Original Article

Comparative Study of Serum LDL And HDL In Alcoholics & Non-Alcoholics

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Background : Alcohol abuse is one of the major form of addiction and a major threat to public health in developed as well as developing countries. Alcohol intake is increased in quantity and frequency over last few decades. Alcohol consumption pre-disposes subjects to changes in serum Low Density Lipoprotein (LDL) and High Density Lipoprotein (HDL) which are associated with cardiovascular risk.

Methods : 100 alcoholics were compared with 100 non-alcoholics. Alcohol drinking history was assessed by interview and questionnaire and we measured serum LDL and HDL level.

Results : There were significant rise in HDL and LDL in chronic alcoholics when compared with non alcoholics with p value <0.00001, 0.019567 respectively. When serum LDL and HDL were compared among moderate and heavy drinkers, we found increased level of LDL with p value <0.00001 in heavy drinkers. There is decrease in HDL cholesterol level among heavy drinkers when compared to moderate drinkers with p value 0.000016, which is significant. But remains elevated compared to non alcoholics.

Conclusion : This study shows that alcohol intake increases the levels of LDL and HDL. Moderate alcohol intake increases HDL cholesterol whereas heavy alcohol consumption increases LDL and decreases HDL.

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Key words **: Alcohol abuse, addiction, cardiovascular risk, heavy drinkers**

Alcohol abuse is one of the major form of addiction seen in developed as well as developing countries. India too carries a significant burden of this. Multiple reasons like financial burden of being low socio economic status, heavy field work leading to physical stress and mental stress. According to World Health Organization (WHO) reports of 2014, globally alcoholism alone causes 5.9% deaths every year and the burden of the disease accounting to 5.1%[7]. In an alarming revelation, the Global Status report on alcohol and health 2014, released by WHO states that the amount of alcohol consumption has raised in India between the periods of 2008 to 2012. In addition to the average volume of alcohol consumption, the patterns of drinking markedly contribute to the associated burden of disease and injury[1,2]. There are different ways by which alcohol alter lipid metabolism. The enhancement of postalimentary lipemia by ethanol has been thought to be due to delay in the rates of gastric emptying and/or fat absorption[3,4]. Alcohol may increase hepatic fatty acid uptake by increasing hepatic blood flow[3]. In alcoholics, the metabolism of alcohol produces increased amounts of reduced hepatic Nicotinamide Adenine Nucleotide (NADH+). Increased NADH2/NAD ratio inhibits the oxidation of fatty acids[3]. Chronic ethanol intake has been shown to reduce the activity of carnitine palmitoyltransferase-1 (CPT-1), which may impair the transport of fatty acids into mitochondria that in turn may result in reduced fatty acid oxidation[3]. Recently researchers have demonstrated that chronic ethanol administration can significantly increase the production of hepatic sterol regulatory element-binding protein (SREBP-1), which is associated with increased expression of lipogenic genes as well as accumulation of triglycerides in the liver[3]. Several animal studies suggest that alcohol intake can increase esterification of free fatty acids into triglycerides. This is primarily due to ethanol-induced up-regulation of Phosphatidate Phosphohydrolase (PAP), the rate limiting enzyme in triglyceride synthesis[3].

So we have come to know that consumption of alcohol in large amounts for a long duration impairs lipid metabolism, thus alters serum LDL and HDL, hence pre-disposes to cardiovascular disease. The aim of the study is to compare the serum LDL and HDL

Editor's Comment :

- Cessation of alcohol intake may significantly reduce the risk of development of cardiovascular disease.

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among Alcoholics (cases) and Non-alcoholics (controls).

Aims and Objectives

Estimation ofserum LDL, serum HDL in Alcoholics (cases) and Non-alcoholics (controls) and its comparison between cases and controls.

Material and Methods

The present study is a comparative case-control study conducted in the Department of General Medicine, Silchar Medical College and Hospital, Silchar, Assam after getting permission from local ethical committee.The study period was from June 2018 to May 2019 for a period of 1year. A total of 200 patients from the OPD and IPD of the Department of General Medicine were included randomly for the study, after fulfilment of inclusion and exclusion criteria. Among them 100 were male alcoholics (of age 30-50 years consuming alcohol more than 8 years) and 100 males who did not consume alcohol were included in the study. Among alcoholics some were heavy drinkers (≥4 drinks per week) and some were moderate drinkers (2-3 drinks per week). Patients with diabetes mellitus, hypertension, renal disease, liver disease other than Alcoholic liver disease, Pancreatitis, Malnutrition, Family history of Hyperlipidemia were excluded from the study. Subjects who are on drugs affecting lipid metabolism also excluded. Individuals belong to other age groups, females and who did not give the consent were excluded from the study.

Sample : About 5ml of fasting blood samples (overnight fast for 8-12 hours) were drawn from the median cubital vein on the anterior forearm (the side within the fold of the elbow) into clot activator tubes (to aid clotting and separate serum). The clotted blood was centrifuged at 2000 revolutions per minute (rpm) for 5 minutes to separate the serum from the deposit. The serum was used to estimate HDL and LDL.The parameters were measured on Beckman Coulter AU analyzer and estimated by the following methods:

(1) High Density Lipoprotein: The method was direct homogenous test. This method has two steps. In the first step, chylomicrons, Very Low Density Lipoprotein (VLDL) and LDL cholesterol are specifically eliminated and destroyed by enzymatic reactions. In the second step, the HDL fraction is determined by well established specific enzymatic reactions in the presence of surfactants for the HDL.

(2) Low density lipoprotein : Estimation of LDL was done by Friedwald's equation {LDL = Total cholesterol (TC) – HDL – [Triglyceride (TG)/5]} and method was direct homogeneous test.

Statistical analysis : Statistical analysis was performed using Microsoft excel program. Pearson's correlation co-efficient was used to examine the association between various continuous parameters. Independent samples t-test was used to compare means of different variables. Data were presented as Mean±Standard Deviation (SD). The results were considered statistically significant when the two tailed p value was < 0.05.

Results

The results and observations are incorporated in the following tables and diagrams and also discussed below (Tables 1&2 and Figs 1-4).

Discussion

In our study, HDL is raised in cases with mean value 43.12±7.06 mg/dl when compared to controls which is 37.49±7.04 mg/dl. P value is 0.004, which is significant. LDL is raised in cases with mean value of 105.74±35.70 mg/dl when compared to controls which is 97.90±12.29 mg/dl. P value is 0.019567, which is significant.This result is consistent with the study conducted by Wiilliam p *et al*[5], which showed alcohol consumption is positively associated with HDL cholesterol. This study is also consistent with the study done by Vaswani M *et al*,[6] in which HDL, LDL

Table 1 — Comparison of Lipid profile in cases and controls

Lipid profile	Controls	Cases	P value
HDL (mg/dl)	37.49±7.04	43.12±7.06	< .00001
LDL (mg/dl)	97.9±12.29	105.74±35.70	.019567

Table 2 — *Comparison of serum LDL and HDL in cases on the basis of quantity of alcohol intake*

Lipid profile	Type of alcoholic		P value
	Moderate drinker	Heavy drinker	
HDL (mg/dl)	46.5±8.85	40.71±4.21	0.000016
LDL (mg/dl)	86±26.17	118.91±35.56	<0.00001

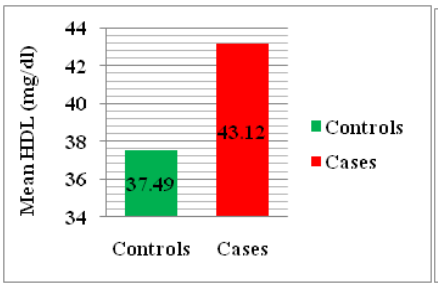

Fig 1

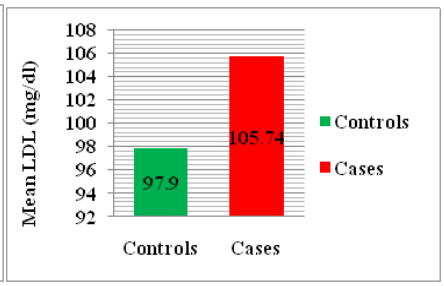

Fig 2

Fig 1 & 2 — Comparison of serum HDL and LDL in cases and controls

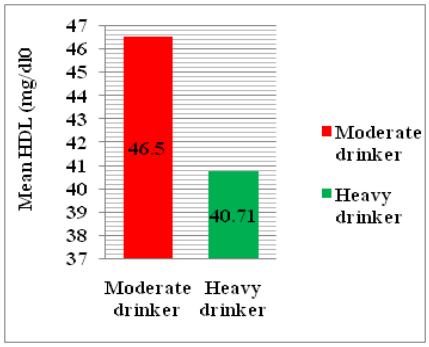

Fig 3

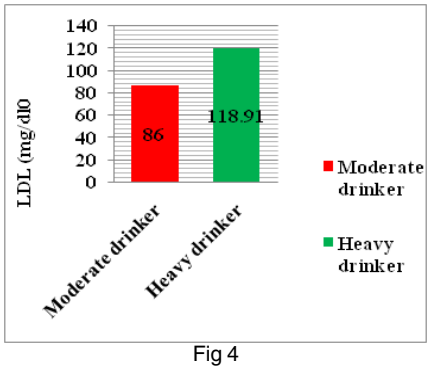

Fig 4

Fig 3 & 4 — Comparison of HDL and LDL in cases on the basis of quantity of alcohol intake

were significantly higher in alcoholics when compared to non alcoholics (p< 0.001). Data showed that HDL cholesterol is low in heavy drinkers with a mean value of 40.71±4.21 mg/dl when compared to moderate drinkers, which is 46.50±8.85 mg/dl. There is a significant change in HDL cholesterol with p value 0.000016. There is raise in LDL cholesterol by mean value 118.91±35.56 mg/dl in heavy drinkers in comparison to moderate drinkers which is 86.26±26.17 mg/dl. Significant change is found with p value <0.00001. This result is similar to the study done by Arun Lakshmipathy *et al*[7] who found that there is an increase in HDL levels when alcohol is consumed in moderation, but there may be a decline in HDL when alcohol is consumed in excessive quantity.There is an another study with similar finding conducted by Sheethal K C *et al*,[8] in which the mean value of HDL, LDLin heavy drinkers were 40.44±4.03, 123.44±38.44 and in moderate drinkers were 47.16±9.32, 80.42±26.31 respectively with p value <0.05.

Conclusion

It is concluded from the present study that there is significant correlation between alcohol intake and serum HDL and LDL levels. Serum HDL has also a significant correlation with drinking pattern whether heavy drinkers or moderate drinkers. It is reasonable to conclude that chronic heavy alcohol intake may be a factor in development and progression of cardiovascular disease by raising LDL and lowering HDL.

Limitation : The study was hospital based study with small sample size of 100 cases and 100 controls, conducted over a limited period of 1 year. So to gather detail information regarding correlation between alcohol intake and serum lipid profile, it needs broader study covering large no population over a longer period of time.

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