



Effect of Smoking on Serum Lipid Levels: A Hospital-based Study

¹Arjun S Shenoi, ²Vijay Kadam, ³Jaishree Ghanekar

ABSTRACT

Aim: To study the effect of smoking on the levels of serum cholesterol, serum triglycerides, HDL-C and LDL-C in men between the age group of 20 and 30 years.

Materials and methods: The study recruited 50 smokers and 50 controls. Controls were age, BMI and dietary habits matched nonsmokers. The variables compared between the two groups were total cholesterol, triglycerides, LDL-C, HDL-C. dyslipidemia with relation to the severity of smoking was studied in smoker group.

Results: The present study showed statistically significant differences in all variables in two groups ($p < 0.001$). Smokers as compared to nonsmokers had higher total cholesterol (189.14 ± 19.888 vs 159.22 ± 23.221 mg/dl), triglycerides (149.98 ± 32.068 vs 116.86 ± 19.937 mg/dl) and LDL-C (121.14 ± 18.212 vs 93.28 ± 23.726 mg/dl). While the mean HDL-C was lower in smokers as compared to nonsmokers (38.02 ± 4.515 vs 42.56 ± 3.215 mg/dl).

Conclusion: The prevalence of dyslipidemia in both smokers as well as nonsmokers according to the NCEP ATP III guidelines was 40% in smokers with just 18% in that of nonsmokers group. The study also showed a linear increase in the values of total cholesterol, triglycerides and LDL-cholesterol with the severity of smoking which was statistically significant. While HDL-C levels linearly decreased when progressing through pack years.

Abbreviations: BMI: Body mass index; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; NCEP ATP III: National Cholesterol Education Program, Adult treatment panel III; VLDL-C: Very low density lipoprotein-cholesterol; FFA: Free fatty acids.

Keywords: Lipid profile, Smoking, Smoking cessation.

How to cite this article: Shenoi AS, Kadam V, Ghanekar J. Effect of Smoking on Serum Lipid Levels: A Hospital-based Study. *MGM J Med Sci* 2015;2(1):13-15.

Source of support: Nil

Conflict of interest: None

¹Resident, ²Assistant Professor, ³Professor and Head

^{1,3}Department of Medicine, MGM Medical College and Hospital Navi Mumbai, Maharashtra, India

²Department of Cardiology, MGM Medical College and Hospital Navi Mumbai, Maharashtra, India

Corresponding Author: Arjun S Shenoi, Resident, Department of Medicine, MGM Medical College and Hospital, Sector-1, Kamothe Navi Mumbai-410209, Maharashtra, India, Phone: 8693097444 e-mail: arjun.shenoi@gmail.com

INTRODUCTION

Smoking has been practiced in one form or the other since ancient times, dating back to 6000 BC where tobacco was first grown in America. Coronary artery disease and cerebrovascular disease are the leading causes of mortality and morbidity in most parts of the world including India.¹ Atherosclerosis is the major lesion leading to these diseases.²

Cigarette smoking is a well-established risk factor for atherogenesis and its sequelae—coronary artery disease, cerebrovascular disease and peripheral vascular disease.³ According to the lipid hypothesis, the oxidized LDL-C plays a key role in atherogenesis. High level of serum cholesterol have been found to be associated with increased mortality due to vascular diseases.⁴ High level of LDL-C and triglycerides are strongly associated with the occurrence of coronary artery disease while low level of HDL-C remains a significant independent predictor of coronary artery disease at any level of LDL-C⁵⁻⁷

MATERIALS AND METHODS

A cross-sectional study was carried out at MGM Medical College and Hospital, Navi Mumbai, Maharashtra, India, over a period of one and half year, after receiving approval from the Institutional Ethics Review Committee (IERC).

Fifty smoker and 50 nonsmoker males in the age group of 20 to 30 years were enrolled.

The exclusion criteria included subjects with familial dyslipidemic disorders; subjects taking drugs affecting serum lipid level and subjects having disease or disorder affecting serum lipid levels, like diabetes mellitus, hypertension, hypo or hyperthyroidism, alcoholism, tuberculosis.

All smokers were classified on the basis of pack years as having smoked for: <1 pack year; 1 to 2 pack years and >2 pack years.

A pack year was taken as a history of smoking a pack containing 20 cigarettes or equivalent per day for 1 year.

A detailed history was elicited followed by clinical examination. All patients were examined carefully for evidence of dyslipidemia. Routine baseline investigations were done in all cases. Instructions were given to subjects to take a normal diet before lipid profile estimation was done. Venous blood from the antecubital vein was drawn

8 to 10 hours after fasting. A complete lipid profile was carried out which included total cholesterol, serum triglycerides, HDL, LDL levels.

RESULTS

The present study was undertaken with the aim of providing a quantitative estimation of the lipid profile in smokers and nonsmokers and comparing the obtained data with literature already published (Fig. 1). In our study, we analyzed the extent to which the collected data supports the relationship between cigarette smoking and altered serum lipid profile in smokers.

The total cholesterol was estimated using the CHOD-PAP method. The mean serum cholesterol for smokers was 189 ± 19.88 mg/dl. In the control group, the mean total cholesterol was 159.24 ± 23.221 mg/dl. The mean LDL-C levels for smokers were 121.14 ± 18.212 mg/dl as compared to the control group with mean levels of 93.28 ± 23.726 mg/dl. The mean HDL-C for smokers in the present study was 38.02 ± 4.515 mg/dl. In the control group, the mean HDL-C was 42.56 ± 3.25 mg/dl. All the findings were found to be statistically significant.

The severity of smoking was assessed according to pack years. The smokers were divided into three groups: <1 pack year; 1 to 2 pack years and >2 pack years.

There was a linear increase in the values of total cholesterol, triglycerides and LDL-C as one progressed through the three groups suggesting a dose response relationship between severity of smoking and derangement in lipid levels which was statistically significant for total cholesterol and LDL-C. Similarly, HDL-C levels linearly decreased when progressing through pack years again suggesting a dose response relationship between severity of smoking and decrease in LDL-C levels (Fig. 2 and Table 1).

DISCUSSION

Cigarette smoke contains more than 4000 different chemicals, which include components, like nicotine, tar, carbon monoxide, hydrogen cyanide well as various irritants. There have been a number of studies done on the relationship between cigarette smoking and its effects on lipids, mainly being international and a few Indian.⁸⁻¹¹

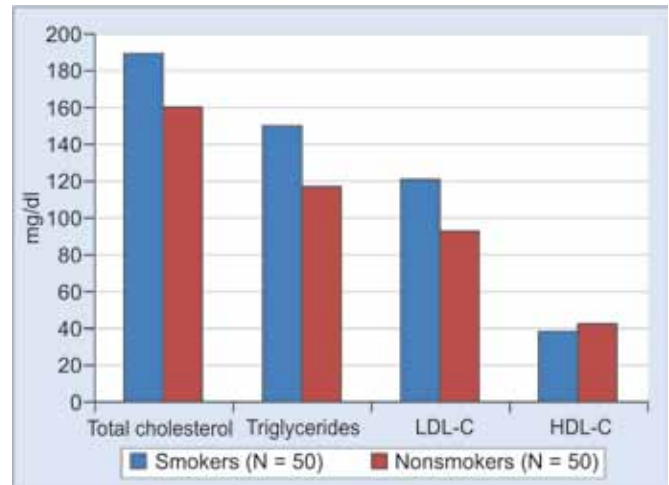


Fig. 1: Lipid profile of smokers vs nonsmokers

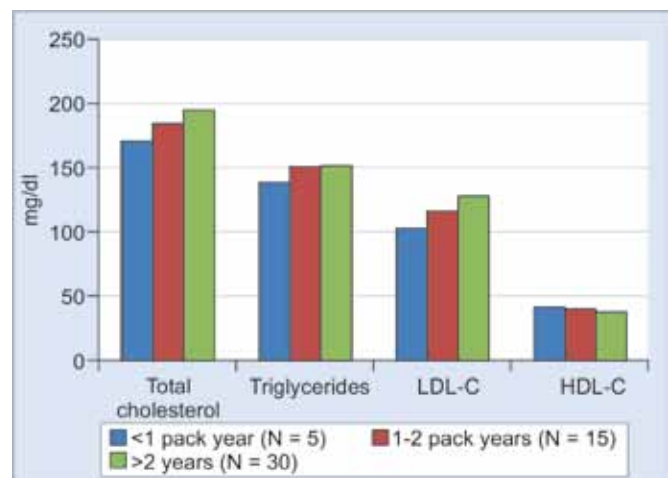


Fig. 2: Lipid profile with relation to severity of smoking

The ill-effects caused by smoking are legion and documented prior. Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increase secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma-free fatty acids (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL-C in the blood stream,¹² (b) fall in estrogen levels occurs due to smoking which further leads to decreased HDL-C¹³ and (c) presence of hyperinsulinemia in smokers leads to increased cholesterol, LDL-C, VLDL-C, and TG due to decreased activity of lipoprotein lipase.¹⁴

Table 1: Lipid profile with relation to severity of smoking

Parameter (mg/dl)	Present study		
	<1 pack year (n = 5)	1-2 pack years (n = 15)	>2 pack years (n = 30)
Total cholesterol	169.4 ± 15.388	184.3 ± 20.089	194.9 ± 18.05
HDL-C	40.0 ± 4.0	38.93 ± 6.766	37.23 ± 2.897
LDL-C	101.8 ± 16.88	114.93 ± 19.207	127.4 ± 14.73
Triglycerides	138.2 ± 6.181	151.53 ± 32.73	151.1 ± 32.73



It is a proven fact that smokers have a higher risk of vascular disease including coronary heart disease and cerebrovascular disease than nonsmokers. There have been many explanations which have been put forward for this association including altered blood coagulability, decreased fibrinolysis, impaired integrity of the endothelial wall and changes in blood lipids and lipoprotein concentrations.^{15,16} Also, cigarette smoke has been identified as a carcinogen with increased risk of cancer of oral, naso oropharynx, larynx, gastrointestinal malignancies, urogenital malignancies. It is responsible for 90% of chronic obstructive pulmonary diseases and is detrimental to maternal and infant health.

CONCLUSION

Smoking of tobacco is a vice, which is harmful not only to the person who smokes but also to the innocent bystander who unfortunately happens to share the same air as the smoker. There has been a growing interest in the effects of smoking on the lipid profile as it has become clear that dyslipidemia is a major risk factor for atherosclerosis and vascular disease. It is a proven fact that smokers have a higher risk of vascular disease including coronary heart disease and cerebrovascular disease than nonsmokers due to an adverse lipid profile in these patients.

A NOTE ON CIGARETTE CESSATION

Smoking is the foremost preventable cause of death. Smoking cessation is a dynamic cyclic process that leads to overcoming addictive behavior. A combination of counseling and pharmacological therapy when necessary to help quit smoking, including nicotine gum, patch, bupropion, varenicline, etc.

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