular blurring), the participants were required to have hepatorenal contrast and liver brightness to be given a diagnosis of fatty liver [6].

Body mass index BMI was calculated from the equation: body weight (kg)/height (m^2). Body composition was categorized according to the Western Pacific Region of WHO criteria pertaining to obesity (WPRO criteria): BMI\18.5 kg/m^2 (underweight), 18.5–22.9 kg/m^2 (normal weight), 23.0–24.9 kg/m^2 (overweight), and 25 kg/m^2 or more (obese).

Venous blood samples were taken from all subjects before nine o'clock after an overnight fast and were analyzed immediately. Alanine aminotransferase (ALT), aspartate aminotransaminase (AST), and alkaline phosphatase (ALP) activities were measured by standard laboratory procedures.

The subjects were investigated for the presence of concomitant metabolic abnormalities Diabetes mellitus (DM) was considered to be present in patients on medication for DM and/or in those with fasting blood glucose (125 mg/dl).

A standardized questionnaire was administered to all participants by the same trained team of interviewers. Habits regarding alcohol consumption were evaluated by asking the participants about the amount and type of alcoholic beverages consumed per week during the past month, then estimating the mean ethanol intake per week. The total amount of alcohol consumed per week was calculated in grams, and then categorized into the following four grades: non or minimal alcohol consumption, < 40 g/ wk; light alcohol consumption, 40-140 g/wk; moderate alcohol consumption, 140-280 g/wk; and excess alcohol consumption, > 280 g/wk [7, 8].

Statistical Analysis

Data analysis was done using SPSS software V16. The prevalence of fatty liver is expressed in percentage. The distributions of each variable were compared between FL subjects and non-FL subjects. Continuous variables, including age, BMI, ALT, AST, ALP were analyzed by t-test, and categorical variables were examined by the chi square test.

Results

Among 100 patients, fatty liver was present in 40% of the patients and among that all had Grade I fatty liver (**Table – 1**).

The alcoholic patients were further dived into groups based on amount of alcohol intake. Maximum of the cases were light drinkers 45% followed by moderate drinkers 40% (**Table – 2**).

Table - 1: Prevalence of fatty liver in alcoholics.

Total patients	FL+	FL -
100	40	60

Table - 2: Prevalence as per Amount of alcohol intake.

Cases	Minimal alcoholic	Light drinker	Moderate drinker	Heavy drinker
100	4	45	40	11

Table - 3: Age distribution.

Age (Years)	Cases	FL+	FL-	P value
Mean	42.7 yrs	46.38	40.25	0.000101
20-30 yrs	N=10	N=5	N=5	
31-40 yrs	N=30	N=10	N=25	
41-50 yrs	N=50	N=15	N=35	
>50 yrs	N=10	N=10	0	

Table - 4: BMI

BMI	Cases	FL+	FL-	P value
Mean	23.7	25.7	22.6	<0.001

Table - 5: Liver enzymes.

Enzymes	Cases	FL+	FL-	P value
AST	24%	32%	14%	<0.001
ALT	30%	52%	16%	<0.001

Table - 6: Average years of alcohol intake.

Average years	Cases	FL+	FL-	P value
	16.55	21.125	13.5	<0.0001

The cases with fatty liver FL+ were significantly elder when compared to non fatty liver cases FL- with p value 0.0001 (**Table – 3**).

BMI was significantly higher in FL (+) subjects in comparison to FL (-) subjects (P<0.001) as per **Table – 4**.

The liver enzymes AST and ALT were analysed between the FL+ and FL- group. The AST was considered to be elevated if its > 40 units⁄ liter and ALT considered to be elevated if its value > 60 units⁄liter (**Table – 5**).

The occurrence of fatty liver has significant positive association between the duration of alcohol intake of a person. Patients with FL+ have longer average years of alcohol intake when compared to FL- patients (**Table – 6**).

About 30% of patients had associated diabetes in which there was no statistical significance between the FL+ and FL- groups, p value=0.0747.

Discussion

Alcohol consumption is a common lifestyle factor and has been associated with cancer, cardiovascular diseases, type 2 diabetes, liver cirrhosis and stroke [9]. However, it has been suggested, in contrast, that moderate alcohol consumption shows a beneficial influence on coronary heart disease, stroke, type 2 diabetes mellitus, and cataract [10]. In our study, we analysed the prevalence of fatty liver in alcoholic men and factors influencing the development of fatty liver. In our study, fatty liver was present in 40% of the patients which is similar to 38.6% prevalence in the study conducted by Hiraminie, et al. [11]. Our study showed positive correlation between presence of FL+ and age. As age advance the chances of development of fatty liver increases.

Alcohol consumption of 30 g/d or more significantly increases BMI and the risk of weight gain [12]. In contrast, another study has reported that light-to-moderate alcohol consumption reduces waist circumference. Moreover, some studies have found no significant association between alcohol consumption and obesity [13, 14]. Our study showed significant correlation between FL+ and BMI.

In FL+ subjects there is significant elevation of AST and ALT. Recent investigations have elucidated some of the mechanisms by which alcohol alters liver metabolism. Two critical nuclear transcription factors, sterol regulatory element binding protein (SREBP) [15] and peroxisome proliferator activated receptor alpha (PPARa) [16], are altered with alcohol consumption. You, et al. [17] reported a role for AMP activated protein kinase activity in the action of ethanol on the liver. In addition, disturbances in the cytokine network, including alterations in the tumor necrosis factor-a (TNF-a)

[18] level, were shown to be involved in ethanol-induced steatosis. These pivotal factors, however, appear to be common in the pathogenesis of both NAFLD and AFLD. Therefore, the inverse association between FL and alcohol consumption cannot be explained by these alterations alone.

Our study showed positive correlation between average years of alcohol intake and fatty liver. As the number of years of alcohol intake increase the chances of fatty liver increases. But in study by Hiraminie, et al. [11] there is an inverse correlation between alcohol intake and FL also the studied showed in there was a significant inverse association between the frequency of alcohol consumption and the risk of FL. On the other hand, alcohol volume was not related to the risk of FL.

Limitation

Some limitations of our study should be noted. First, although ultrasonography has been validated for detecting fatty liver, it may give an incorrect diagnosis compared to liver biopsy [6]. Second, self-reported information regarding alcohol intake is frequently subject to underreporting, and misreporting could be a source of bias. Third, the generalizability of our study to women and other population is uncertain.

Conclusion

In conclusion, this study demonstrates that the major risk factors for FL in alcoholic are factors related to adiposity, alcohol consumption, and that consistent consumption of alcohol for prolonged period may lead to FL. These results suggest that lifestyle modifications aimed at fighting central obesity and metabolic abnormalities should be the most important recommendations for the management of FL. In addition, it seems unlikely that the risk of FL can be reduced by the discontinuation and/or reduction of alcohol consumption alone. Further studies are required to better understand FL pathogenesis and management.

References

1. Reddy JK, Rao MS. Lipid metabolism and liver inflammation. II. Fatty liver disease and fatty acid oxidation. American Journal of Physiology-Gastrointestinal and Liver Physiology, 2006; 290: G852–G858.
2. Kojima S, Watanabe N, Numata M, Ogawa T, Matsuzaki S. Increase in the prevalence of fatty liver in Japan over the past 12 years: analysis of clinical background. J Gastroenterol., 2003; 38: 954–61.
3. Eguchi Y, Eguchi T, Mizuta T, Mizuta T, Ide Y, Yasutake T, et al. Visceral fat accumulation and insulin resistance are important factors in non-alcoholic fatty liver disease. J Gastroenterol., 2006; 41: 462–9.
4. Siler SQ, Neese RA, Hellersein MK. De novo lipogenesis, lipid kinetics, and whole-body lipid balances in human after acute alcohol consumption. Am J Clin Nutr., 1999; 70: 923–36.
5. Moriya A, Iwasaki Y, Ohguchi S, Kayashima E, Mitsumune T, Ikeda F, Ando M, Yamamoto K. Roles of alcohol drinking pattern in fatty liver in Japanese women. Hepatology International, 2013; 7: 859–868.
6. Hamaguchi M, Kojima T, Itoh Y, Harano Y, Fujii K, Nakajima T, Kato T, Takeda N, Okuda J, Ida K, Kawahito Y, Yoshikawa T, Okanoue T. The severity of ultrasonographic findings in nonalcoholic fatty liver disease reflects the metabolic syndrome and visceral fat accumulation. Am J Gastroenterol., 2007; 102: 2708-2715.
7. Suzuki A, Angulo P, St Sauver J, Muto A, Okada T, Lindor K. Light to moderate alcohol consumption is associated with lower frequency of hyper transaminasemia. Am J Gastroenterol., 2007; 102: 1912-1919.
8. Gunji T, Matsuhashi N, Sato H, Fujibayashi K, Okumura M, Sasabe N,

Urabe A. Light and moderate alcohol consumption significantly reduces the prevalence of fatty liver in the Japanese male population. Am J Gastroenterol., 2009; 104: 2189-2195.

9. Corrao G, Bagnardi V, Zambon A, LaVecchia C. A meta-analysis of alcohol consumption and the risk of 15 diseases. Preventive Medicine, 2004; 38: 613–619.
10. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. British Medical Journal, 1999; 319: 1523–1528.
11. Yasunari Hiramine, Yasushi Imamura, Hirofumi Uto, Chihaya Koriyama, Masahisa Horiuchi, Makoto Oketani, Kaori Hosoyamada, Ken Kusano, Akio Ido, Hirohito Tsubouchi. Alcohol drinking patterns and the risk of fatty liver in Japanese men. J Gastroenterol., 2011; 46: 519–528.
12. Wannamethee SG, Shaper AG. Alcohol, body weight, and weight gain in middle-aged men. Am J Clin Nutr., 2003; 77: 1312-1317.
13. Sherwood NE, Jeffery RW, French SA, Hannan PJ, Murray DM. Predictors of weight gain in the Pound of Prevention study. Int J Obes Relat Metab Disord., 2000; 24: 395-403.
14. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: The Healthy Worker Project. Int J Obes Relat Metab Disord., 1994; 18: 145-15.
15. You M, Fischer M, Matsumoto M, Crabb DW. Ethanol induces fatty acid synthesis pathways by activation of sterol regulatory element-binding protein (SREBP). J Biol Chem., 2002; 277: 29342–7.
16. Galli A, Pinaire J, Fischer M, Dorris R, Crabb DW. The transcriptional activation of peroxisome proliferators-activated receptor a is inhibited by ethanol metabolism: novel mechanism for the development of ethanol-induced fatty liver. J Biol Chem., 2001; 276: 68–75.
17. You M, Matsumoto M, Pacold CM, Cho WK, Crabb DW. The role of AMP-activated protein kinase in the action of ethanol in the liver. Gastroenterology, 2004; 127: 1798–808.
18. Zhou Z, Wang L, Song Z, Lambert JC, McClain CJ, Kang YJ. A critical involvement of oxidative stress in acute alcohol induced hepatic TNF-alpha production. Am J Pathol., 2003; 163: 1137–46.