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A study of effect of alcohol consumption on cardiovascular biomarkers in a tertiary care centre in the Kumaon Region

Devi Prasad Shukla[1], Basant Joshi[2], Sangeeta Singh[3,*], Ashok Kumar[4]

[1]*Dept. of Biochemistry, Government Medical College, Haldwani, Uttarakhand, India*
[2]*Dept. of Biochemistry, Rohilkhand Medical College and Nospital, Bareilly, Uttar Pradesh, India*
[3]*Dept. of Biochemistry, Soban Singh Jeena Government Institute of Medical Sciences and Research, Almora, Uttarakhand, India*
[4]*Dept of Medicine, Soban Singh Jeena Government Institute of Medical Sciences and Research, Almora, Uttarakhand, India*

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ABSTRACT

Introduction: An important mechanism responsible for increased cardiovascular risks in chronic excessive alcohol use is the pro-oxidant effects of alcohol. There are some emerging risk factors like :- (i) lipoprotein(a) {Lp[a]}, (ii) High-sensitivity C-reactive protein (hs-CRP), (iii) Lipid profile, (iv) Prothrombotic and proinflammatory factors that play an important role in the pathogenesis of atherosclerosis. So we investigated the relation between the levels of cardiovascular biomarkers & the degree of alcohol intake in alcoholic subjects.

Materials and Methods: The present study was carried out in the Department of Biochemistry, in association with the Department of Medicine, Government Medical College, Haldwani. Estimation of Serum Level of hs-CRP & Lp (a) by turbidimetric immunoassay. Serum Cholesterol by CHOD-POD & Triglycerides by enzymatic colorimetric method. LDL cholesterol was calculated by Friedwald equation.

Results: The mean total cholesterol, TG, LDLc, hs-CRP, Lp(a) & including HDLc levels were significantly raised ($p < 0.05$) in cases as compared to controls.

The mean serum total cholesterol & Lp (a) levels showed no significant association across different alcohol drinking groups ($p>0.05$). The mean TG & LDLc levels were significantly ($p<0.05$) higher in occasional drinkers and heavy drinkers than that of low-moderate & moderate drinkers. The mean serum HDL cholesterol levels in the occasional drinkers were significantly elevated in comparison to the low-moderate drinkers, moderate drinkers and heavy drinkers.

The mean serum hs-CRP level in the mediocre and heavy drinkers was significantly low ($p<0.05$) as compared occasional drinkers and low-moderate drinkers.

Conclusion: Our study suggests heavy drinking (>30drinks/week) must be strongly discouraged as it may lead to changes in cardiovascular markers and dyslipidemia. Our study also showed a beneficial effect of occasional drinking on HDLc and moderate drinking on hs-CRP.

1. Introduction

Alcohol refers to ethyl alcohol or ethanol a volatile liquid derived from fermentation of food stuffs, having formula C_nH_{2n}-OH (R-OH) with hydroxyl group attached to carbon chain. Alcohol has been in social use as a beverage since ages, widely used in various civilizations world over. Alcohol has been associated with dependence potential upon unregulated excessive use, it may even lead to impaired control, resulting in physical compulsions and repeated drinking leading to alcoholism.

* *Corresponding author*.
E-mail address: bjoshi86@gmail.com (S. Singh).

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In India, The National Family Health Survey (2005) reported national prevalence of alcohol use was 4.5%.[1] The unidentified consumption of alcohol for population older than 15 years, after 1995, in India is estimated to be 1.7 L pure alcohol per capita.[2]

The direct and indirect several biomarkers provide certain ways to calculate the amount of alcohol consumed and the duration of alcohol ingestion, the body resulting from as well as long-term alcohol misuse the harsh effects on consumption of alcohol.[3]

The high ethanol consumption is considered to contribute to oxidative stress and inflammation somehow. An important mechanism responsible for increased cardiovascular risks in chronic excessive alcohol use is the pro-oxidant effects of alcohol.[4] However, several studies of epidemiological data suggest that light to moderate alcohol consumption is associated with lower all-cause mortality than abstention or heavy alcohol intake, primarily through reduced risk of coronary heart disease.[5]

The consumption of alcohol as per the dose, individual susceptibility, genetic factors and diet can affect the lipid protein. The triglyceride (TG) synthesis and VLDL secretion by liver are stimulated because of alcohol intake.[6]The moderate intake of alcohol results in normal or slight increase in plasma triglyceride levels. The increased levels of HDL cholesterol is caused due to prolonged alcohol; this returns to reference range within two weeks of abstinence.[7]

Cardiovascular risk is frequently associated with the increased levels of many systemic inflammatory biomarkers and regulator proteins of the acute phase response.[8] Moderate alcohol intake has beneficial effects, recently anti-inflammatory mechanisms have been suggested to contribute to these beneficial effects of moderate alcohol intake in cardiovascular disease beyond changes in lipids and haemostatic factors (10). .There are some emerging risk factors like :- (i) lipoprotein(a) {Lp[a]}, (ii) high-sensitivity C-reactive protein (hs-CRP), (iii) Lipid profile, (iv) prothrombotic and proinflammatory factors that play an important role in the pathogenesis of atherosclerosis.[9]

High sensitivity C-reactive protein (hs-CRP) is an acute phase marker of inflammation. The hypothesis that CRP is a highly sensitive marker of systemic (micro)-inflammation (atherosclerosis), tissue damage and infection is supported by the observations from prior studies.[10]

Lp(a) is the main congenital lipid atherosclerosis risk factor. There's a high cardiovascular risk when the Lp(a) level is above 30mg/dl, almost two times and almost five times when LDL level is simultaneously increasedindependently of other lipid levels. The main congenital lipid atherosclerosis risk factor is Lp(a). The remaining 20% being related to environmental factors and cytokine concentrations (which function as acute phase proteins)about 80% of plasma Lp(a) concentration is genetically determined.[11]

Alcohol drinking has been associated with low grade inflammatory changes which can be depicted as changes in the levels of cardiovascular markers. The aim of the present study was to study the levels of serum biomarkers of cardiovascular- high sensitivity C-reactive protein (hs-CRP), lipoprotein-a {Lp-(a)} and lipid profile in alcoholic subjects and their age and sex matched non-alcoholic controls, to investigate the relation between the levels of cardiovascular biomarkers & the degree of alcohol intake in alcoholic subjects.

2. Materials and Methods

The present study was carried out in the Department of Biochemistry, in association with the Department of Medicine, Government Medical College, Haldwani, during the period of Sept 2017 to Aug 2019. The protocol was approved by the Institutional Board of Studies and passed by the Ethical Committee of the Institution.

2.1. Inclusion criteria

100 Alcoholic subjects, in the age group 25-60 years, randomly selected from the areas and ward, in and around Haldwani were included in the study.

2.2. Exclusion criteria

Patients of diabetes mellitus, hypertension, chronic liver diseases, cardiovascular diseases, renal diseases, thyroid dysfunction, Smokers, Pregnant females, Patients on anti-inflammatory drugs, Patients suffering from infectious and inflammatory diseases were excluded from the study.

2.3. Control

50 Age & sex matched non alcoholic subjects, randomly selected from the areas and wards, in and around Haldwani, meeting the exclusion criteria.

2.4. Study procedure

A detailed history from alcoholic subjects comprising type of alcoholic beverages consumed, amount, frequency and duration of alcohol consumption was recorded on participant proforma. Subjects were classified into different groups based on frequency and amount of alcohol consumption.

After a written informed consent, all patients were subjected to detailed history and thorough clinical examination. Alcoholic subjects were classified into different categories based on their alcohol intake in terms of drinks/week as group I (occasional drinkers, 1-10 drinks/week), group II (low-moderate drinkers, 11-20 drinks/week), group III (moderate drinkers, 21-30 drinks/week) and group IV (heavy drinkers, >30

drinks/week). {*A Standard drink consists of 10-14 gram of ethanol, which is equal to 12 Ounce or 300-360cc of Beer (5-7%), 120-150ml of wine(12%), 30-45ml of hard liquor (40-50% alcohol)}

Taking all aseptic precautions, about 5 ml of blood was drawn by venipuncture from a peripheral vein, with a disposable syringe. Sample for serum hs-CRP, lipoprotein (a) and lipid profile, were collected in plain vials in the morning after an overnight fast & were allowed to stand for 30 minutes at room temperature for the retraction of clot. It was then centrifuged at 3000 revolutions per minute for 10 minutes to separate the serum. The serum was stored at 4°C in the refrigerator for analysis. Care was taken to avoid haemolysis of samples.

Estimation of Serum Level of High sensitivity C-reactive protein & Lipoprotein(a) by turbidimetric immunoassay using semiautoanalyzer**.** Serum Cholesterol by CHOD-POD & Triglycerides by Enzymatic colorimetric method using (Roche Diagnostics) Roche/Hitachi Cobas c 501 analyzer. LDL cholesterol was calculated by Friedewald equation.

2.5. Statistical analysis

Using the statistical Package for the Social Sciences (SPSS 19.0.2) for windows, to analyze the data for qualitative significance unpaired t test and Duncan Multiple Range Test (DMRT) were used to show the inter-category significance ($p<0.05$).

3. Results

In the present study 37% of the cases were in the age group 25-33 years followed by 26% within 34-42 years, 25% within 43-51 years, 12% within 52-60 years. 87% of the cases were males.Table 1

The mean total cholesterol & LDLc levels were significantly raised ($p < 0.05$) in cases (181.17±32.88) & (101.290±31.01) as compared to controls (166.76± 31.98), (89.76±28.04) respectively. The mean HDLc level was also significantly raised ($p < 0.05$) in cases (50.390±7.10) as compared to controls (45.26±9.73).Table 2

The mean serum Total cholesterol levels showed no significant association across different alcohol drinking groups.

The mean TG & LDLc levels were significantly ($p<0.05$) higher in occasional (176.25±71.11), (105.12±27.52) drinkers and heavy drinkers (153.86±61.22), (109.31±40.60) than that of low-moderate (133.38±17.57), (98.61±26.39) & moderate (129.75±16.50), (92.40±29.54) drinkers respectively.Table 3

In the occasional drinkers (52.45±8.43) the mean serum HDL cholesterol levels were significantly elevated as compared to the low-moderate (50.94±4.98), moderate drinkers (50.15±7.98) and heavy drinkers (47.50±6.99).

The mean serum hs-CRP & Lp(a) levels were significantly raised ($p < 0.05$) in cases (0.334±0.24), (24.388±28.19) as compared to the controls (0.299±0.20), (22.116±20.13) respectively.Table 4

The mean serum hs-CRP level in the moderate drinkers (21-30 drinks/week) (0.290 ± 0.20) and heavy drinkers (>30 drinks/week) (0.290±0.26) was significantly lower ($p<0.05$) as compared occasional drinkers (1-10 drinks/week) (0.393 ± 0.28) and low-moderate drinkers (11-20 drinks/week) (0.324 ± 0.19) respectively. Whereas the mean serum Lp (a) levels in different alcoholic groups did not show any significant ($p>0.05$) association.Table 5

4. Discussion

The utilization of alcohol is associated in a dose dependent manner with cardiovascular morbidity and in mortality. Although there's a notion that increased the risk of cardiovascular (CVD) disease with the elevated inflammatory markers, underlying pathways and mechanisms remain to be illucidated. Specifically, cardiovascular and inflammation are mostly closely associated with each other by different mechanisms.[12]

The mechanisms of the process involving development of atherosclerosis are unclear. As reported in other studies, lipoprotein metabolism can be affected by inflammation, decreased plasma HDLc levels and impaired atheroprotective HDL functions which is reflected by these mechanisms. In addition to this the plasma lipoprotein levels is independent associated with relationship between inflammatory markers and atherosclerosis.

Conversely, the reason for the stimulation in the inflammatory process can be the presence of dyslipidemia. There are modified lipoproteins such as oxidized LDL are evidences that inflammation could be elicited by TG-rich lipoprotein remnants, as well as the oxidized LDL. Thus, vicious cycle is results the interplay between the lipid metabolism and inflammation at multiple levels may exacerbate the development of atherosclerosis resulting in a vicious cycle.

In our study, the mean serum Total cholesterol levels showed no significant association across different alcohol drinking groups. The mean LDLc levels were significantly higher ($p<0.05$) in occasional and heavy drinkers as compared to low-moderate and moderate drinkers (Tables 3 and 4). In our study the mean serum HDL cholesterol levels in the occasional drinkers were significantly elevated as compared to the low-moderate, moderate drinkers and heavy drinkers (Tables 3 and 4).

Van der Gaag et al, 1999 noted that consumption of alcohol at moderate level increases serum HDL cholesterol concentrations significantly but did not affect serum triglyceride levels. These results were determined previously as well.[13]

Table 1: Distribution of age and sex among the cases (n=100)

Age group (years)	Males		Females		Total	
	No	%	No	%	No	%
25-33	28	32.18	9	69.23	37	37.00
34-42	23	26.44	3	23.08	26	26.00
43-51	24	27.59	1	7.69	25	25.00
52-60	12	13.79	0	0.00	12	12.00
Total	87	100	13	100	100	100

Table 2: Lipid profile in the studied subjects

Parameter	**Cases** (n= 100) **{Mean ± SD}**	**Control** (n= 50) **{Mean ± SD}**
Total cholesterol (mg/dL)	181.17±32.88	166.76*± 31.98
HDLc (mg/dL)	50.390±7.10	45.26*±9.73
LDLc (mg/dL)	101.290±31.01	89.760*±28.04
TG (mg/dL)	147.450±49.66	158.7±100.14

*significance level as compared to controls: $p < 0.05$

Table 3: Serum levels of Lipid Biomarkers in cases according to alcohol intake

Parameters (mg/dl)	Degree of alcohol Intake			
	1-10 drinks/week(n=24)mean ± SD	**11- 20 drinks/week (n=34)mean ± SD**	**21-30 drinks/week(n=20)mean ± SD**	**>30 drinks/week(n= 22)mean ± SD**
Total cholesterol (mg/dL)	192.833^{a} ±33.24	176.235^{a} ±27.27	168.500^{a} ±26.10	187.591^{a} ±41.36
HDLc (mg/dL)	52.458^{a} ±8.43	50.941^{b} ±4.98	50.150^{b} ±7.98	47.500^{c} ±6.99
LDLc (mg/dL)	105.125^{a} ±27.52	98.618^{a} ±26.39	92.400^{b} ±29.54	109.318^{a} ±40.60
TG (mg/dL)	176.250^{a} ±71.11	133.382^{c} ±17.57	129.750^{c} ±16.50	153.864^{b} ±61.22

*Different alphabets shows significance (P<0.05) with each other and same alphabets shows no significant (P>0.05) association with each other.

Table 4: Serum levels of Biomarkers of cardiovascular inflammation in cases and controls

Parameter	**Cases** (n= 100) **mean ± SD**	**Control** (n= 50) **mean ± SD**
Serum hs-CRP (mg/dL)	0.334±0.24	0.299*±0.20
Serum Lp(a) (mg/dL)	24.39±28.19	22.12*±20.13

*significance level as compared to controls: $p < 0.05$

Table 5: Serum levels of Biomarkers of cardiovascular inflammation in cases according to alcohol intake

Parameter	Cases			
	1-10 drinks/week(n=24)mean ± SD	**11- 20 drinks/week (n=34) mean ± SD**	**21-30 drinks/week(n=20)mean ± SD**	**>30 drinks/week(n= 22)mean ± SD**
Serum hs-CRP (mg/dL)	0.393^{a} ± 0.28	0.324^{b} ± 0.19	0.290^{c} ± 0.20	0.290^{c} ±0.26
Lp (a) (mg/dL)	22.13^{a} ±22.19	23.23^{a} ±22.88	24.90^{a} ±31.25	28.16^{a} ±38.43

* Different alphabets show significance (P<0.05) with each other and same alphabets indicate no significance (P>0.05) with each other.

The benefits of alcohol may attenuate the small but a constant fair association between triglyceride (TG) concentration and amount of alcohol consumed. The various health study reports by physicians' show that there is increase TG level of 5.69 mg/dl increase due to consuming 30 g of alcohol in a day that may leads to increase 1.9% (0.5% to 3.3%) the risk of coronary heart disease and 4.6% after adjustment for intra individual variability in triglyceride concentration. We observed the mean TG levels in occasional drinkers and heavy drinkers were significantly ($p<0.05$) higher as compared to low-moderate, moderate drinkers. However light to moderate alcohol consumption may be associated with decreased plasma triglycerides, probably determined by the type of alcoholic, beverage consumed, genetic polymorphisms & lifestyle factors.[14]

In present study the mean serum hs-CRP level was found to be significantly raised ($p < 0.05$) in cases as compared to the controls, while comparing between groups according

to alcohol intake, the mean serum hs-CRP level in the moderate & heavy drinkers was significantly less ($p<0.05$) as compared occasional drinkers and low-moderate drinkers respectively, while low-moderate drinkers had hs-CRP levels significantly lower ($p<0.05$) than occasional drinkers. This is in line with the earlier works showing linear relation between hs- CRP and increasing alcohol intake.

O'Keefe et al., 2014, shows that the maximal survival benefit is between 0.5 and 1 drinks (7-14 g) daily for women and between 1 and 2 drinks (14-28 g) for men.[15] Previous studies have reported a beneficial effect of moderate alcohol consumption on hs-CRP levels. Most of them showed J-shaped associations between alcohol and hs-CRP in both men and women.[12,16] Imhof et al.,2001 have recently reported that there is a U-shaped relation between alcohol intake and C-reactive protein (CRP) with the lowest CRP concentration at alcohol intake of 40–60 g day) of ethanol.[17]

Yudkin JS et al., 1999 observed inflammatory factors (e.g. cytokines and CRP) to be associated with dyslipidemia, hypertension, and insulin resistance while previous studies have provided plausible biochemical mechanisms for the suggested role of inflammation in the pathophysiology of cardiovascular disorders in different population.[18]

As per our examination the mean serum Lp(a) level was significantly raised ($p < 0.05$) in cases as compared to the controls. However, the mean serum Lp (a) levels in different alcoholic groups did not show any significant ($p>0.05$) association in between them.

In the study of willeit et al[19] showed that higher tendency towards high levels of lp(a) was associated with increase drinking of alcohol but the relationship between quantity of alcohol intake and plasma concentration of Lp(a) has negative correlation this was observed by Valimaki et al.,[20] Isoet al.[21] and Paassilta et al.,[22] whereas soon after alcohol drinking cessation highest Lp(a) level becomes observed and succeeding months it is decreased as per the study by J. Budzynski et al.

Belfarge P et al.,[23] and Johansson BG et al.[24] There's a high cardiovascular risk when the Lp(a) level is above 30mg/dl , almost two times, level of other lipids independently and simultaneously almost level of LDL five times more increased. It is proportional to the alcohol intake.

Coronary heart disease mortality can be decreased due to it has protective effects in moderate quantity of alcohol consumption, while leads to cardiomyopathy, coronary heart disease or hypertension and haemorrhage stroke due to excessive intake of alcohol has detrimental effects on cardiovascular system.

5. Conclusion

In conclusion our study demonstrated significant changes in lipid profile (decreased level of serum HDLc, increased level of serum LDLc & Tg) in heavy drinkers (>30drinks/week) and in occasional drinkers (1-10 drinks/ week) had significantly high HDLc levels as compared to moderate (21-30 drinks/week) and heavy drinkers.

Levels of Lp(a) were significantly elevated in all categories of alcoholics as compared to non-alcoholic controls while hs-CRP level was comparatively less in moderate and heavy drinkers to those of low moderate and occasional drinkers.

Our study suggests that heavy drinking may lead to significant dyslipidemia and inflammatory changes and adversely affect the cardiovascular system but has shown a beneficial effect of occasional drinking on HDLc levels and moderate drinking on hs-CRP levels. However a large scale study needs to be done to confirm these beneficial effects of occasional to moderate drinking on the cardiovascular system

6. Limitations

The study was confined to the dwellers of Haldwani and their involvement in the study especially in case of females. Due to lack of fund, time and manpower, the inclusion of a large study group and other sensitive biochemical markers of alcoholics was not possible.

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None.

10. Conflict of Interest

None.

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Author biography

Devi Prasad Shukla, Post Graduate Student

Basant Joshi, Assistant Professor https://orcid.org/0000-0003-1312-6983

Sangeeta Singh, Associate Professor

Ashok Kumar, Associate Professor

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