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Original Article
Effects of chronic alcohol consumption on lipid levels in general population

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Abstract
Introduction: *Associations between alcohol consumption and the risk of cardiovascular disease, including myocardial infarction and coronary heart disease, are mediated, in large part, by the differential influence on the lipid levels. This study aimed to compare the lipid levels among alcohol consumers and non-consumers.*

Methodology: *An observational study was designed to study the lipid levels among alcohol consumers and compare them against non-consumers. Persons taking approximately 26 ml of alcohol per day, for at least three to five days per week for at least five years were labelled as alcohol consuming participants. Subjects who had never consumed alcohol in life were labelled as non-alcohol consuming participants. Lipid levels of alcohol consuming and non-consuming participants were compared using independent t test in SPSS software.*

Results: *A total of 100 alcohol consuming (Group I) and 50 non-alcohol consuming (Group II) participants were included in the study. HDL cholesterol was similar in alcohol consuming as well as non-alcohol consuming participants. LDL cholesterol was found to be significantly higher among alcohol consuming participants (139.43 ± 35.03 vs 128.62 ± 30.43 mg/dl, p value < 0.05). Total cholesterol was also found to be significantly higher among alcohol consuming participants (193.41 ± 23.54 vs 185.48 ± 26.30 mg/dl, p value < 0.05). Serum triglycerides were found to be higher among alcohol consuming participants as compared to alcohol non-consuming participants, however the difference was not statistically significant.*

Conclusions: *The present study showed that heavy alcohol consumption is associated with significantly higher levels of serum triglycerides, total cholesterol, low density lipoprotein and decreased level of serum high density lipoproteins.*

Keywords: *alcohol, lipid, cardiovascular risk.*

Introduction
Alcohol intake, when chronic or excessive, can lead to a variety of adverse effects including liver disease, heart failure, increased cancer risk, neurologic complications, and unintentional injuries. In addition, even moderate alcohol

consumption should be avoided whenever consumption would put individuals at risk (like during pregnancy or before driving a vehicle). Association between alcohol consumption and the development of coronary heart disease is complex and not fully understood.[1] Some of the health benefits may be mediated by genetic factors. Interestingly, epidemiological data from the general population have shown that the effect of alcohol on many diseases has a biphasic pattern, depending on the amount of alcohol consumed, such that the incidence of coronary heart disease is known to be lowered by moderate alcohol consumption and increased by high alcohol consumption.[2] Observational studies support that light to moderate alcohol consumption is associated with lower risk of myocardial infarction and cardiovascular death. In contrast, heavier alcohol consumption is associated with no change or even an increase in coronary risk.[3] Additionally, the associations between alcohol consumption and the risk of cardiovascular disease, including myocardial infarction and coronary heart disease, are mediated, in large part, by the differential influence on the levels of high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C).[4] Consequently, numerous investigators have studied the effect of alcohol consumption on blood lipid concentrations within specific population groups. However, the results of many of these studies have been inconsistent because of the differences in age and gender structure as well as the variations in ethnicity or comorbidities among the study participants. This study aimed to compare the lipid levels among alcohol consumers and non-consumers.

Methodology

An observational study was designed to study the lipid levels among alcohol consumers and compare them against non-alcohol consumers. Asymptomatic middle aged men, aged 30 to 65 years of age, presenting to our hospital with their spouses or children for treatment of the latter's illness were asked history of alcohol intake. If the person revealed that he drank alcohol more than once a week, he was asked details about alcohol consumption. Persons taking approximately 26 ml of alcohol per day, for at least three to five days per week for at least five years were labelled as alcohol consuming participants. Subjects who had never consumed alcohol in life were labelled as non-alcohol consuming participants. For this study, 100 alcohol consumers and 50 alcohol non-consumers were included in the study. We excluded participants who gave a history of smoking, diabetes mellitus, nephrotic syndrome, hypertension, thyrotoxicosis and coronary artery disease. Participants with medication history of statins, fibric acid derivatives, nicotinic acid, beta blockers and diuretics were excluded from the study and those on any dietary restriction were excluded from the study. Based on their drinking history and screener questions about their medical history, 100 alcohol consumers and 50 alcohol non-consumers were included in the study. They were explained the purpose of the study and written informed consent was taken from them.

Using a pre-tested semi-structured questionnaire, socio-demographic profile of the participants was ascertained. These included questions on age, education, income, exercise and obesity. Body mass index (BMI) was then calculated as weight (kg) divided by the square of height (m^2).Blood samples were collected by venipuncture after 10 to 12 hours of fasting. Total cholesterol, HDL-C and triglycerides were measured by enzymatic methods with commercially available kits. Non-HDL-C was calculated as total cholesterol minus HDL-C. All blood analyses were carried out within 2 hours of blood sampling by a laboratory within the premises of the our hospital. We calculated the frequency and percentage or mean and standard deviation where appropriate for demographic characteristics to describe the alcohol consuming and non-consuming sample. Similarly, lipid levels were also described as means and standard deviation. Lipid levels of alcohol consuming and non-consuming

participants were compared using independent t test in SPSS software. Statistical significance was denoted by p value less than 0.05.

Results

In the present study, a total of 100 alcohol consuming (Group I) and 50 non-alcohol consuming (Group II) participants were included in the study. Table 1 describes the socio-demographic of the participants included in the study. Mean age of group I participants was 54.4 ± 11.8 years and that of Group II participants was 55.3 ± 14.6 years (Table 1). BMI was found to be 25.3 kg/m^{2}and 23.6 kg/m^{2}in participants of Group I and Group II respectively. Further, 27% of Group I and 42% of Group II participants did regular exercise. Table 2 compares the lipid levels among the participants. HDL cholesterol was similar in alcohol consuming as well as non-alcohol consuming participants (p value >0.05).

LDL cholesterol was found to be significantly higher among alcohol consuming participants (139.43 ± 35.03 vs 128.62 ± 30.43 mg/dl, p value < 0.05). Total cholesterol was also found to be significantly higher among alcohol consuming participants as compared to alcohol non-consuming participants (193.41 ± 23.54 vs 185.48 ± 26.30 mg/dl, p value < 0.05). Serum triglycerides were found to be higher among alcohol consuming participants (138.19 ± 68.22 mg/dl) as compared to alcohol non-consuming participants (125.98 ± 28.74 mg/dl), however the difference was not statistically significant. Apolipoprotein A1 and B were found to be significantly higher among alcohol consuming participants as compared to alcoholic non-consumers (p value < 0.05). Lipoprotein a levels were higher among alcohol consumers, but the difference was not statistically different.

Table 1. Baseline characteristics of participants included in the study

Variables	Group I Alcohol consumers (n=100)	Group II Alcohol non-consumers (n=50)
Mean age (SD)	54.4 ± 11.8	55.3 ± 14.6
BMI, kg/m^2 (SD)	25.3 ± 3.5	23.6 ± 4.1
Education (%)		
Till primary school	45 (45%)	26 (52%)
Till high school	22 (22%)	15 (30%)
Higher than high school	33 (33%)	9 (18%)
Monthly income, INR (SD)	14532.5 ± 3025.4	15804.4 ± 2743.7
Regular exercise (%)	27 (27%)	21 (42%)

Table 2. Comparison of lipid levels among alcohol consuming and non-alcohol consuming participants

Variables	Group I Alcohol consumers (n=100)	Group II Alcoholic non-consumers (n=50)	p value
HDL- cholesterol (mg/dl)	40.01 ± 7.37	40.18 ± 3.73	> 0.05
LDL-cholesterol (mg/dl)	139.43 ± 35.03	128.62 ± 30.43	< 0.05
Total cholesterol (mg/dl)	193.41 ± 23.54	185.48 ± 26.30	< 0.05
Triglycerides (mg/dl)	138.19 ± 68.22	125.98 ± 28.74	> 0.05
Apolipoprotein A1 (mg/dl)	152.32 ± 33.92	143.65 ± 31.85	< 0.05
Apolipoprotein B (mg/dl)	127.41 ± 18	114.26 ± 26	< 0.05
Lipoprotein a (mg/dl)	21.3 ± 8.4	18.7 ± 6.4	>0.05

Discussion

Studies conducted in general population have reported that heavy alcohol consumption was associated with higher HDL-C levels and lower non-HDL-C levels.[5] However, the relationship between alcohol consumption and triglyceride levels in the general population are not consistent among different ethnic groups. In many

epidemiological studies performed in Europeans[6] and the Japanese[7], light alcohol drinking was found to be associated with a decrease in blood triglyceride level and heavy drinking associated with an increase. On the other hand, some results from Turkish population, showed a linear increase in triglycerides with an increase in alcohol intake. There are many epidemiological studies on the relationship between alcohol consumption and the risk of dyslipidaemia in various general populations. Moreover, a Greek study showed that alcohol drinking was associated with lower risk of low HDL-C.[8] However, an American study showed no significant relationship between alcohol consumption and the risk of low HDL-C level or the risk of high triglycerides.[9] Difference in age structure and ethnicities might explain such discrepancies, because genetic factors are known to play an important role in lipid metabolism.[10] Another important confounder may be the existence of concurrent diseases such as hypertension, which can be worsened by chronic drinking.

Alcohol after ingestion makes two metabolites, acetaldehyde and acetate, which are found in the plasma compartment. In normolipidemic subjects, an acute alcohol dose induces a mild lipemia (50% increase in plasma triglycerides).[11] And there is a dramatic decrease in plasma non-esterified fatty acid (NEFA) levels concurrently. It has been proposed that it is due to inhibition of intracellular lipolysis in peripheral tissue, mainly fat, an effect that has been observed in vitro experiments as well.[12] The increase in plasma triglycerides after alcohol consumption has been thought to be due to enhanced hepatic lipogenesis. However, alcohol only mildly activates de novo lipogenesis; most of the acetate formed from alcohol is released into the plasma compartment and delivered to peripheral tissue sites, where it is oxidized.[13] Thus, the decrease in plasma NEFA after alcohol consumption is likely due to an increase in plasma acetate on lipolysis within adipocytes. Consumption of alcohol enhances postprandial lipemia and produces several other changes in plasma lipid levels and lipoprotein composition. van Tol et al[14] and Hendriks et al[15] conducted a series of studies in which changes in plasma lipids were followed during and after the consumption of alcohol. In addition to enhancement of postprandial lipemia, substitution of alcohol for water increased the rate of cholesteryl ester transfer protein (CETP)-mediated exchange of the CE of HDL for the TG of apolipoprotein B-containing lipoproteins. The plasma concentrations of HDL cholesterol, and apolipoproteins A-I, A-II, and B were not changed during the postprandial period. However, the CE content of HDL was reduced.

Conclusion

The present study showed that heavy alcohol consumption is associated with significantly higher levels of serum triglycerides, total cholesterol, low density lipoprotein and decreased level of serum high density lipoproteins A high HDL, low cholesterol and low triglycerides are often considered to be desirable features of an individual's lipid profile. However, the present study cannot establish a causal evidence, and therefore further studies are needed to study the effect of alcohol consumption on lipid profile.

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