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Original Article

A Study of the Effect of Cigarette Smoking on Lipid Profile of People of Kolhan Region

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Abstract

Introduction: Cigarette smoking is an important and independent risk factor of atherosclerosis, coronary artery disease, and peripheral vascular disorders. It adversely affects the concentration of the plasma lipids and lipoprotein levels.

Materials and Methods: The present study was conducted in the Department of Physiology, Mahatma Gandhi Memorial Medical College and Hospital, Jamshedpur. Sixty healthy cigarette smokers were compared with forty healthy age-matched, non-obese, non-smokers. Participants in both the groups were in the age range of 25–35 years having no history of alcohol abuse or diseases such as diabetes mellitus and hypertension.

Conclusion: Total cholesterol and low-density lipoprotein (LDL) showed a highly significant upper range in smokers with $P < 0.001$. High-density lipoprotein and very LDL were also increased to a partially significant level in smokers with $P < 0.1$.

Key words: Coronary artery disease, High-density lipoprotein, Low-density lipoprotein, Total cholesterol, Triglyceride, Very low-density lipoprotein

INTRODUCTION

Smoking is one of the most potent and prevalent addictive, influencing behaviors of human beings for over four centuries. It in different forms is a major risk factor for atherosclerosis and coronary heart disease.[1] There is a dose–response relationship between the number of cigarette/bidi smoked and cardiovascular morbidity and mortality.[2,3] Smoking leads to increase in the concentration of serum total cholesterol (TC), triglyceride (TG), low-density lipoprotein-cholesterol (LDL-C), very LDL-C (VLDL-C), and fall in the level of antiatherogenic high-density lipoprotein-cholesterol (HDL-C) as reported by various workers.[4] Various mechanisms leading to lipid alteration by smoking are as follows:

a. Nicotine stimulates a sympathetic, adrenal system leading to increased secretion of catecholamines resulting in lipolysis and increased concentration of plasma-free fatty acids (FFAs), which further results in increased secretion of hepatic FFAs and hepatic TG along with VLDL-C in the blood stream[2,3]
b. Fall in estrogen levels occurs due to smoking, which further leads to decreased HDL-C[5]
c. Presence of hyperinsulinemia in smokers leads to increased cholesterol, LDL-C, VLDL-C, and TGs due to decreased activity of lipoprotein lipase[6]
d. Consumption of a diet rich in fat and cholesterol as well as a diet low in fiber and cereal content by smokers as compared to non-smokers.[7]

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MATERIALS AND METHODS

Blood samples were obtained after an overnight fast. About 5 ml of blood was collected from the left antecubital vein, of which about 2 ml is transferred into an overfast vial and mixed well and centrifuged at a speed of 3000 revolutions per minute for 10 min to separate the plasma, which was used for biochemical analysis. Rest 3 ml of blood is transferred to the test tube, and this blood was allowed to clot to get serum. This serum was separated in a centrifuge tube at 3000 revolutions per min to get a clear

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sample of serum. This clear supernatant serum was used for biochemical investigation.

Estimation of Serum TC

Method – Enzymatic colorimetric Trinder end point.

The reagents were allowed to attain room temperature prior to use.

Pipette into tube marked	Blank (µL)	Standard (µL)	Sample (µL)
Reagent R	1000	1000	1000
Standard	--------	10	--------
Sample	--------	-------	10

They were incubated for 5 min at 37°C; the reading was done against blank at 500 nm, and calculation was made. The concentration of cholesterol in the sample is directly proportional to the intensity of red complex (red Quinone), which was measured at 500 nm.

Calculation

Cholesterol = Absorbance of sample/absorbance of standard × Concentration of standard.

Estimation of Serum TG

Method – Enzymatic colorimetric method.

Contents were mixed and incubated for 5 min at 37°C. The reading was done against blank at 546 nm.

Pipette into tube marked	Blank (µL)	Standard (µL)	Sample (µL)
Reagent R	1000	1000	1000
Standard	--------	10	-------
Sample	--------	-------	10

Calculation

Serum TG = Absorbance of sample/Absorbance of standard × n.

n = Standard concentration
Reference values: >150 mg/dl.

Estimation of HDL-C

Method – Phosphotungstate method.

Principle – chylomicrons, LDL, and VLDL are precipitated by addition of phosphotungstic acid and magnesium chloride. After centrifugation, the HDL fraction remains in the supernatant, which is determined with CHOD-PAP method.

Reference value: >40 mg/dl.

Table 1: Lipid-level variation among smokers and non-smokers (mean+SD) in mg/dl

Types of lipids	Smokers (*n*=60)	Non-smokers (*n*=40)	*t*	*P*	Significance
TC	192.51+18.94	181.29+19.20	4.01	<0.0001	HS
TG	167.49+16.74	155.14+15.41	5.17	<0.001	HS
HDL	37.66+3.35	45.36+7.18	2.78	<0.1	PS
LDL	120.94+18.18	104.88+19.56	5.85	<0.001	HS
VLDL	33.50+3.35	31.03+3.08	1.08	<0.1	PS

TC, TG, and LDL showed a highly significant upper range in smokers. HDL and VLDL were also increased to a partially significant level in smokers. TC: Total cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL:Very low-density lipoprotein

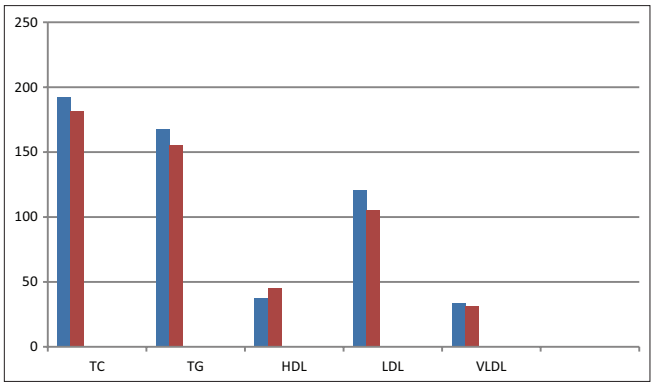

Figure 1: Lipid-level variations among smokers and non-smokers (mean in mg/dl)

Calculation of LDL and VLDL by Friedewald's Formula:

LDL = TC − (HDL + VLDL),

VLDL = TG/5,

Reference value:
LDL = Up to 190 mg/dl
VLDL = 14–31.8 mg/dl.

RESULT AND OBSERVATION

Table 1 and Figure 1 shows lipid level variations among smoker and non- smoker. Definitely smokers show highly significant variations in lipd levels from non- smoker. Smokers have higher level of TC, TG and LDL while HDL and VLDL is partially significant.

CONCLUSION

Our study clearly shows a strong relationship between elevation of serum lipids and cigarette smoking. It is revealed that Total cholesterol, triglyceride and LDL were significantly higher in smokers as compared to non-smokers. Smoking generates oxidised platelet activity

factor derivatives. These stimulate interaction between platelets- leucocytes and endothelial cell and thus augment inflammation and thrombosis.[8] A high level of LDL-C, TG, and TC is strongly associated with development of coronary artery disease, whereas a low level of HDL remains a significant independent predictor of coronary artery disease.[9,10]

REFERENCES

1. Wilhelmsen L. Coronary heart disease: Epidemiology of smoking and intervention studies of smoking. Am Heart J 1988;115:242-9.
2. Carlson LA, Böttiger LE, Ahfeldt PE. Risk factors for myocardial infarction in the Stockholm prospective study. A 14-year follow-up focussing on the role of plasma triglycerides and cholesterol. Acta Med Scand 1979;206:351- 60.
3. Muscat JE, Harris RE, Haley NJ, Wynder EL. Cigarette smoking and plasma cholesterol. Am Heart J 1991;121:141-7.
4. Kannel WB. Update on the role of cigarette smoking in coronary artery disease. Am Heart J 1981;101:319-28.
5. Benowitz NL. Drug therapy. Pharmacologic aspects of cigarette smoking and nicotine addiction. N Engl J Med 1988;319:1318-30.
6. Stalder M, Pometta D, Suenram A. Relationship between plasma insulin levels and high density lipoprotein cholesterol levels in healthy men. Diabetologia 1981;21:544-8.
7. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988;37:1595-607.
8. Zimmerman M, McGeachie J. The effect of nicotine on aortic endothelial cell turnover. An autoradiographic study. Atherosclerosis 1985;58:39-47.
9. Report of the national cholesterol education program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. The expert panel. Arch Intern Med 1988;148:36-69.
10. Stampfer MJ, Sacks FM, Salvini S, Willett WC, Hennekens CH. A prospective study of cholesterol, apolipoproteins, and the risk of myocardial infarction. N Engl J Med 1991;325:373-81.

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