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Impact of Alcohol Intake on Cardiovascular Biomarkers: A Prospective Analysis

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ABSTRACT

The aim of this study was to investigate the relationship between cardiovascular biomarker levels and the degree of alcohol intake in alcoholic subjects, considering the pro-oxidant effects of alcohol as a significant mechanism contributing to increased cardiovascular risks. Emerging risk factors such as lipoproteins, high-sensitivity C-reactive protein (hs-CRP), lipid profile and prothrombotic and pro-inflammatory factors were also examined. One hundred twenty three alcoholic subjects aged 18-60 years were randomly selected. Serum levels of hs-CRP and Lp (a) were determined using turbidimetric immunoassay, while serum cholesterol was measured using CHOD-POD and triglycerides using an enzymatic colorimetric method. LDL cholesterol was calculated using the Friedwald equation. There was no significant association observed between mean serum total cholesterol levels and different alcohol drinking groups. However, mean TG and LDL levels were significantly higher in occasional and heavy drinkers compared to low-moderate and moderate drinkers. Among occasional drinkers, mean serum HDL cholesterol levels were significantly elevated compared to other groups. Our findings suggest that heavy drinking may lead to significant dyslipidemia and inflammatory changes, adversely affecting the cardiovascular system. However, occasional drinking demonstrated a beneficial effect on HDL levels, while moderate drinking showed favorable effects on hs-CRP levels. Nonetheless, further large-scale studies are warranted to confirm these potential benefits of occasional to moderate drinking on cardiovascular health.

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Key Words
Alcohol, lipid profile, HS-CRP, cardiovascular biomarkers

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INTRODUCTION

The connection between alcohol consumption and cardiovascular disease (CVD) the primary cause of global mortality, has been a subject of debate. Observational studies consistently indicate a lower CVD risk with light to moderate alcohol intake, presenting a J- or U-shaped epidemiological association compared to abstinence or heavy drinking. However, the perceived cardiac benefits of alcohol consumption have been proposed to stem from residual confounding due to favorable lifestyle, socioeconomic, and behavioral factors associated with moderate alcohol use[1-3].

Alcohol consumption exerts multifaceted effects on cardiovascular health, impacting various CV diseases such as hypertension, coronary heart disease, stroke, peripheral arterial disease and cardiomyopathy [4]. While numerous behavioral, genetic and biological factors influence the interaction between alcohol use and CV disease the dose and pattern of alcohol consumption appear to play a crucial role[4].

Low-to-moderate alcohol consumption may ameliorate certain mechanisms affecting atherosclerosis and inflammation, key pathophysiological processes underlying most CV diseases. However, the potential benefits of drinking must be balanced against significant physiological consequences, including mitochondrial dysfunction, alterations in circulation, inflammatory responses, oxidative stress, programmed cell death, and structural damage to the cardiovascular system, particularly the heart[5-8].

Our objective was to investigate the relationship between cardiovascular biomarker levels and the degree of alcohol intake in alcoholic subjects, considering the pro-oxidant effects of alcohol as a significant mechanism contributing to increased cardiovascular risks.

MATERIAL AND METHODS

123 alcoholic subjects aged between 18 and 60 years were randomly chosen for this study. Patients with diabetes mellitus, hypertension, chronic liver diseases, cardiovascular diseases, renal diseases, thyroid dysfunction, smokers, pregnant females, patients taking anti-inflammatory drugs and those suffering from infectious and inflammatory diseases were excluded from the study. A total of 78 nonalcoholic subjects, matched for age and sex, were randomly selected from the same areas and wards, meeting the exclusion criteria.

Comprehensive histories of alcoholic subjects, detailing the type, amount, frequency and duration of alcohol consumption, were recorded using a standardized participant format. Subjects were categorized based on the frequency and amount of alcohol intake. All participants underwent detailed history taking and thorough clinical examinations. Alcoholic subjects were stratified into different groups based on their weekly alcohol consumption 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).

Blood samples (approximately 5 mL) were drawn via venipuncture from a peripheral vein using disposable syringes, with aseptic precautions. Serum samples for high sensitivity C-reactive protein (hs-CRP), lipoprotein (a) and lipid profile were collected in plain vials in the morning after an overnight fast. These samples were allowed to clot for 30 minutes at room temperature, then centrifuged at 3000 revolutions per minute for 10 minutes to obtain serum, which was stored at 4°C in a refrigerator prior to analysis. Measures were taken to prevent hemolysis of samples.

Serum levels of hs-CRP and lipoprotein (a) were estimated using turbidimetric immunoassays. Serum cholesterol was assessed using the CHOD-POD method, and triglycerides were measured using an enzymatic colorimetric method with a Roche/Hitachi Cobas c 501 analyzer. LDL cholesterol was calculated using the Friedewald equation.

RESULTS

In this study, the majority of cases comprised males aged 51-60 years (Table 1). Comparing cases to controls, there was a significant increase in mean total cholesterol and LDL levels ($p<0.05$), along with a significant decrease in mean HDL level ($p<0.05$) (Table 2). Regarding alcohol consumption, no significant association was observed between mean serum total cholesterol levels and different alcohol drinking groups. However, mean TG and LDL levels were significantly higher ($p<0.05$) in occasional drinkers and heavy drinkers compared to low-moderate and moderate drinkers. Among occasional drinkers, mean serum HDL cholesterol levels were notably elevated compared to other drinker groups (Table 3).

Furthermore, mean serum levels of hs-CRP and Lp (a) were significantly higher ($p<0.05$) in cases compared to controls (Table 4). Notably, the mean serum hs-CRP level was significantly lower ($p<0.05$) in moderate drinkers and heavy drinkers compared to occasional drinkers and low-moderate drinkers. However, there was no significant association ($p>0.05$) between mean serum Lp (a) levels and different alcoholic groups (Table 5).

DISCUSSIONS

In a large-scale prospective study, individuals who reported consuming alcohol at a weekly rate of 1-150 g had a decreased risk of cardiovascular disease (CVD), cancer and mortality compared to both non-drinkers and heavy drinkers. This level of alcohol intake

Table 1: Demographic details of study participants

	Cases (123)		Controls (78)	
Gender	No.	Percentage	No.	Percentage
Male	108	87.80	64	82.05
Female	15	12.20	14	17.95
Total	123	100.00	78	100.00
Age groups				
18-30 years	18	14.63	14	17.95
31-40 years	23	18.70	18	23.08
41-50 years	40	32.52	21	26.92
51-60 years	42	34.15	25	32.05
Total	123	100.00	78	100.00

Table 2: Lipid profile parameters of study participants

Parameters (mg dL^{-1})	Cases (123) Mean±SD	Controls (78) Mean±SD
Total cholesterol	210.75±27.78	150.92±28.19
Triglycerides	195.74±21.57	137.00±19.89
High-density lipoprotein (HDL)	38.12±4.72	41.48±6.54
Low-density lipoprotein (LDL)	135.09±18.64	83.81±18.09
Very Low-density lipoprotein (VLDL)	38.61±4.49	27.91±4.12

Table 3: Degree of alcohol intake wise Lipid profile parameters

Degree of alcohol Intake (drinks per week)	Total cholesterol	Triglycerides	HDL	LDL
1-10 (n = 29)	200.91±30.79	174.87±61.78	34.79±5.99	130.68±12.03
11-20 (n = 42)	192.78±25.84	165.89±14.62	34.20±2.67	126.21±10.98
21-30 (n = 45)	177.29±21.92	149.82±13.92	35.02±5.87	110.64±14.54
>30 (n = 27)	178.04±21.35	155.07±55.79	31.68±5.34	116.02±16.08

Table 4: Cardiovascular inflammatory biomarkers in study participants

Variable (mgdL^{-1})	Cases (123) Mean±SD	Controls (78) Mean±SD
Serum hs-CRP	0.361±0.21	0.235±0.06
Serum Lp (a)	25.89±2.52	21.17±6.03

Table 5: Degree of alcohol intake wise cardiovascular inflammatory biomarkers

Degree of alcohol Intake (drinks per week)	Serum hs-CRP	Serum Lp(a)
1-10 (n=29)	0.329±0.13	20.81±2.26
11-20 (n=42)	0.314±0.15	22.61±2.35
21-30 (n=45)	0.276±0.16	23.19±2.68
>30 (n=27)	0.269±0.12	27.78±3.12

corresponds to approximately no more than 10 servings of alcohol per week, a range consistent with most global dietary guidelines for low-risk drinking cutoffs (100-300 g per week)[9]. However, within the Indian cohort, the lowest risk was observed among those consuming approximately 25g per week, equivalent to around 2 servings per week. Since the mechanisms for each CVD disorder or site-specific cancer differ, it's essential to consider both individual and composite outcomes when formulating dietary policies. Currently, India lacks national policies on alcohol sales restrictions and dietary guidelines for alcoholic beverages. This study provides evidence supporting the recommendation for light alcohol consumption.

Our study conducted in an Indian cohort corroborates previous findings of a J-shaped association between alcohol consumption and CVD. We observed a lower overall CVD risk among those consuming 1-10 drinks per week. Alcohol may impact CVD risk through its favorable effects on raising high-density lipoprotein (HDL) concentration and reducing inflammation, as well as its unfavorable effect of increasing blood pressure. However, the influence of these risk factors on different CVDs, such as myocardial infarction versus stroke, may vary, potentially mediating their association with alcohol consumption. For instance, the association between HDL-C and myocardial infarction might be stronger than its association with stroke. Moreover, high blood pressure consistently emerges as the strongest risk factor for stroke risk, contributing to diverse patterns in the associations between alcohol consumption and myocardial infarction or heart failure versus stroke [10-13].

The precise causal pathway of alcohol remains unclear for all cancer types. For instance, in female breast cancer, alcohol might elevate risk by altering estrogen levels and estrogen receptors[14]. Therefore, the inverse association between light alcohol intake (<25 g per week) and overall cancer risk likely stemmed from the lower risk of non-alcohol-related cancer. However, limited statistical power due to the small number of individual non-alcohol-related cancer cases prevented us from fully addressing this hypothesis.

Our findings indicate that light to moderate alcohol intake consistently correlates with a reduced risk of total mortality and mortality specific to CVD and cancer. The former observation aligns with the results of a recent meta-analysis of 87 studies, which revealed that compared to non-drinkers or heavy alcohol consumers, low-volume alcohol consumers (9.1-174 g alcohol per week) had a 14% lower risk of overall mortality[15].

In other cohorts, the association between alcohol consumption and major chronic disease risk was influenced by smoking status. Given that alcohol consumption and smoking commonly co-occur as addictive behaviors, smoking might obscure the effect of alcohol alone on health status[16]. As anticipated, although the protective association of light-to-moderate alcohol consumption against CVD, cancer and mortality was significant in both smokers and non-smokers, it was stronger in non-smokers. Nonetheless, even for lung cancer, light alcohol consumption still exhibited the lowest risk compared to non-drinkers and heavy drinkers[17].

The potential biological mechanism underlying the overall beneficial effect of alcohol consumption remains incompletely understood but may involve alterations in cholesterol concentrations, particularly HDL-C and triglyceride concentrations, improvements in insulin sensitivity, reductions in the inflammatory process during cell signaling, decreases in platelet aggregation and blood clotting and interactions with genetic variation in alcohol dehydrogenase polymorphism. Although recent genetic epidemiological studies suggested that drinkers with ALDH2 deficiency had a more favorable profile for CVD risk factors, independent of alcohol consumption, no relation was reported in non-drinkers[18-20].

CONCLUSION

Our findings indicate that heavy drinking may contribute to notable dyslipidemia and inflammatory alterations, potentially exerting adverse effects on the cardiovascular system. Conversely, occasional drinking appears to have a beneficial impact on HDL levels, while moderate drinking demonstrates favorable effects on hs-CRP levels. Nevertheless, to validate these potential benefits of occasional to moderate drinking on cardiovascular health, a comprehensive large-scale study is warranted.

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