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Biochemical changes in Lipid parameters Due To Chronic Alcoholism

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ABSTRACT It is well known that chronic alcoholics have altered lipid profile. Alcohol ingestion produces changes in blood lipids as well as changes in liver lipids. The administration of alcohol causes elevation in plasma triglycerides. In present study we examined lipid profile in moderate and heavy alcohol drinkers. Present study is case control type in which fifty chronic alcoholics were selected and divided into two groups on the basis of amount and duration of alcohol consumption. Lipid profile measurement was done at fasting states which include cholesterol, triglycerides, HDL-C, LDL-C and VLDL-C. and findings were analyzed and compared with matched controls. Data were presented as mean±SD and t test is performed. The level of significance was taken as p values< 0.05. All lipid profile parameters are significantly higher in cases when compared with controls. Significant result was obtained when moderate drinkers were compared with heavy drinkers. Our findings show significantly altered lipid profile in moderate and heavy drinkers and increased risk of various disorders.

KEYWORDS : Lipid Profile, Alcohol Cholesterol and Triglycerides.

INTRODUCTION

Alcohol is a non-essential nutrient. It acts as a sedative, tranquillizer, hypnotic or anesthetic depending upon the quantity consumed. The consequences of alcohol consumption on health vary according to the extent and habits of usage (excessive or not, acute or chronic) and depend on numerous environmental and individual factors. India has been identified as the third largest market for alcoholic beverages in the world. Changing social norms, urbanization, increased availability, high intensity mass marketing along with poor levels of awareness related to alcohol has contributed to increase in alcohol use. [1]

Lipids are one of the necessary components which control cellular functions and homeostasis. Liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation. Therefore, it is reasonable to expect an abnormal lipid profile in those with severe liver dysfunction.[2]

Influence of alcohol use on lipid metabolism is well recognized [3]. Chronic alcohol abuse can potentially affect almost every organ system resulting in serious illness such as liver diseases [4], impaired heart function and inflammation of the pancreas [5]. Alcoholic liver disease remains one of the most common causes of chronic liver disease worldwide and is usually accompanied by hepatitis, cirrhosis and/or hepatocellular cancer [6]. Alcohol promotes accumulation of fat in liver mainly by substitution of ethanol for fatty acids as the major hepatic fuel [7] [8]. Alcohol facilitates esterification of the accumulated fatty acids to triglycerides, phospholipids and cholesterol esters, all of which accumulate in the liver [9]. The accumulated lipids are disposed of in part as serum lipoprotein, resulting in moderate hyperlipemia. In some individuals with pre existing alterations of lipids metabolism, even small ethanol doses may induce marked hyperlipemia, which responds to alcohol withdrawal [10]. Inhibition of catabolism of cholesterol to bile salt may contribute to the hepatic accumulation and hypercholesterolemia. Lipoprotein production and hyperlipemia increases during chronic alcohol consumption but as liver injury aggravates, hyperlipemia wanes and liver steatosis is exaggerated. Therefore changes in serum lipids may be a sensitive indicator of the progression of the liver damage in alcohol dependent individuals.

Various previous studies have shown that, long-term alcohol abusers have altered lipid profile, in our study we have examined lipid profile in a group of moderate and heavy drinkers compared with age matched controls. The purpose of our study is to investigate the long term effect of alcohol on serum lipids.

MATERIAL AND METHODS

The sample of our study comprised of 100 subjects which are randomly enrolled over 1 year period in tertiary care center in Hamidia Hospital associated with GMC Bhopal. Informed consent was obtained from each participant whose participation in project was voluntary detailed information of the objectives of the study and research therapeutic protocol was provided to all subjects. Permission for study was obtained from scientific committee and institutional ethical committee for biomedical research.

Inclusion Criteria: all the patients selected were confirmed for alcohol dependence after taking complete history and all were of age group 20-60 years. Control group include mainly employee of the hospital and relatives of patients which are age matched with morbid group and non-alcoholics.

Exclusion Criteria: patient with liver injury other than due to alcoholism and family history of alcohol dependence are excluded from the study.

5 ml blood was collected from anterior cubital vein under all aseptic precautions. Subjects were fasted for 12 hours. Lipid profile include estimation of Total cholesterol (TC), High density lipoprotein (HDL), low density lipoprotein (LDL), Triglycerides (TG), and Very low density lipoprotein (VLDL).

Estimation of lipid profile

Fasting lipid profile was done in all subjects. Serum cholesterol, HDL-C and VLDL-C were determined by CHOD-PAP method (Roche Diagnostic). Serum Triglycerides was measured enzymatic GPO-POD method (Roche Diagnostic). LDL-C calculated by using Friedwalds Formula (LDL = Total cholesterol-1/5 TG-HDL)[11]

Statistical Analysis

Data are presented as mean±SD. To test the significance b/w the study group and control groups were analyzed by student's t test. The p value ($p<0.05$) was considered to be significant.

RESULT

Table 1. Age wise distribution of cases (n=50)

S.No	Cases (Age-wise)	No of patients	M±SD
1.	20-29	15	27.2±1.74
2.	30-39	10	34.5 ±3.02

3.	40-49	12	45.5± 2.87
4.	50-59	13	54.33 ±2.83

Table No 1 shows distribution of cases according to age group, four groups are made. Number of patients and their mean age of each group have been presented.

Table 2. Comparison of lipid profile is cases and control

No	Lipid profile	Control		Cases		p. value
		NO	M± SD	No	M± SD	
1.	HDL	50	42.34±8.44	50	69.25±15.56	<0.001*
2.	LDL	50	80.45±12.22	50	92.41±13.43	<0.001*
3.	VLDL	50	37.250±4.100	50	54.58±8.430	<0.001 *
4.	TC	50	162.72±13.73	50	204.62±25.23	<0.001 *
5.	TG	50	185.44±19.08	50	273.15±42.30	<0.001 *

* Significant

Table No 2 shows comparison of lipid profile in controls and cases. All the parameters HDL, LDL, VLDL, TC, and TG were elevated in cases when compared to controls, the elevation was highly significant (p<0.001).

Table 3. Comparison of cases on the basis of duration of alcohol (Lipid profile)

S. NO	Lipid Profile	8-10Y	10<Y	p.VALUE
1.	Duration	9.41±0.74	12.26±1.46	< 0.001**
2.	HDL	65.49±71.04	71.04±12.01	< 0.01*
3.	LDL	89.90±13.70	90.00±13.05	>0.05
4.	VLDL	53.50±8.36	54.86±8.45	>0.05
5.	TC	198.80±20.8	207.02±27.17	>0.05
6.	TG	267.53±41.81	274.65±42.49	>0.05

* Significant

The cases were divided into two groups on the basis of duration of alcohol consumption; first group was in range of 8-10 years with mean of 9.41±0.74 and second group comprised of patients who consume alcohol for more than 10 years with the mean of 12.26±1.46. The difference b/w two groups were highly significant. However result shows that only HDL was significantly higher in cases when compared to controls, and other parameters shows non- significant result.

Table 4. Comparison of lipid profile in cases on the basis of amount of alcohol

NO		MODERATE DRINKERS (80-100ML)	HEAVY DRINKERS (100<ML)	P.VALUE
1	AMOUNT	90.37± 9.09	127.61±15.33	<0.001*
2	HDL	66.42± 17.42	66.35±25.68	>0.05
3	LDL	90.64±14.18	89.91±12.26	>0.05
4	VLDL	52.27±9.82	57.30±5.46	<0.01*
5	TC	200.28±28.86	209.71±19.59	>0.05
6	TG	261.39±49.12	286.96± 27.64	<0.01*

*Significant

The cases were divided into two groups on the basis of amount of alcohol consumption; first group was in range of 80-100 ml / day with mean of 90.37± 9.09 and second group comprised of patients who consume alcohol more than 100 ml/day with the mean of 127.61±15.33. The difference b/w two groups were highly significant. However result shows that only VLDL and TG were significantly higher in cases when compared to controls, and other parameters shows non- significant result.

DISCUSSION

Alcohol is major psychoactive drug used worldwide approx 13% of adult had alcohol dependence at some point in their life. Symptoms of the effects of ethyl alcohol may be due to uncomplicated physiological changes or complications, which have resulted from habitual intake or addition of alcohol as in alcoholics. Alcohol is eliminated through metabolism. More than 90% of alcohol is removed from the body by oxidation of ethyl alcohol to carbon-di-oxide and water. This process primarily occurs in liver. There are many factors which are responsible for hepatic damage due to alcoholism s/a free radicals, hypoxia, inflammatory agents and adduct formation.

This study was focused on lipid profile assessment in subjects 'at risk drinking' of alcohol for Prolonged duration and analyzed in reference to normal parameters[12].

Alcohol consumption stimulates hepatic secretion of VLDL, possibly by inhibiting the hepatic oxidation of free fatty acids which then promote TG synthesis and VLDL secretion. The usual lipoprotein pattern seen in alcohol consumption is type IV i.e. increased VLDL and may also have hypertriglyceridemia.

Our results are consistent with Vaswani M et al. who observed that TC, VLDL-C, TG were higher in alcohol dependents as compared to non- dependent subjects. Our study result is in contrast with Vaswani M et al. in that, LDL-C was significantly increased in alcoholics and there was no significant difference in HDL-C levels between the two groups (Vaswani M et al. found increased HDL-C in alcoholics and no difference in LDL-C between the two groups)[13].

Similar study by Whitfield JB et al. [14] showed that the TG levels increases with increased alcohol intake. However, studies by Riuidavets JB et al.[15] and Hans Hoffmeister et al.[16] found that blood levels of HDL-C increased with increased alcohol intake. A study conducted by Barboriak JJ et al.[17] showed significantly higher HDL-C levels in the alcoholic group. They also noticed that, after 2weeks of alcohol abstinence, the HDL-C levels decreased to normal range. Choudhary Sohel R et al. [18] found higher Serum TG and HDL-C and lower LDL-C in heavy alcohol drinkers.

This finding is in contrast with the previous study done by Oduola T et al.[19] in which there were no association between alcohol intakes with total cholesterol levels. Similar observations were also found in a study conducted by Marita Passilta et al.[20] in which there was no difference in LDL-C concentrations between controls and in those with highest alcohol intake. In a similar study by Seppa K et al. [21] it was found that the HDL-C/TC ratio between controls and heavy drinkers was unaltered.

CONCLUSION

In conclusion significantly altered lipid profile is found in chronic alcoholics as compared to controls. There are several possible causes of bias in present study. Lipids are also influenced by diet, physical activity which was not surveyed in the present study. Present study is a male dominant, further studies with large sample size, including both the sexes and type of alcohol beverages is required to establish lipid profile as a marker of alcoholism.

REFERENCES:

1. Louis W Sullivan, Victor Herbert. 1964. Suppression of hematopoesis by ethanol. J Clin Invest. 43(11):2048-62.
2. Halsted CH. Nutrition and alcoholic liver disease. Semin Liver Dis. 2004; 24(3):289–304.
3. Hopkein, D.N. & Williams, R.R. (1981) A survey of suggested coronary risk factors. Atherosclerosis. 40, 1-52.
4. Brunzell JD, Chait A, Bierman EL (1978). Pathophysiology of lipoprotein transport. Metabolism 27, 1109-1127.
5. Gau GT, Wright RS (2006). Pathophysiology, diagnosis and management of dyslipidemia. Curr Probl Cardiol 31, 445-486.
6. Ahmed SM, Clasen ME, Donnelly JE (1998). Management of dyslipidemia in adults. Am Fam Physician 57, 9, 20192-2204, 2207-2208.
7. Day CP, Yeaman SJ (1994). The biochemistry of alcohol induced fatty liver. Biochim Biophys Acta 1215, 33-48.
8. Purohit V, Gao B, Song B Molecular mechanisms of alcoholic fatty liver. Alcohol Clin Exp Res.
9. Reuben A (2006). Alcohol and the liver. Curr Opin Gasastroenterol 22, 263-271.
10. Durrington PN (1990). Secondary hyperlipidemia. Br Med Bull 46,4, 1005-1024.
11. Friedwald WT, Levy RS, Friedrickson DS. Estimation of concentration of low density lipoprotein cholesterol in plasma without rise of preparative ultracentrifuge. Clin chem 1972:18:499-502.
12. E.B. Rimm, E.L. Giovannucci, W.C. Willett, et al. Prospective study of alcohol

consumption and risk of coronary disease in men. Lancet 1991; 338:464-8.
13. Vaswani M, Rao RV. 2005. Biochemical measures in the diagnosis of alcohol dependence using discriminant analysis. Indian J Med Sci. 59:423-30.
14. Whitfield JB, Allen JK, Adena M et al. 1981. A multivariate assessment of alcohol consumption. Int J Epidemiol 10(3):281-8.
15. Ruidavets JB, Ducimetiere P, Arveiler D et al. 2002. Types of alcoholic beverages and blood lipids in a French population. J Epidemiol Community Health. 56:24-8.
16. Hans Hoffmeister, Frank Peter Schelp, Gert BM Mensink et al. 1999. The relationship between alcohol consumption, health indicators and mortality in the German population. Int J Epidemiol. 28:1066-72.
17. Barboriak JJ, Jacobson GR, Cushman P et al. 1980. Chronic alcohol abuse and high density lipoprotein cholesterol. Alcohol Clin Exp Res 4(4):346-9.
18. Choudhary Sohel R, Hirotsugu Ueshima, Yoshikuni Kita et al. 1994. Alcohol intake and serum lipids in a Japanese population. Int J Epidemiol. 23(5):940-7.
19. Oduola T, Adeosun OG, Oduola TA et al. 2005. Drinking patterns: biochemical and hematological findings in alcohol consumers in Ile-Ife, Nigeria. Afr J Biotechnol. 4(11):1304-8.
20. Marita Paassilta, Kari Kervinen, Asko O Rantala et al. 1998. Social alcohol consumption and low lipoprotein concentrations in middle aged Finnish men: population based study. BMJ. 316:594.
21. Seppa K, Sillanaukee P, Pitkajarvi T et al. 1992. Moderate and heavy alcohol consumption has no favorable effect on lipid values. Arch Intern Med. 152(2):297-300.