International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)

ISSN:2347-6567

IJAMSCR |Volume 5 | Issue 1 | Jan - Mar - 2017
www.ijamscr.com

Research article *Medical research*

The relationship between alcohol consumption, liver enzymes and high density lipoprotein cholesterol in a general population in Punjab

[1*]Rohit Rana, [2]Arun Acharya

[1]Department of Biochemistry, Punjab Institute of Medical Sciences, Punjab, India*
[2]Department of Biochemistry, Norvic International hospital, Kathmandu, Nepal
Corresponding Author**: **Rohit Rana

ABSTRACT

Background

Health problems related to lifestyle and behavior are increasingly common in modern societies. Recent studies have indicated that the common liver enzymes, gamma-glutamyltransferase (GGT), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) are increased in alcoholics. In this study we analyzed blood sample of chronic alcoholics.

Methods

Alcohol consumption, smoking, coffee drinking, income, education, food habits were analyzed using detailed questionnaires. The mean age of study group was 41.95 ± 8.45 years.

Results

Serum AST was found to be 36.92 ± 26.35 U/L, serum ALT was found to be 53.59 ± 31.24 U/L, serum ALP was found to be 102.47 ± 29.03 U/L. and serum GGT was found to be 66.63 ± 30.96 U/L. Serum HDL-Cholesterol was found to be 44.68 ± 11.66 mg/dl.

Conclusion

The result showed that alcoholics had increased serum liver enzymes and decreased serum HDL- Cholesterol.

Keywords: Alcohol, Alcohol liver disease, Liver enzymes, High density lipoprotein.

INTRODUCTION

Alcoholism is a chronic illness with a slow, insidious onset, which may occur at any age. The cause is unknown, but genetic, cultural and psychosocial factors are suspected and families of alcoholics have a higher incidence of this disease. Chronic alcohol abuse can lead to feelings of guilt and shame as well as to broken relationships and broken family due to family's lack of control over the alcohol intake [1]. According to Organization for Economic Cooperation and Development (OECD) report released in May 2015, alcoholism increased by about 55 percent between 1992 and 2012. It is a continuously rising and is a cause of concern among the youth of the World [2]. In 2014, the World Health Organization released its Global Status report on Alcohol and Health. According to the report, about 38.3 percent of the world's populations consume alcohol regularly. On

an average an individual consumption amounts to 6.2 liters of alcohol every year. According to the WHO report published in 2010, 30 percent of India's population, (just less than a third of the country's population) consumed alcohol regularly. Some 11 percent are moderate to heavy drinkers. The average Indian consumes about 4.3 liters of alcohol per annum. The rural average is much higher at about 11.4 liters a year [3]. Prolonged alcohol consumption affects the liver enzymes. Four enzymes are measured in the laboratory to evaluate function of the liver. These enzymes include Aspartate Aminotransferase (AST),

Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), and Gamma Gutamyltransferase (GGT). Alanine aminotransferase (ALT) originates primarily from the hepatocytes, whereas Aspartate aminotransferase (AST) is found additionally in the heart, skeletal muscle tissue, kidney and brain. As a consequence, serum AST may also increase in response to pathological processes in the heart or skeletal muscle, while serum ALT is considered a fairly specific marker of liver disease. Elevated serum aminotransferase levels can be found in asymptomatic patients for a variety of reasons, e.g. excessive alcohol intake, overweight, viral or autoimmune hepatitis, hemochromatosis, Wilson's disease, alpha 1-antitrypsin deficiency, celiac disease, genetic disorders in muscle metabolism, acquired muscle diseases, or strenuous exercise [4]. Alkaline phosphatase (ALP) is an enzyme and its pathological elevations are commonly observed in liver and bone diseases, although the enzyme may originate from several other tissues [5]. Since elevated ALP is a somewhat unspecific parameter, it needs to be interpreted in the context of a clinical diagnosis and other laboratory markers.

Increased serum GGT activity has long been used in clinical practice as a marker of both liver dysfunction and excessive alcohol intake [6]. GGT is known to increase in all forms of liver disease, especially in cases of biliary obstruction, with small increases (2–5 times normal) observed in connection with fatty liver, so that GGT is of limited value for the purpose of screening alcohol consumption per se in patients with non-alcoholic liver diseases or in hospitalized patients, for instance [7, 8]. In this study we found that the effects of alcohol on lipoproteins in cholesterol transport, as well as the novel effects of lipoproteins on vascular cell wall, comprise a complex mechanism through which alcohol is cardio protective [9]. Elizabeth R et al. in a study showed that alcohol consumption raises HDL cholesterol levels by increasing the transport rate of Apo-lipoproteins A1 and A2. It concluded that alcohol intake increases HDL-C in a dose dependent fashion, associated with and possibly caused by an increase in the transport rate (TR) of HDL Apo-lipoproteins A1 and A2 [10]. During three weeks of moderate alcohol consumption, an increase in Apo A-1 is followed by an increase in HDL cholesterol. The kinetics and sequence of these increases may be an additional mechanism of action underlying the reduced coronary heart disease risk in moderate drinkers [11]. Heavy alcohol consumption can adversely affect essentially every organ system [12]. There is evidence that chronic consumption of as little as two drinks per day increases the risk of upper respiratory and upper digestive tract malignancies and breast cancer. The relative risk of oral and pharyngeal cancers associated with two drinks per day is 1.75; the same level of alcohol consumption is associated with a relative risk of 1.51 for esophageal cancer [13]. The relative risk of colon cancer associated with two drinks per day is 1.08. In a meta-analysis of 53 studies the relative risk of breast cancer in women was 1.32 for an average intake of 35 to 44 g/d of alcohol per day, and 1.46 for those consuming more than 44 g/d [14].

MATERIALS & METHODS

Study population

One hundred and thirty two alcoholics were enrolled in this study. The mean age of the alcoholics was 41.95 ± 4.45 years .All the subjects were male. The longest duration of alcohol abuse was 15 years while the shortest duration of abuse was 2 years. Majority of the study population ingested at least 150 ml of alcohol daily. Majority of the study population had studied up to class XII. Few subjects of the study population were cigarette smokers. Average income of the study population was above 30,000 rupee per month. Majority of study population had no physical activity. Around 50% of the alcoholic subjects had hypertension. More than 25% of the study population had either of the parents suffering from diabetes. Hundred and

eight males of age group 20 to 60 years were taken as controls. They had no history of alcoholism. The liver enzymes, (AST, ALT, GGT, ALP) and HDL-C concentration in the control subjects was within the normal reference range. Informed written consent was also obtained from these subjects. Subjects between 20 to 60 years taking at least 150 ml of alcohol daily for one year and above were included in the study. In this study, pregnant women, elderly (above 60 years) and children below 20 were excluded. A subject addicted to any other drug was also excluded from the study. Patients suffering from liver cancer and chronic heart disease were also not included in the study.

Alcohol consumption assessment

Alcohol consumption was assessed during the interview using the questionnaire. Participants were asked to provide information regarding whether they regularly consumed alcohol, their average alcohol consumption per day, and the number of days per month that they consumed alcohol. The ethanol weight content differed among beverages: 5% for beer, 12.5% for red wine, and 45% for hard liquor. One drink was defined as an average of 15 g of ethanol [15]. We used cut-off values based on the definition of daily alcohol consumption from the National Institute on Alcohol Abuse and Alcoholism to classify the participants' level of consumption: non-drinkers (abstainers, no alcohol consumption history), moderate drinkers (up to 1 drink/day for women and up to 2 drinks/day for men), and heavy drinkers (>1 drink/day for women and >2 drinks/day for men) [16].

Laboratory measurements

The various biochemical parameters were measured in the laboratory of the Punjab Institute of Medical Sciences using standard clinical chemical methods. Serum AST, ALT, ALP and GGT activities were measured by standard kinetic methods following the recommendations of the test according to International Federation of Clinical Chemistry and Laboratory Medicine (IFCC), and Serum high-density lipoprotein cholesterol (HDL-C) activity was measured by HDL- C Immune FS homogeneous method using BS 400 clinical chemistry analyzer (fully automated Mindray Machine).

STATISTICAL ANALYSIS

Values were expressed as means ± SD or means ± 95% confidence interval (CI), as indicated. Differences between correlations were analyzed with the t-test for comparison between alcoholics and non-alcoholic parameters. The SPSS 24 version, statistical software packages for Windows was used for the statistical analyses (SPSS Inc., Chicago, IL, USA). P-value of < 0.05 was considered statistically significant.

RESULTS

The mean age of patients was 41.95 ± 8.45 years while that of control was 41.03 ± 9.44 years, r value = 0.44. There was no statistical significant difference between the age of alcoholics and control subjects considering $p < 0.05$.

Independent sample t-test was used to analyze the result of liver enzymes and HDL cholesterol among alcoholics and non- alcoholics of matched aged group. The mean serum AST of alcoholics was 36.92 ± 26.35 U/L, which shows a moderate rise from the normal range, while that of the control group was 24 ± 4.62 U/L, which is typical for a normal population (0-40 I/U). To test the hypothesis that alcoholics and non-alcoholics were associated with derangement in liver enzymes and HDL cholesterol, an independent sample t- test was performed. The alcoholic and non- alcoholic subjects' distribution was sufficiently normal for conducting a t-test. The independent sample t-test was statistically significant at $p < 0.05$ ($p = 0.000$) for AST. Among alcohol consumers 26.5% increase in serum AST was observed.

The mean SGPT of alcoholic patients was 53.59 ± 31.24 U/L (higher than reference range). While that of control group was 27.52 ± 7.22 U/L (within reference range). There was a statistically significant difference observed between the alcoholic group and non- alcoholic group at $p < 0.05$ ($p=0.000$). Among alcohol consumers, 68.1% increase in serum ALT was observed (range 0-40 U/L).

The mean value of ALP value of alcoholic patients was 102.47 ± 29.03 U/L (higher than reference range) and that of control was 71.95 ± 9.93 U/L (which is within in normal range). There was a statistically significant difference between the ALP value of alcoholics and non- alcoholics

subjects at p <0.05 (p=0.000). Among alcohol consumers, 66% increase in Serum ALP was observed (range 25-90 U/L).

The mean GGT value of alcoholic patients was 66.63 ± 30.96 U/L, while that of control group was 29.42 ± 7.38 U/L. There was a statistically significant difference between the alcoholics and control group at p<0.05 (p=0.000). Among alcohol consumers, 58.3% increase in serum GGT was observed (range 11-50 U/L).

The mean HDL value of alcoholic patients was 44.68 ± 11.66 mg/dl, while that of control group was 50.90 ± 7.26 mg/dl. This shows that the HDL-C value is higher in controls than alcoholic consumers. Statistically significant difference was observed between the control group and alcoholics at p<0.05 (p=0.000). This suggests that alcohol consumption leads to decrease in HDL cholesterol. Among alcohol consumers 37.80% decrease in HDL cholesterol was observed (range 35-70 mg/dl).

Table 1: Data has been reported as mean ± SD in group in two different groups.

PARAMETERS	ALCOHOLIC MEAN ± S.D (n= 132)	CONTROL MEAN ± S.D (n=108)	p-value
AGE	41.95±8.45	41.03±9.44	0.44
AST	36.92±26.35	24±4.62	0.000
ALT	53.59±31.24	27.52±7.22	0.000
ALP	102.47±29.03	71.95±9.93	0.000
GGT	66.63±30.96	29.42±7.38	0.000
HDL	44.68±11.66	50.9±7.26	0.000

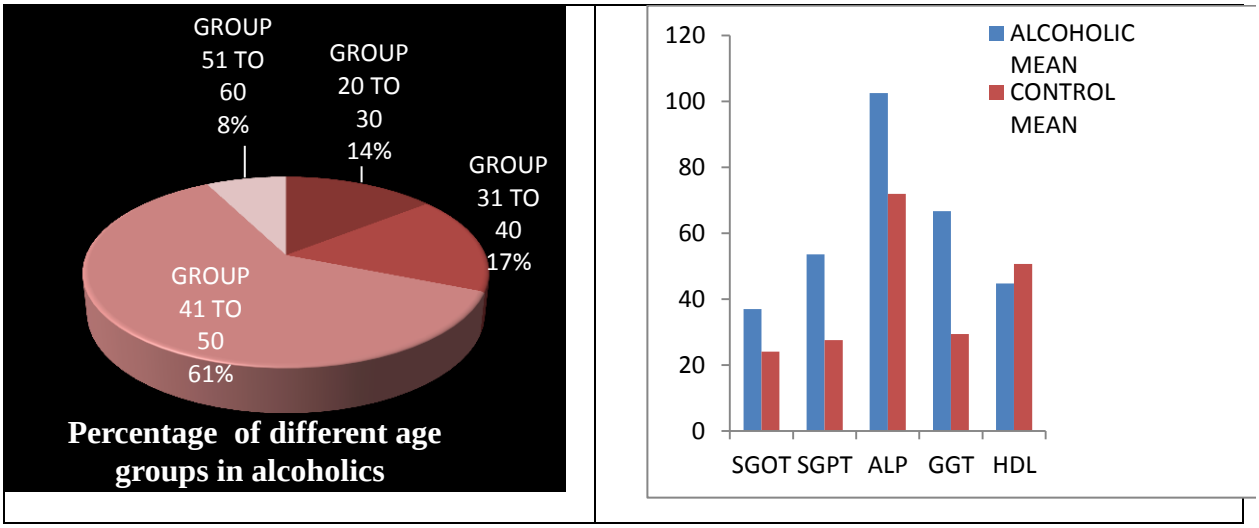

Fig 1: Graphs representing alcoholic mean and control mean.

DISCUSSION

The study shows that alcoholics have higher value of liver enzymes such as AST, ALT, ALP and GGT, when compared with non-alcoholic, age matched subjects. A brief dietary history showed that alcoholics consumed more of dietary fats, had less physical activity leading to positive calorie balance and obesity. There was an increase in the value of AST by 26.5%, ALT by 68.1%, ALP by 66% and GGT by 58.3% in alcoholics. Teddy Charles Adias et al. in a study found that the value of prothrombin time, activated partial thromboplastin time, and ALT, AST, GGT were highly elevated in chronic alcoholics [17]. The present study shows that there was a significant decrease in serum HDL-C among alcoholics when compared with non-alcoholic subjects. HDL cholesterol value was decreased by 37.80% in alcoholics. This finding is contrary to a study by Hidekatsu Yanai et al. which reported an increase in serum HDL-C in moderate alcoholics [18].

CONCLUSION

From the result of this study, it can be concluded that alcohol has detrimental effects on the liver. It was observed that the liver enzymes (AST, ALT, ALP and GGT), were raised above the reference range in the alcoholic subjects. This rise is due to the deleterious effect of ethanol on hepatocytes, causing leakage of cytosolic enzymes into the blood stream. Also a decrease in high density lipoprotein cholesterol (HDL-C), in sera of alcoholic subjects observed, can lead to higher risk of development of coronary heart disease.

Acknowledgement

This study was supported by grants from the Punjab Institute of Medical Sciences (PIMS) Jalandhar (Punjab) India.

REFERENCE

[1]. Merriam Webster's medical dictionary. Definition of Alcoholism. 2017. www.merriamwebster.com/dictionary/alcoholism.
[2]. Alcohol consumption rising fast in India; OECD report. The Indian Express. 2015.
[3]. WHO. Global status report on alcohol and health [Internet]. 2014. Available from: http://www.who.int/substance_abuse/publications/global_alcohol_report/msb_gsr_2014_1.pdf.
[4]. Niemelä O. Biomarkers in alcoholism.Clin Chim Acta, 377(1-2), 2007, 39-49.
[5]. Edoardo G. Giannini, Roberto Testa, Vincenzo Savarino. Liver enzyme alteration: a guide for clinicians. CMAJ, 172(3), 2005, 367–79.
[6]. Giannini EG, Testa R, Savarino V. Liver enzyme alteration: a guide for clinicians. CMAJ, 172(3), 2005, 367-79.
[7]. Whitfield JB. Gamma glutamyl transferase. Crit Rev Clin Lab Sci., 38(4), 2001, 263-355.
[8]. Salaspuro M. Alcohol consumption and cancer of the gastrointestinal tract. Best pract Res Clin Gastroenterology, 17, 2003, 679-94.
[9]. Hannuksela ML, Liisanantt MK, Savolainen MJ. Effect of alcohol on lipids and lipoproteins in relation to atherosclerosis. Crit Rev Clin Lab Sci, 39(3), 2002, 225-83.
[10]. Aggarwal DP. Cardioprotective effects of light-moderate consumption of alcohol: A review of putative mechanisms. Alcohol, 37, 2002, 409-15.
[11]. Perret B, Ruidavets JB, Vieu C, et al. Alcohol consumption is associated with enrichment of high-density lipoprotein particles in polyunsaturated lipids and increased cholesterol esterification rate. Alcohol Clin Exp Res, 26, 2002, 1134-40.
[12]. Saitz R. Overview of medical and surgical complications. In: Graham AW, Schultz TK, Mayo-Smith MF, editors. Principles of Addiction Medicine. American Society of Addiction Medicine; 2003, 1027-52.
[13]. Corrao G, Bagnardi V, Zambon A, et al. Exploring the dose-response relationship between alcohol consumption and the risk of several alcohol-related conditions: A meta-analysis. Addiction, 94, 1999, 1551-73.
[14]. Bagnardi V, Blangiardo M, La Vecchia C, et al. A meta-analysis of alcohol drinking and cancer risk. Br J Cancer, 85, 2001, 1700-05.
[15]. Qiao Y., Shi R., Hou B., Wu L., Zheng L., Ding L., et al. Impact of alcohol consumption on substrate remodeling and ablation outcome of paroxysmal atrial fibrillation. J. Am. Heart Assoc, 9, 2015, 4: 1-10. Available from: DOI: 10.1161/JAHA.115.002349.
[16]. National Institute on Alcohol Abuse and Alcoholism. The Physicians' Guide to Helping Patients with Alcohol Problem. NIH; Bethesda, MD, USA: 1995.
[17]. Teddy Charles A, Everton E, Osaro E. Evaluation of coagulation parameters and liver enzymes among alcohol drinkers in Harcourt, Nigeria. Int J Gen Med, 6, 2013, 489–94.
[18]. Kyoko Kowaga Sato et al. Liver Enzymes Compared With Alcohol Consumption in Predicting the Risk of Type 2 Diabetes. Diabetes Care, 31, 2008, 6.

How to cite this article: Rohit Rana, Arun Acharya. The relationship between alcohol consumption, liver enzymes and high density lipoprotein cholesterol in a general population in Punjab. Int J of Allied Med Sci and Clin Res 2017; 5(1): 168-172.
Source of Support: Nil. **Conflict of Interest:** None declared.