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*GJMR-I Classification :* *NLMC Code: QV 665, QV 664, QV 662*


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# Effect of Cigarette Smoking on Blood Lipids – A Study in Belgaum, Northern Karnataka, India

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## I. Introduction

Lipids play an important role virtually in all aspects of biological life. Some of these roles include serving as hormones or hormone precursors, helping in digestion, providing energy, storage function and metabolic fuels; acting as functional and structural compounds in biomembranes and forming insulation to allow nerve conduction or to prevent heat loss (1).

Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease and peripheral vascular disorders. (2). There is a dose response relationship between the number of cigarettes smoked per day and cardiovascular morbidity and mortality (3). Long delay between smoking and onset of smoking related diseases resulted in the ignorance of ill effects of smoking (4). On an average smoker lose more than a day of their life span for every week of smoking. Smoking kills more than one in three regular smokers (5). In India consumers not paid much attention to the tobacco smoking related diseases. (6). Qualities of Indian cigarettes are far away from western standards (7). India is the 3rd largest producer and exporter of tobacco in the world. About 550 million kgs of tobacco is grown in 4.2 lakh hectares of land and 250 million kgs of tobacco is released for local consumption (8). In India 337 million people above 10 years of age consume tobacco. Every year 1 million people die prematurely due to tobacco smoking related diseases (9).

The mechanism by which smoking increases the cardiovascular diseases are unclear. Recently it has been suggested that smoking adversely affects the concentration of plasma lipids and lipoprotein levels. However studies to date have revealed incomplete, inconclusive or conflicting results about the association of smoking on the plasma lipids and lipoproteins (10). It has been estimated that 1% increase in plasma concentration is associated with a 2.7% increase in risk (11).

As tobacco is grown more in northern Karnataka and also due to paucity of work done in this part the present study was undertaken. The present study provides a detailed profile of the plasma lipid and lipoprotein levels depending on duration and intensity of smoking.

## II. Materials and Methods

The present study composed of 100 selected age and sex matched smokers and non-smokers between the age group of 20 to 60 years. All the subjects were consuming vegetarian diet and few of them were taking non-vegetarian diet occasionally, and belonging to different walks of the community. The subjects were volunteer participants in the study and gave informed consent.

All subjects were evaluated and selected by detailed medical history, physical examination, systemic examination and routine investigations to rule out any underlying diseases. Subjects having diseases, which are known to influence the blood lipids or patients on lipid lowering drugs or a diet restriction for any reason and persons chewing tobacco, ex-smokers, obese persons, alcoholics and having risk factors like hypertension, diabetes mellitus were excluded from the present study. Each patient gave informed consent and the study was approved by ethical and research committee of J.N. Medical College, Belgaum.

The present study comprises of 2 groups

Group I- Non smokers (Control) n=25

Group II – Cigarette smokers n = 75

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Cigarette smokers (Group –II) were divided into 3 subgroups depending upon duration and intensity of smoking.

Each group comprises about 25 volunteers

Group II A - Mild smokers (n =25)

(Duration - 1 to 5 years, smoking 10- 15 cigarettes / day)

Group II B - Moderate smokers (n=25)

(Duration 6 to 10 years, smoking 16-20 cigarettes / day)

Group II C - Heavy smokers (n= 25)

(Duration - more than 10 years, smoking >20 cigarettes/day)

In order to ensure accurate and reproducible results overnight 12-14 hours fasting blood samples were collected from these subjects. Serum was separated by centrifugation at 3600 rpm for six minutes. The clear serum samples were employed for the estimation of total cholesterol (12), Triglycerides (13) and HDL-Cholesterol (14). The levels of LDL cholesterol and VLDL cholesterol were calculated by using Friedewalds formula (15).

LDL Cholesterol (mg%) = Total Cholesterol – (HDL cholesterol + TG/5)

VLDL Cholesterol (mg%) = TG/5

The significance level of different parameters between the study groups were carried out using students "t" test.

## III. Results

The present study was composed of 25 healthy non-smokers as controls and 75 active smokers between the age group of 20 to 60 years as the test group. Depending upon the duration and intensity of smoking, cigarette smokers were divided into 3 sub groups of mild, moderate and heavy smokers, as stated above.

The results of the present study are given in table-1 and graph-1. Table-1 gives the levels of total cholesterol, TG, HDL-C, LDL-C and VLDL-C in normal (Group- I), in mild smokers (Group-II A), in moderate smokers (Group –II B) and in heavy smokers (Group –II C). Graph-I depicts the comparison of the parameters in different test groups (Group- IIA, II B and IIC).

As it is evident from the table-1 and graph-1, the intensity and duration of smoking shows a significant increase in levels of cholesterol, triglyceride, LDL-C, VLDL-C in almost all the groups of cigarette smokers as compared to non smokers. Simultaneously a significant reduction in level of HDL-C is observed in cigarette smokers as compared to non-smokers and a parallel increase in these parameters with the increase in intensity and duration of smoking.

*Table 1 :* Table showing serum lipid profile in cigarette smokers and non-smokers in relation to duration and intensity of smoking.

| PARAMETERS | NONSMOKERS | CIGARETTE SMOKERS | | |
|---|---|---|---|---|
| | GROUP – I (n=25) | GROUP –II A (n=25) | GROUP –II B (n=25) | GROUP –II C (n=25) |
| Total Cholesterol (mg%) | 155.28 ± 24.09 | 196.38 ±18.54 P<0.001 | 202.78 ± 20.56 P<0.001 | 214.13 ± 26.98 P<0.001 |
| Triglycerides (mg%) | 121.20 ± 32.70 | 167.78 ± 25.41 P<0.001 | 171.57± 32.42 P<0.001 | 191.16 ± 35.45 P<0.001 |
| HDL-Cholesterol (mg%) | 46.90 ± 6.71 | 38.63 ± 3.06 P<0.001 | 35.46 ± 3.50 P<0.001 | 31.64 ± 2.56 P<0.001 |
| LDL-Cholesterol (mg%) | 84.08 ± 24.42 | 124.01 ± 18.12 P<0.001 | 133.50 ± 21.76 P<0.001 | 143.87 ± 24.32 P<0.001 |
| VLDL-Cholesterol (mg%) | 24.24 ± 6.54 | 33.78 ± 4.91 P<0.001 | 34.35 ± 6.53 P<0.001 | 38.62 ± 7.34 P<0.001 |

*Values are expressed as Mean ± SD. All P values are in comparison with nonsmokers.*



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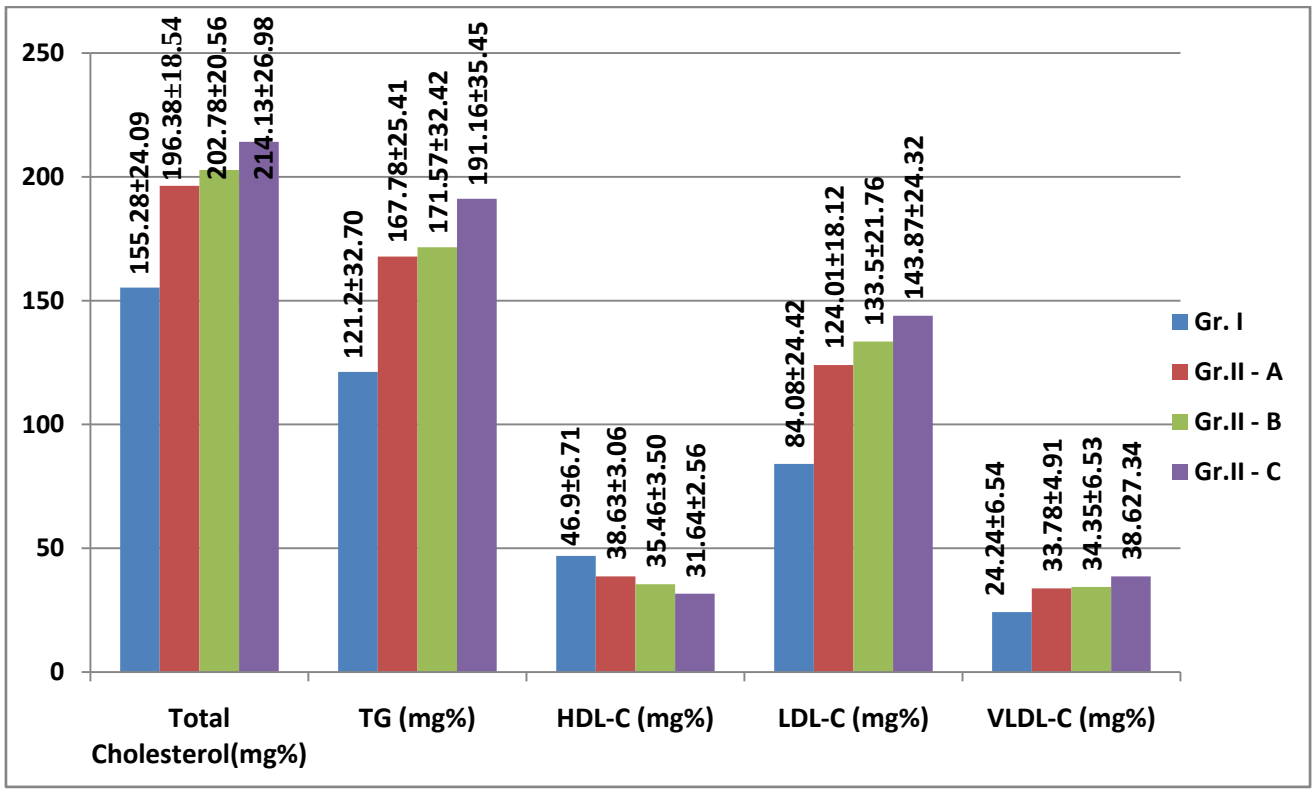
*Graph 1 :* Graph showing Lipid profile in cigarette smokers and non-smokers in relation to duration and intensity of smoking.




## IV. Discussion

Cigarette smokers have a high risk of coronary heart disease than nonsmokers. Several possible explanations have been offered for this association altered blood coagulation, impaired integrity of the arterial walls, changes in the blood lipid and lipoprotein concentration.

Smoking promotes CHD and atherosclerosis. This may be due to nicotine in cigarette smoke causes an increase in myocardial oxygen requirement by increasing the use of free fatty acid and also smoking by an unknown mechanism lowers the antiatherogenic factor HDL– C, remains a significant independent predictor of coronary artery disease.

In our study the mean value of serum total cholesterol in cigarette smokers is significantly higher ($P<0.001$) as compared to nonsmokers (refer Table -1). It is observed that cholesterol levels are raised in all groups of cigarette smokers but the risk is more in heavy smokers.

Analyzing the results with regard to the duration of smoking it is observed that on the whole there is a significant increase in the level of serum cholesterol with regard to an increase in duration and intensity of cigarette smoking.

The mean values of serum LDL-C and VLDL-C are observed to be significantly high ($P < 0.001$) in all groups of cigarette smokers (refer Table -1).

The present study also showed a significant increase ($P<0.001$) in serum triglycerides in cigarette smokers as compared to non-smokers (refer Table -1.)

The P values obtained with regards to all fractions of serum lipid profile are found to be highly significant in smokers who smoked more number of cigarettes as compared to nonsmokers.

The same characteristics are analyzed with regard to duration of smoking again a significant increase in VLDL-C, LDL-C, triglyceride and total cholesterol is noted in almost all groups of cigarette smokers as compared to nonsmokers. On the whole, a significant reduction in the level of HDL-C is observed in cigarette smokers smoking for longer duration.

There are contradicting and varying results regarding total cholesterol, TG, LDL-C and VLDL-C in smokers. An increase in the total cholesterol, TG, LDL-C and VLDL-C and a significant decrease in HDL-C found in the present study in smokers as compared to nonsmokers agrees with earlier reports (16-20). Further parallel increase is seen in these parameters in mild to heavy smokers (refer table-I). The rise in blood lipid levels in smokers may be through catecholamine and adenyl cyclase axis induced tissue lipolysis as suggested in chart -1.



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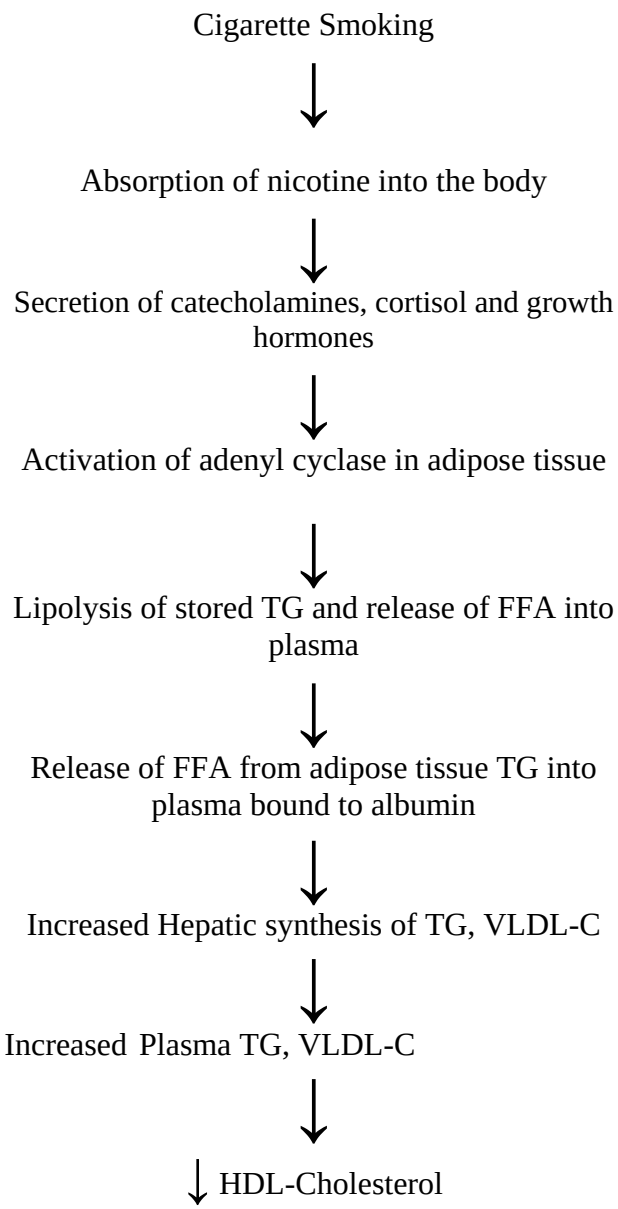
Majos O. D. et al. (21) in their study reported that there is significant decrease in HDL-C, but there is no change in total cholesterol and triglycerides in cigarette smokers as compared to non-smokers. The above findings, except for decrease in HDL-C are contradictory to our findings.

Chart -1 : Chart showing a possible mechanism by which nicotine absorbed from cigarette smoke may elevate plasma lipids and lipoproteins.




## V. Conclusion

Our study clearly shows a strong relationship between elevation of serum lipids and cigarette smoking. It also emphasis that the changes in the serum lipids tends to be high with the increase in duration and intensity of smoking. The risk of increase in serum cholesterol with an increase in LDL-C and decrease in HDL-C assume a great significance since this has been the pattern associated with CHD.

The low level of HDL-C in cigarette smokers and the increased exposure of the vascular endothelium to potentially atherogenic lipoproteins as a consequence of impaired clearance of triglyceride rich lipoproteins may provide a mechanism whereby smoking predisposes to greater risk of developing atherosclerotic plaques and CHD.

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