International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article

Joint Effects of Alcohol Consumption and Cigarette Smoking on Atherogenic Lipid Profile: In Men at Durg (Chhattisgarh)

N.S. Dixit[1], Suvimal Dutta[2], Karan Singh Chandrakar[3*]

[1]Assistant Professor, Department of Medicine, CCM Medical College, Kachandur, Durg, Chattisgarh, India.
[2]Assistant Professor, Department of Surgery, CCM Medical College, Kachandur, Durg, Chattisgarh, India.
[3*]Assistant Professor, Department of Pathology, CCM Medical College, Kachandur, Durg, Chattisgarh, India.

ABSTRACT

Purpose: The present study examines the Joint effects of alcohol consumption and cigarette smoking on atherogenic lipid profile among male adults in Durg.

Methods: A total number of 490 male subjects were selected at CCM Medical college Hospital Durg. 297 males are alcohol takers and chronic smokers, who were smoke more than 10-15 cigarettes / bidi per day and take more than 180 ml alcohol in the form of whiskey, rum or bear more than 3 times per week from last 5 years with an average range of 37 to 50 years. 193 nonalcoholic and non-smokers age matched males were selected for control group. Controls were clinically and physically normal and healthy.

Results: The frequency of consumption of alcohol was; daily 166 patients (55.89 %), 3-5 times per week in 131 patients (44.10 %) and all patients 297 (100%) smokes 10-15 cigarettes or bidi per day. Majority alcohol dependent patients drink alcohol alone and some are claimed to drink only in company or with friends. In the observed population, the value of TC, TG in cigarette / bidi smokers increased significantly and monotonically with increased level of cigarette smoking and alcohol consumption. In addition alcohol intake significantly associated with increased HDL-cholesterol and decreased LDL level in a dose dependent manner. The effect of alcohol consumption on HDL-c and TG levels was substantially greater for heavy smokers than light smokers, while alcohol intake exerted a strong positive influence on HDL-c level regardless of level of cigarette / bidi smoking.

Conclusion: On the basis of our study results we concluded that the lipid profile changes in patients of alcohol drinkers and cigarette smokers, with some correlation to the liver dysfunction. Alcohol causes alteration in lipid profile level in blood including those which predispose to CHD. Cigarette smoking and alcohol consumption were confirmed to have similar effect on lipid and lipoproteins as in Caucasians. A significance of joint exposure to drinking and smoking predict lipid levels were evident. Low or moderate drinking alcohol over prolonged periods has been linked to have protective influence for development of CHD, through increase in HDL-c levels.

Keywords: Total Cholesterol, Triglyceride, High Density Lipoprotein, Low Density Lipoprotein, Very Low Density Lipoprotein, ADS (Alcohol Dependence Syndrome), CHD (Coronary Heart Disease).

***Correspondence to:**

Dr. Karan Singh Chandrakar,
Assistant Professor Dept. of Pathology,
CCM Medical College,
Kachandur, Durg Chhattisgarh.

Article History:

Received: 23-03-2017, **Revised:** 17-04-2017, **Accepted:** 01-05-2017

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2017.3.3.033	

INTRODUCTION

Cigarette smoking and alcohol consumption are the most potent & prevalent addictive habits influencing behavior of human beings. Alcohol consumption can be the cause of several different diseases and high burden if its consumption over mortality around the world.[1] Alcohol is only drug that provides energy (7.1 kcal /g). However alcohol intake can increase the weigh and develop obesity. Additionally the qualities, frequency in which alcoholic beverages are consumed are important factors that should be include in the evaluation of the risk for cardiovascular disease. Depending upon the age of subjects and dose of alcohol ingestion. the alcohol consumption lead to different effects. Alcohol dependence syndrome is defined as "A cluster of physiological, behavioral and cognitive phenomena in which the use of substance or a class of substance take on a much higher priority for a given individual than other behavior that once had greater value".[2]

A world Health organization project psychological problem in general practice has shown that 6% patients attending primary care uses harmful alcohol, after major depression and generalized anxiety.[3]

Drinking of heavy alcohol or harmful alcohol makes a substantial contribution to the burden of disease and premature mortality.[4] The liver is the organ mostly affected by alcoholism. In urban areas, Cirrhosis is the main and fourth leading cause of death in young ages. Alcohol abuse is a major health as well as social problem in community.

Smoking is a different from is a major risk factor for cardiovascular disease, Atherosclerosis and coronary heart disease.[5] Smoking is now increasing rapidly throughout the world and it is one of the highest threat to current & feature world health. Cigarette or bidi smoking is associated with a more atherogenic lipid profile.[6]

Alcohol consumption and Cigarette or bidi smoking are known to be an important factor influencing lipid metabolism. Alcohol consumption strongly related to high density lipoprotein cholesterol (HDL-C), total cholesterol and lesser extend to triglyceride, while cigarette or bidi smoking lead to increase in the concentration of serum total cholesterol, triglyceride, LDL-cholesterol, VLDL- cholesterol. The association between alcohol consumption and blood lipid is complicated by the strong interrelationship between alcohol and smoking, which has been shown to have an influence an lipid profile.[7,8]

There is generally serum HDL- cholesterol is increased by alcohol consumption and it is decreased by smoking but the dose relationship between HDL- cholesterol and the combined use of these two social habits has not been quantified. The effects of alcohol consumption on serum total cholesterol are inconsistent; although the increase in cholesterol with cigarette smoking is clear and has been shown to be dose related.[9] Few studies have examined the simultaneous effects of smoking and alcohol on blood lipids. Because of the strong interrelationship between alcohol and smoking and the known association of both with body weight, which itself influences lipid profile.

The aim of this study was to investigate serum lipid profile in chronic smokers and heavy alcoholism's at Durg city Chhattisgarh. This will help to determine the independence of such relationship.

MATERIAL AND METHODS

A total number of 490 male subjects were selected at CCM Medical college Hospital Durg. 297 males are alcohol takers and chronic smokers, who were smoke more than 10-15 cigarettes / bidi per day and take more than 180 ml alcohol in the form of whiskey, rum or bear more than 3 times per week from last 5 years with an average range of 37 to 50 years. 193 nonalcoholic and non-smokers age matched males were selected for control group. Controls were clinically and physically normal and healthy.

Exclusion Criteria

1. Smoking from last 5 years
2. Ex- Smokers
3. Alcoholics
4. Hypertension
5. Familial Coronary heart disease
6. Obesity
7. Lipid lowering drugs

Sample Collection

Fasting 5 ml blood sample was collected in plain dry test tube. Serum sample was obtained by centrifugation and sample were immediately separated into another plain dry test tube and stored at -20^0 C. Serum sample was used to estimate serum total Cholesterol (TC), serum Triglyceride (TG), serum low density lipoprotein (LDL-C) and serum High density lipoprotein (HDL-C) by using fully autoanalyser by enzymatic , colorimetric method.

Data were expressed as mean ±SD. Mean values were assessed for significance by unpaired student –t test. A statistical analysis was performed using the Stastical Package for the Social Science program (SPSS, 21.0). Frequencies and percentages were used for the categorical measures. Probability values $p < 0.001$ were considered statistically significant.

OBSERVATION AND RESULTS

Mean age of the patients and control subjects was 36.29 ± 4.93 and 33.96 ± 3.86 years respectively. Range age was 37 to 50 years for patients and for control also. The majority of subjects had begun voluntarily and some are claimed to have done so due to pressure. The average daily intake of alcohol was more than 300 ml near about 80 gm per day and more than 10-15 cigarettes per day. Mean duration of drinking alcohol and smoking was 6 years. The frequency of consumption of alcohol was; daily 166 patients (55.89 %), 3-5 times per week in 131 patients (44.10 %) and all patients 297 (100%) smokes 10-15 cigarettes or bidi per day. Majority alcohol dependent patients drink alcohol alone and some are claimed to drink only in company or with friends.

Table 1: Comparison of Mean age of patients and control subjects

Subject Class	Mean age SD	P- value
Control subject	36.29 ± 4.93	0.189
Alcoholic and smokers patients	33.96 ± 3.86	

Table 2: Lipid Profile parameters in alcoholic, smokers and Control subjects

Name of Parameters	Alcoholic with smokers	Control (Nonalcoholic nonsmokers)	P- Value
Total Cholesterol mg/dl	312.78 ±31.59	165.39 ± 24.72	<0.05
Triglyceride mg/dl	208.18 ± 60.37	102.69 ± 29.47	<0.05
VLDL- Cholesterol mg/dl	42.59 ± 10.98	21.52 ± 5.93	<0.05
LDL- Cholesterol mg/dl	203.28 ± 19.38	112.83 ± 11.92	<0.05
HDL- Cholesterol mg/dl	40.70 ± 9.27	57.29 ± 6.14	<0.05

Above table shows the lipid profile parameters such as TC, TG, VLDL-c, and LDL-c activity were significantly higher in alcoholic with smoker patients than control subjects but activity of HDL-c was significantly decreased in alcoholic with smoker patients than control subjects.

In the observed population, the value of TC, TG in cigarette / bidi smokers increased significantly and monotonically with increased level of cigarette smoking and alcohol consumption. In addition alcohol intake significantly associated with increased HDL-cholesterol and decreased LDL level in a dose dependent manner. The effect of alcohol consumption on HDL-c and TG levels was substantially greater for heavy smokers than light smokers, while alcohol intake exerted a strong positive influence on HDL-c level regardless of level of cigarette / bidi smoking.

DISCUSSION

The total cholesterol levels were found to be higher in patient group (alcohol drinker and cigarette or bidi smokers) than normal controls with strong statistical significance. The total TG LDL-C and VLDL-C levels were also increased patient group than normal control subjects, but the level of HDL-c level was not significantly increased or decreased in patients group but slightly decreased found when compared to normal control subjects.

Also, among the case, majority had increased TG level. LDL-c was normal in some patients. In heavy drinkers the level of HDL-c elevated found in most of the patients. These features are in the line of the notion of cardiac protective effect of alcohol consumption by maintaining the level of LDL-c and HDL-c with an expense of slightly raised TG level. The increase in HDL-c has been estimated to account for half of the beneficial effect of alcohol consumption on cardiovascular disease.[10] Prolonged excessive drinking causes various structural and functional abnormalities of heart.

The UK Regional Heart study[11] reported that cigarette smoking and alcohol consumption had little or no influence on total cholesterol or triglyceride concentrations. Triglyceride concentration was raised in heavy drinkers, but not conspicuously. For HDL cholesterol they found a positive relationship with alcohol consumption, but no clear relationship with cigarette smoking. Only when the subjects' studies were divided into current smokers and a never smoked group was a significantly lower HDL-C found in the smokers. The present study, involving much larger number of subjects, found significant increase in the cholesterol concentration with both smoking and dirking, and a marked negative dose-response relationship between cigarette smoking and HDL-C.

A recent Japanese study[12,13] found alcohol drinkers to have higher HDL-C triglyceride levels then non-drinkers, after adjusting for differences in age, body mass index and smoking habits. Total cholesterol was not significantly related to alcohol consumption. Although their finding in relation to cholesterol conflict with those of the current study, the authors come to the same conclusion, namely that the most beneficial protective effect of a moderate alcohol intake is to be found in non-smoking.

This study has investigated the changes in serum lipid level with smoking and drinking in a large sample of men. Both social habits were found to raise serum cholesterol level in a dose-related manner-the more cigarettes a subject smokes and the more alcohol consumed, the higher the total serum cholesterol.

The well-known increase in HDL-C concentration with alcohol consumption, even in those dirking only small amounts and a highly significant fall in HDL-C with smoking were observed. Smoking only 10 cigarettes or less per day was sufficient to negate the beneficial effects of moderate drinking on HDL-C particularly when considering the latter as a % of total cholesterol. This study does not confirm an often held view that alcohol consumption raises serum triglyceride concentrations, but it clearly shown an increase in triglyceride with smoking.

There is epidemiological evidence that light to moderate consumption of alcohol has beneficial effects on overall health and on coronary disease in particular. The increase in HDL-C with alcohol consumption has provided a possible biological mechanism.[14]

On the other hand, Haffner et al.[15] found that cigarette smoking and alcohol consumption principally effect the HDL-3 subfraction of HDL-C, rather than HDL-2. Since heart disease is associated more strongly with lower HDL-2 levels; they concluded that 'the increase in risk of ischemic heart disease with smoking and the possible decrease with alcohol consumption may be mediated through mechanisms other than their effects on HDL-C'. Although this study was based on only 33 men and 17 women similar results were found in a later study on 366 men.[16,17]

CONCLUSION

On the basis of our study results we concluded that the lipid profile changes in patients of alcohol drinkers and cigarette smokers, with some correlation to the liver dysfunction. Alcohol causes alteration in lipid profile level in blood including those which predispose to CHD. Cigarette smoking and alcohol consumption were confirmed to have similar effect on lipid and lipoproteins as in Caucasians. A significance of joint exposure to drinking and smoking predict lipid levels were evident. Low or moderate drinking alcohol over prolonged periods has been linked to have protective influence for development of CHD, through increase in HDL-c levels.

REFERENCES

1. World health organization (WHO). Global status report an alcohol & health 2011.
2. World health organization (WHO). International classification of disease & related health problems 10th edition 1992.
3. Goldberg D, lecrubier Y. Forms and frequency of mental disorders across centers New York John Wiley 1995:324:-34.
4. Balabanovaa D, Maker M. Pattern of alcohol consumption in Bulgaria, alcohol and alcoholism 1999.34: 622-628.
5. Mc Gill Hc. Cardiovascular pathology of smoking Am heart J. 1988:115:242-247.
6. Cempbell Sc, Mofatt RJ. Stamford BA. Smoking and smoking cessation. The relationship between Cardiovascular disease & lipoprotein metabolism; A reissueAtherosclerosis2008:201:225-35.
7. Gordon T, Doyle JT. Alcohol consumption and its relationship to smoking weigh BP and blood lipid. The Albany study. Arch Jntern Med 1988:146:262-265.
8. Ceiqui M, Wallace R. Cigarette smoking and plasma HDL-C the lipids research clinics program prevalence study. Circulation 1980, 62-70.
9. Mascat JE, Harris RE Haley NJ et al. Cigarette smoking and plasma cholesterol. Am Heart J 1991:121: 141-147.

10. M.H. Criqui, L.D. Cowan H.A. Tyroler et al. Lipoproteins as a mediators for effect of alcohol consumption and cigarette smoking on cardiovascular mortality: results from the lipid research Clinics Follow up Study. Am J Epidermiol 1987;126:629-637.
11. Theile DS, Shaper AG, Whitehead TP, Bullock 00, Ashby D, Patel I. Blood lipids in middle-aged British men. Br Heart J 1983; 49: 205-213.
12. Garrison RJ, Kannel WB, Feinleib M, Castelli WP, McNamara PM, Padgett SJ. Cigarette smoking and HDL cholesterol. The Framingham Offspring Study. Atherosclerosis 1978; 30: 17-25.
13. Choudhury SR, Ueshima H, Kita Y, Kobayashi KM, Okayarna A, Yamakawa M, et al. Alcohol intake and serum lipids in a Japanese population. Int J Epidemiol 1994; 23: 940-947.
14. Suh I, Shaten J, Cutler JA, Kuller LH. Alcohol use and mortality from coronary heart disease: the role of high density lipoprotein cholesterol. Ann Intern Med 1992; 16: 881-887.
15. Haffner SM, Applebaum-Bowden D, Wahl PW, Hoover, Warnick GR, Albers 11, et al. Epidemiological correlates of high density lipoprotein subfractions, apolipoproteins A-I, A-II, and D, and lecithincholesterol acyltransferase. Effects of smoking, alcohol and adiposity. Arteriosclerosis 1985;S: 169-177.
16. Gofman JW, Young W, Tandy R. Ischemic heart disease, atherosclerosis and longevity. Circulation 1966; 34: 667-697.
17. Robinson D, Ferns GAA, Bevan EA, Stocks J, Williams PT, Galton DJ. High density lipoprotein subfractions and coronary risk factors in normal men. Arteriosclerosis 1987; 7: 341-346.

Source of Support: Nil. **Conflict of Interest:** None Declared.

Cite this article as: N.S. Dixit, Suvimal Dutta, Karan Singh Chandrakar. Joint Effects of Alcohol Consumption and Cigarette Smoking on Atherogenic Lipid Profile: In Men at Durg (Chhattisgarh). Int J Med Res Prof. 2017; 3(3):158-61.
DOI:10.21276/ijmrp.2017.3.3.033