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Original Research Article

Lipid profile and alcoholism

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ABSTRACT

Background: Alcoholism is a major threat to public health in both developed and developing countries. Alcohol has many effects on lipid profile, including inducing de novo fatty acid synthesis and inhibiting fatty acid oxidation in liver. Aim of this study was to study the effect of alcohol consumption on serum lipid profile in alcoholics and to compare with those of non-alcoholic controls.
Methods: 100 cases and 100 age and sex matched controls were selected. These subjects were classified into, 1. Moderate alcoholics: Consuming ≤210 g per week 2. Heavy alcoholics: Consuming >210 g per week. Blood was taken in fasting state and lipids were estimated.
Results: Among 200 subjects, 94% were male. 58.5% of the male and 83.3% of the female were heavy drinkers. Maximum number of alcoholics were seen in the age group of 41-50 years. The height correlated well with both control and study group. The moderate dose alcoholics had a higher weight when compared to control group and heavy dose alcoholics. The heavy dose alcoholics had a lower BMI when compared with the control group and moderate dose alcoholics. Heavy dose alcohol consumption was associated with a higher increase in triglyceride level. The total cholesterol level was significantly higher in the heavy dose alcoholics when compared with the control group. The HDL level was highest in the moderate dose alcoholics. The LDL level was highest in heavy dose alcoholics whereas, the moderate dose alcoholics had a lesser LDL level when compared with the control group. The VLDL level steadily increased with alcohol consumption.
Conclusions: Patients with heavy alcohol consumption had significant increase in total cholesterol, triglycerides, LDL and VLDL. The moderate alcohol consumers had significantly increased HDL and decreased LDL. These protective effects declined after heavy alcohol consumption.

Keywords: Alcohol, Heavy alcoholics, Lipid profile, Moderate alcoholics

INTRODUCTION

Alcohol (beverage ethanol) is a chemical substance which distributes itself throughout the body, affecting almost all systems and altering nearly every neurochemical process in the brain. This drug is likely to exacerbate most medical conditions, affect almost any medication metabolized in the liver, and temporarily mimic many medical (Eg: diabetes) and psychiatric (Eg: depression) conditions.[1] Alcoholism is defined as a chronic and progressive disease characterised by loss of control over the use of alcohol with subsequent social, legal, psychological and physical consequences.

Globally, there are 2 billion alcohol consumers with 2.3 million cases of alcoholism and 1.8 million deaths every year. Alcoholism is a major threat to public health in both developed and developing countries. It has been estimated that 5% of adult males in India are alcoholics with dependence symptoms. Over the last 30-40 years alcohol consumption has increased in quantity and frequency. Today alcohol abuse has become a worldwide social and medical problem. With the rapid socioeconomic and cultural changes, the alcohol is viewed as a symbol of social status and prestige as projected by their role models. The age at which people start using alcohol has also declined.[2] Alcohol has many effects on lipid metabolism, including inducing de novo fatty acid synthesis and inhibiting fatty acid oxidation in the liver. The most common effect of alcohol is to increase plasma triglyceride levels. Alcohol consumption stimulates hepatic secretion of VLDL, possibly by inhibiting the hepatic oxidation of free fatty acids, which then promote hepatic triglyceride synthesis and VLDL secretion.

The usual lipoprotein pattern seen with alcohol consumption is Type IV (increased VLDLs), but persons with an underlying primary lipid disorder may develop severe hypertriglyceridemia (Type V) if they drink alcohol. Regular alcohol use also raises plasma levels of HDL.[3,4] While low doses of alcohol have some healthful benefits, the intake of more than three standard drinks per day on a regular basis enhances the risk for cancer and vascular disease, and alcohol use disorders decrease the life span by about 10 years.[1] Recognition of an association between alcohol and coronary disease, has led to an increasing interest in alcohol induced changes in lipids and lipoproteins. Population studies have revealed a positive correlation between alcohol intake and plasma high density lipoprotein and triglycerides.[5,6] A leading protective mechanism has been suggested by alcohol's ability to produce changes in plasma lipoproteins especially HDL and LDL.

Increase in plasma level of HDL is often associated with decrease in the prevalence of coronary artery disease. It is believed by several group of investigations based on the epidemiological studies that daily ingestion of low dose of alcohol may enhance the level of HDL. Such findings have generated a great deal of interest in the probably protective effect of alcohol against the atherosclerotic disease. The coronary heart disease has been reported to decrease with moderate drinking in both sexes, but such benefit disappears with high consumption.[7,8]

In light to moderate drinkers, the inverse association between alcohol consumption and death from coronary heart disease can be explained, in large part, by the HDL cholesterol level, which increases with alcohol consumption.[9] Regular consumption of small to moderate amounts of alcoholic beverages, regardless of the type, reduces the risk of Myocardial Infarction (MI), and further suggests that there is benefit, in large part, from increases in HDL levels.[10] Heavy alcohol consumption for prolonged periods result in marked perturbation of the lipid transport system, reflecting both effects of alcohol on lipid metabolism in hepatic and extra hepatic tissue, as well as its marked toxic effects on liver function.[11]

Hence, this present study was undertaken to determine the correlation between alcohol consumption and changes in lipid profile among patients attending the General Medicine Department in K.V.G Medical College Hospital, Sullia. Also, such a study was not attempted here before.

METHODS

Source of data

Both cases and controls were selected among patients attending General Medicine Department of K.V.G Medical College Hospital from November 2017 to November 2018. The study subjects were selected randomly. These subjects were classified on the basis of self-reported drinking records into two groups:

- Moderate to borderline alcoholics: Consuming less than or equal to 210 g per week.
- Heavy alcoholics: Consuming more than 210 g per week.

The Study type was Case control study and Duration of Study was Twelve months (November 2017 – November 2018). The Sample size was taken:

- 100 cases and similar number of age and sex matched controls satisfying the inclusion and exclusion criteria given below were selected by purposive sampling.
- Detailed history was taken, clinical examination was performed as per proforma and routine investigations were done

In both cases and controls blood for lipids was taken in fasting state and lipid estimation was done in clinical laboratory at K.V.G Medical College Hospital, Sullia-D.K.

Inclusion criteria

- Patients coming with history of alcoholism.
- Age and sex matched controls.

Exclusion criteria

- Patients and controls with intake of drugs like thiazides, beta blockers and steroids.
- Patients and controls with:

  a. Diabetes mellitus
  b. Chronic renal failure
  c. Cirrhosis of liver

d. Acute infections
e. Nephrotic syndrome
f. Patients taking hypolipidemic drugs (including ocps).

Serum lipid estimation

Each sample of serum was analysed for Total Cholesterol, Triglycerides and HDL.

- Serum Total Cholesterol level was measured by using cholesterol peroxidase methodology.
- Serum Triglyceride level was measured by using GPO-Pap method.
- Serum HDL level was measured by precipitation method using phosphotungstate magnesium reagent.

The serum LDL and VLDL level was estimated by using Friedewald formulae.

VLDL =Triglycerides/5
LDL =Total cholesterol-(HDL+VLDL)

Statistical analysis

The data was analysed on IBM SPSS version 19.
The age and sex were analysed using Chi-square test.
The height, weight and BMI were analysed using ANOVA test.
The lipid profiles (Total cholesterol, Triglycerides, LDL, HDL, VLDL) of cases and controls were analysed using student's t - test.

RESULTS

A total of 200 subjects were included who comprised of 100 alcoholics and 100 age and sex-matched nonalcoholic controls who attended the General Medicine department during the study period.

Table 1: Sex-distribution of study subjects and comparison of alcohol consumption in males and females.

Sex	Control (N = 100)	Alcoholics			p- value
		Moderate dose (n=40)	Heavy dose (n=60)	Total	
Male	94 (94.0%)	39 (97.5%)	55 (91.7%)	188	0.581 NS
Female	6 (6.0%)	1 (2.5%)	5 (8.3%)	12	

NS - Not Significant

Table 2: Comparison of age

Age group	Control (N = 100)	Moderate dose (N = 40)	Heavy dose (N = 60)	p -value
21 - 30	11 (11.0%)	5 (12.5%)	6 (10.0%)	0.828 NS
31 - 40	32 (32.0%)	10 (25.0%)	22 (36.7%)	
41 - 50	45 (45.0%)	18 (45.0%)	27 (45.0%)	
51 - 60	12 (12.0%)	7 (17.5%)	5 (8.3%)	

NS - Not Significant

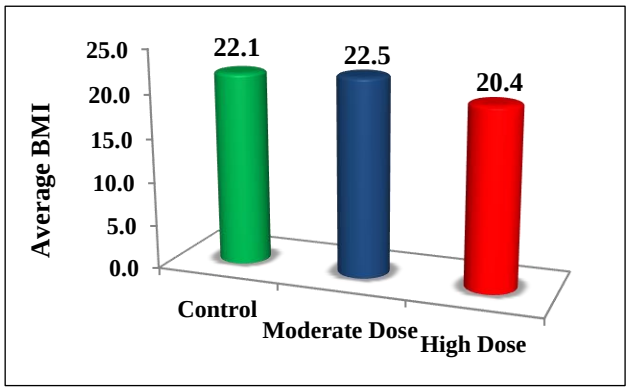

Figure 1: Comparison of BMI.

Among the total 200 study subjects, 94% were male and 6% were female. 58.5% (55) of the male and 83.3% (5) of the female were heavy drinkers. (Table 1).

Maximum number of alcoholics were seen in the age group of 41-50 years (45%) followed by 31-40 years (32%) (Table 2).

The height correlated well with the control group and study group with no significant difference. There was a significant difference between the weights of control group and study group. The moderate dose alcoholics had a higher weight when compared with the control group and heavy dose alcoholics.

The heavy dose alcoholics had a lower body mass index (20.4) when compared with the control group (22.1) and moderate dose alcoholics (22.5). The BMI of heavy dose alcoholics was significantly lower when compared with the control group, while that of moderate dose alcoholics was at par with the control group. (Table 3) (Figure 1).

Table 3: Comparison of height, weight and BMI.

Variables	Control (N = 100)	Moderate dose (N = 40)	Heavy dose (N = 60)	p - value
Height	1.64±0.05	1.64±0.06	1.64±0.05	0.843 NS
Weight	59.1±4.36	61.0±7.16	54.9±4.53	0.000**
BMI	22.1±2.02	22.5±1.64	20.4±1.57	0.000**

NS - Not Significant, ** Significant at 0.01 level

Table 4: Comparison of lipid profile (moderate dose vs. heavy dose).

Parameters	Moderate dose (N = 40)	Heavy dose (N = 60)	p - value
TG	150.9±21.92	211.3±29.80	0.000**
TC	199.3±15.16	232.2±20.83	0.000**
HDL	51.2±10.35	43.9±3.75	0.000**
LDL	117.9±19.24	146.1±22.79	0.000**
VLDL	30.2±4.38	42.3±5.96	0.000**

** Significant at 0.01 level.

There was a significant increase in triglyceride level with alcohol consumption. Heavy dose consumption of alcohol was associated with a higher increase in triglyceride level. (Table 4, Figure 2).

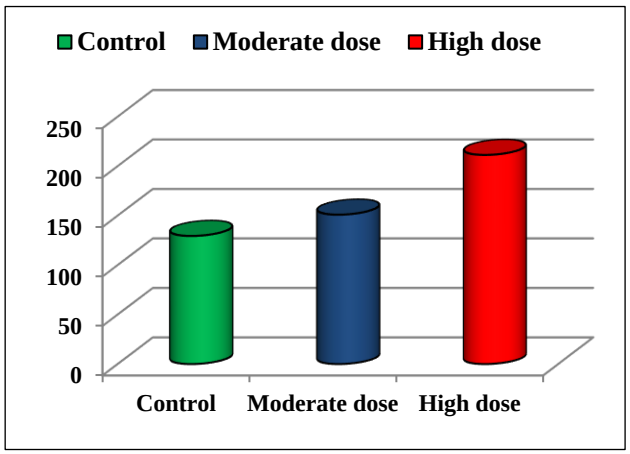

Figure 2: Comparison of triglyceride level.

The total cholesterol level was significantly higher in the heavy dose alcoholics when compared with the control group. The moderate dose alcoholics did not have much significant difference in the total cholesterol level when compared with the control group. (Table 4, Figure 3).

The HDL level was highest in the moderate dose alcoholics. The heavy dose alcoholics did not have much significant difference in HDL level when compared with the control group (Table 4, Figure 4).

Among the 3 study groups, the LDL level was highest in the heavy dose alcohol consumers suggesting the detrimental effect of heavy dose alcohol on health.

Whereas, the moderate dose alcohol consumers had a lesser LDL level when compared with the control group, demonstrating the beneficial effect of moderate dose alcohol consumption. (Table 4, Figure 5) The VLDL level steadily increased with alcohol consumption when compared with the control group. (Table 4, Figure 6).

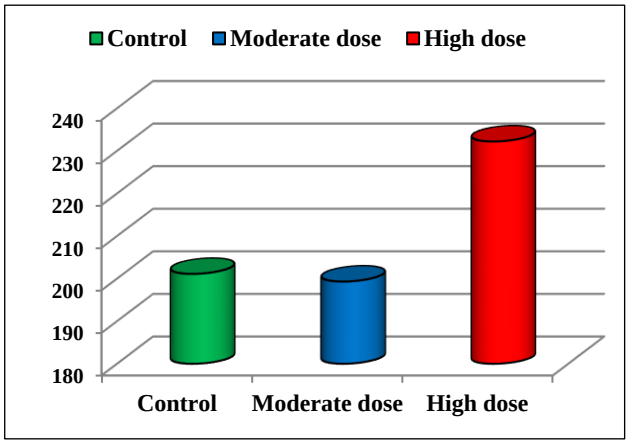

Figure 3: Comparison of total cholesterol level.

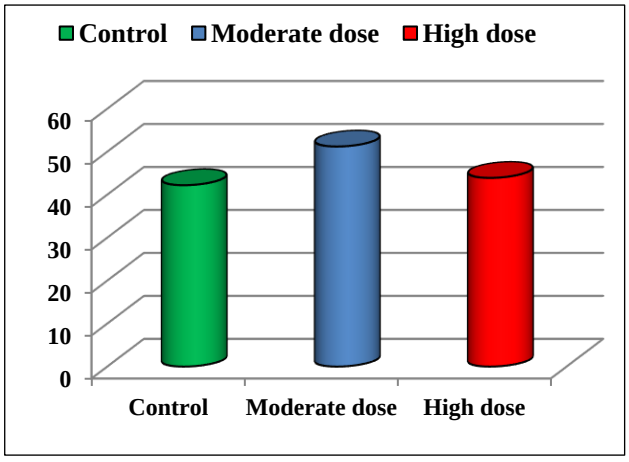

Figure 4: Comparison of HDL level.

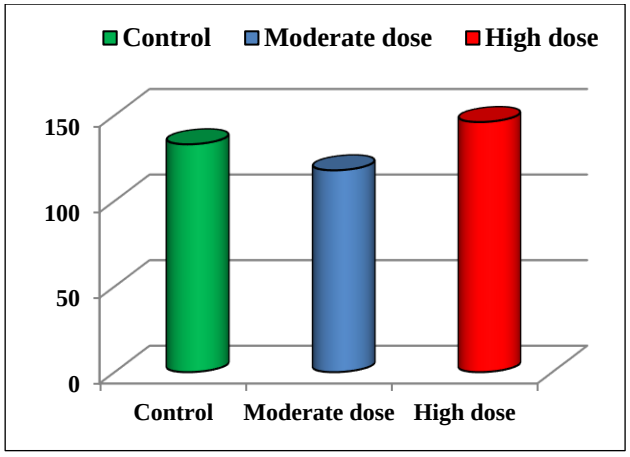

Figure 5: Comparison of LDL level.

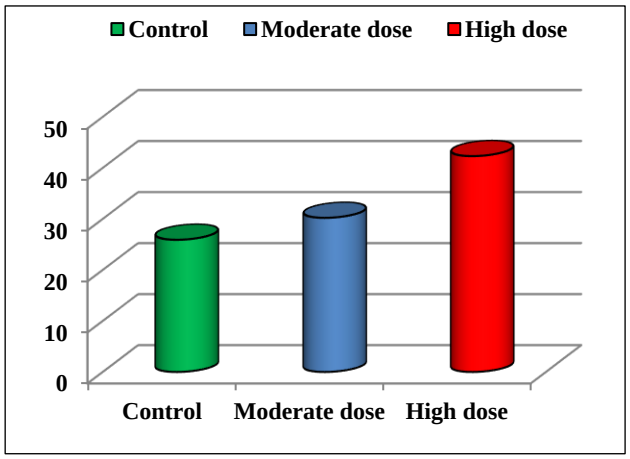

Figure 6: Comparison of VLDL level.

DISCUSSION

In the present study, 100 cases and 100 age and sex matched controls were selected randomly who satisfied the inclusion and exclusion criteria. Among the study subjects 94% were male and 6% were female.

The age distribution of the study subjects ranged from 21-60 years with maximum number of alcoholics seen in the age group of 41-50 years (45%) followed by 31-40 years (32%). This correlated well with the Indian study conducted by Vaswani et al, in which the age range was 18-60 years and mean age of alcoholics was 42.1±8.2.[11,12]

Among the study groups namely control, moderate dose and heavy dose alcoholics, the body weight of heavy dose alcoholics ((54.9±4.53) p value<0.01) was significantly lower when compared with the moderate dose alcoholics (61.0±7.16) and control group (59.1±4.36).

The BMI also showed a similar pattern in which the heavy dose group ((20.4±1.57) p value<0.01) had a significantly lower BMI when compared with the moderate dose (22.5±1.64) and control group (22.1±2.02) but none of the subjects were malnourished.

This result is consistent with the Indian study conducted by Drago et al, who also observed that none of the subjects who were chronic alcoholics were malnourished.[13] In contrast, their study showed that 14 out of 50 chronic alcoholics were overweight/obese with BMI>25, but in the present study none of the study subjects were overweight/obese.

Serum triglycerides

In the present study there was a statistically significant elevation of serum triglyceride level with both moderate ((150.9±21.92) p value<0.01) and heavy ((211.3±29.80) p value<0.01) dose alcohol consumption when compared with the control group (129.4±17.32). There was also a linear relationship between the dose of alcohol consumption and serum triglyceride level.

Table 5: Effect of alcohol consumption on serum triglycerides in various studies.

Studies	Control Mean ± SD	Moderate dose Mean±SD	Heavy dose Mean±SD
Goldberg et al[14]	223.5	250.2	281.1
Vasisht et al[5]	129±35.4	148±56	216±35.4
Sheetal et al[15]		132.41±56.41	254.94±107.79
Vaswani et al[12]	151.1±52.0		195.5±126.1
Present study	129.4±17.32	150.9±21.92	211.3±29.80

The Table 5 shows the effect of alcohol consumption on serum triglyceride level in various studies. The results of the present study correlate well with the study conducted by Goldberg et al, Vasisht et al, Sheetal et al, and Vaswani et al, which also showed a dose related increase in the level of serum triglyceride with regular alcohol consumption.[14,5,15,12]

Serum total cholesterol

The Serum Total Cholesterol level was significantly higher in the heavy dose alcoholics ((232.2±20.83) p value<0.01) when compared with the control group (201.1±12.88).

The moderate dose alcoholics ((199.3±15.16) p value 0.463) did not have much significant difference in the total cholesterol level when compared with the control group.

Table 6: Effect of alcohol consumption on serum total cholesterol in various studies.

Studies	Control Mean±SD	Moderate dose Mean±SD	Heavy dose Mean±SD
Goldberg et al[14]	221.2	218.8	209.0
Vasisht et al[5]	213±33.7	228±34.8	300±62.7
Sheetal et al[15]		154.08±25.38	214.33±40.74
Vaswani et al[12]	156.9±36.9		202.5±38.9
Present study	201.1±12.88	199.3±15.16	232.2±20.83

The Table 6 shows the effect of alcohol consumption on serum total cholesterol level in various studies. The results of the present study correlate well with the study conducted by Vasisht et al, Sheetal et al, and Vaswani M et al, which also showed an increase in serum total cholesterol level with heavy dose alcohol consumption.[5,15,12]

In contrast, the study conducted by Goldberg et al, showed a decrease in serum total cholesterol level with heavy dose alcohol consumption.[14]

The present study also correlates well with study conducted by Goldberg et al, and Vasisht et al, with respect to moderate dose alcohol consumption in which case there was no significant difference in serum total cholesterol level between moderate dose group and control group.[14,5]

Serum HDL

The serum HDL level was highest in the moderate dose alcoholics ((51.2±10.35) p value <0.01)) when compared with control group (42.2±5.35) indicating that moderate dose alcohol consumption is protective to the heart. The heavy dose alcoholics ((43.9±3.75) p value 0.026) did not have any significant difference in serum HDL level when compared with the control group.

Table 7: Effect of alcohol consumption on serum HDL in various studies.

Studies	Control Mean±SD	Moderate dose Mean±SD	Heavy dose Mean±SD
Vasisht et al[5]	47.2±6.2	52.6±5.5	60.2±7.18
Sheetal et al[15]		47.16±9.32	40.44±4.03
Drago et al[13]	41.9±7.6		60.2±7.18
Vaswani et al[12]	39.8±10.2		67.6±32.1
Present Study	42.2±5.35	51.2±10.35	43.9±3.75

The Table 7 shows the effect of alcohol consumption on serum HDL level in various studies. The present study correlates well with the study conducted by Vasisht et al, Sheetal et al, Drago et al, Vaswani et al, where there was an elevation in serum HDL level with moderate dose alcohol consumption.[5,15,13,12] The present study also correlates well with the study conducted by Sheetal et al, in which case serum HDL level was highest in the moderate dose alcoholic group which is in agreement with the statement that moderate alcohol consumption protects the heart.[15]

Serum LDL

The serum LDL level was significantly decreased in the moderate dose alcoholics ((117.9±19.24) p value <0.01) when compared with the control group (133.1±14.99) demonstrating the beneficial effect of moderate dose alcohol consumption whereas heavy dose alcoholics had significantly higher level ((146.1±22.79) p value <0.01) of serum LDL when compared with the control group suggesting the detrimental effect of heavy dose alcohol on health.

The Table 8 shows the effect of alcohol consumption on serum LDL level in various studies. The present study correlates well with the study conducted by Vashist et al, Drago et al, and Sheetal et al, with respect to the heavy dose group in which case serum LDL level was higher when compared with the control group.[5,13,15]

In contrast, the study conducted by Vaswani et al, showed a decreasing trend in serum LDL level with heavy dose alcohol consumption.[12] The present study also

shows a lower level of LDL in the moderate dose group when compared with the heavy dose group, again proving the protective effect of moderate alcohol consumption on the heart.

Table 8: Effect of alcohol consumption on serum LDL in various studies.

Studies	Control Mean±SD	Moderate dose Mean±SD	Heavy dose Mean±SD
Vasisht et al[5]	119.6±18.2	129.6±27	184±12
Sheetal et al[15]		80.42±26.31	123.44±38.44
Drago et al[13]	124.6±25		148.4±29
Vaswani et al[12]	90.1±28.8		79.6±38.3
Present Study	133.1±14.99	117.9±19.24	146.1±22.79

Serum VLDL

The serum VLDL level was steadily increasing with alcohol consumption in both moderate dose [(30.2±4.38) p value<0.01] and heavy dose [(42.3±5.96) p value <0.01] alcoholics when compared with the control group (25.9±3.46). The Table 9 shows the effect of alcohol consumption on serum VLDL level in various studies.

The present study correlates well with the study conducted by Vasisht et al, Sheetal et al, and Vaswani et al, in which case the serum VLDL level elevated with alcohol consumption in a dose dependent manner.[5,15,12] In contrast, the study conducted by Drago et al, showed a decrease in serum VLDL level with respect to heavy dose alcohol consumption.[13]

Table 9: Effect of alcohol consumption on serum VLDL in various studies.

Studies	Control Mean ± SD	Moderate dose Mean±SD	Heavy dose Mean±SD
Vasisht et al[5]	47.8±19	56±19.3	83.7±29.9
Sheetal et al[15]		26.50±11.29	51.00±21.55
Drago et al[13]	27.1±3.8		24.4±4.93
Vaswani et al[12]	29.8±9.1		57.7±39.2
Present study	25.9±3.46	30.2±4.38	42.3±5.96

CONCLUSION

From the present study the following conclusions can be drawn, patients with heavy alcohol consumption had significant increase in level of total cholesterol, triglycerides, LDL and VLDL when compared with the control group.

The mean serum level of HDL in moderate alcohol consumers had significantly increased when compared with the control group and declined after heavy alcohol consumption.

So, it can be concluded that moderate alcohol intake may increase HDL level and heavy alcohol intake is associated with increase in total cholesterol, triglycerides, LDL and VLDL level. Although moderate dose of alcohol consumption increases HDL level and seems cardio protective, its use should not be encouraged due to its deleterious effect on all spheres of life including social, economic and health.

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