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Effect of Cigarette Smoking on Serum Lipid Profile in Male Population of Udaipur (Rajasthan)

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Abstract

Cigarette Smoking is associated with adverse effects on lipid profile and Homocysteine thus increasing risk for atherosclerosis and coronary heart disease. Smoking is a prominent risk factor for coronary artery disease, atherosclerosis and peripheral vascular disorders. This study was undertaken to evaluate serum lipid profile in chronic smokers and to compare it with healthy non-smokers, considered as controls. Serum lipid profile was measured in 300 male subjects. Out of which 150 were smokers and 150 non-smokers (controls) with an age range of 50 to 60 years. Only chronic smokers who were smoking for more than 20 years were included in the study. It was revealed that mean serum Total Cholesterol (268.88±29.23 mg/dl), Triglyceride (192.12±56.42 mg/dl), Low Density Lipoprotein Cholesterol (189.76±15.74 mg/dl), Very Low Density Lipoprotein Cholesterol (38.42±11.28 mg/dl) were significantly higher in chronic smokers as compared to non-smokers with mean serum Total Cholesterol (182.56±21.33 mg/dl), Triglyceride (115.71±32.11mg/dl), Low Density Lipoprotein Cholesterol (107.68±9.55 mg/dl), Very Low Density Lipoprotein Cholesterol (23.14±6.42 mg/dl). On the other hand value of mean serum High Density Lipoprotein Cholesterol was lower in chronic smokers (40.7±2.21 mg/dl) than in non-smokers (51.74±5.36 mg/dl). Thus this study concludes that cigarette smoking produced adverse effects on lipid profile, leading to increase cardiovascular disease risk among smokers.

Keywords: Dyslipidaemia, Smokers, Cardiovascular Diseases

Introduction

Smoking is one of the most potent and prevalent addictive habits, influencing behaviour of human beings. Smoking is now increasing rapidly throughout the developing world and is one of the biggest threats to current and future world health. Nearly 20% of all coronary heart disease deaths can be attributed to smoking[1,2]. Cigarette smoking is a prominent risk factor for coronary artery disease, atherosclerosis and peripheral vascular disorders. Smoking is associated with a more atherogenic lipid profile[3-5]. It increases the concentration of serum total Cholesterol, triglycerides, LDL-Cholesterol, VLDL-Cholesterol and decreases the level of good Cholesterol i.e. HDL-Cholesterol[6-10]. Thus, smoking is a major risk factor for atherosclerosis and coronary artery disease[11,12]. Various mechanisms leading to lipid alteration by smoking are: (a) nicotine results in increased secretion of hepatic free fatty acids and triglycerides along with VLDL-C in the blood stream by increasing the secretion of catecholamines and thus stimulating sympathetic adrenal system resulting in increased lipolysis[13]; (b) consumption of a diet lacking in fibre and cereal content but enriched with fat and cholesterol by smokers as compared to non-smokers[14]; (c) cigarette smoking is known to be associated with raised plasma Homocysteine level (15,16) which causes oxidative modification of LDL-Cholesterol and decreases HDL-Cholesterol[17], several studies reported homocysteine inhibited Apo A-I protein expression and decreased HDL Cholesterol[18,19].

The aim of this study was to investigate serum lipid profile pattern in male chronic smokers of Udaipur city.

Materials and Methods

The study was designed to compare values of lipid profile in chronic smokers smoking more than one pack or above daily vs non-smokers. A total number of 300 subjects were evaluated from April 2016 to July 2016, at Arth Diagnostic Private Limited, Udaipur. 150 male chronic smokers who were smoking for more than 20 years (one pack or above per day), with an age range of 50 to 60 years, were included for this study after obtaining written informed consent (Group I). 150 male non-smokers, whom age and weight was approximately matched with the subjects in (Group I) were recruited as controls (Group II). Controls were clinically healthy and from a similar background to cases as possible. The results were analysed for statistical significance by p value with 95% confidence interval.

The following criteria were used for exclusion:

1. Alcoholics
2. Ex-smokers
3. Diabetes mellitus
4. Renal disease
5. Hypertension
6. Previous and family history of coronary heart disease
7. Chronic hepatic dysfunction
8. Endocrine disorders and obesity
9. Lipid lowering drugs

Venous blood samples were collected after 12 hours of an overnight fast into plain tubes. Serum was obtained by centrifugation and samples were immediately separated into aliquot and stored at -20˚C until analysed. Total Cholesterol, Triglyceride, LDL-Cholesterol and HDL-Cholesterol levels were analysed on fully autoanalyser of Roche, Cobas Integra 400 Plus by

Enzymatic, colorimetric method. The analysis of Total Cholesterol, LDL-Cholesterol and HDL-Cholesterol was done by enzyme cholesterol esterase while determination of Triglyceride is based on the work by Wahlefeld using a lipoprotein lipase from microorganisms. The reference values were obtained from Manual of Roche Cobas Integra 400 Plus. The reference range of normal, borderline and high Total Cholesterol was <200, 200-250, >250 mg/dl respectively. The expected value for Triglyceride and LDL-C was <150 mg/dl. The expected value of HDL-C in males was 35-55 mg/dl. The level of VLDL-Cholesterol was calculated by using Friedewald's formula[20]. The expected value of VLDL-C was <30 mg/dl.

Results

As shown in Table 1 the lipid profile parameters such as Total Cholesterol, Triglyceride, VLDL-Cholesterol, LDL-Cholesterol were significantly higher in smokers as compared to non-smokers while this was reverse the case with HDL-Cholesterol. HDL-Cholesterol was significantly lower in smokers than in non-smokers.

Table 1

Name of Parameter	Smokers Group I	Non Smokers Group II (Control)	P value
	Mean±SD	Mean±SD	
Total Cholesterol mg/dl	268.88±29.23	182.56±21.33	<0.05
Triglyceride mg/dl	192.12±56.42	115.71±32.11	
VLDL-Cholesterol mg/dl	38.42±11.28	23.14±6.42	
LDL-Cholesterol mg/dl	189.76±15.74	107.68±9.55	
HDL-Cholesterol mg/dl	40.7±2.21	51.74±5.36	

Discussion

The risk for coronary heart disease is more in cigarette smokers than non-smokers. This may be explained by various associations like impairment in the integrity of arterial walls, derangements in the blood lipid and lipoprotein concentration, alterations in blood coagulation. This study revealed significantly high concentration of Total Cholesterol, Triglycerides, VLDL-C, LDL-C in chronic smokers (Group I) as compared to non-smokers (Group II) with the p-value <0.05 as shown in Table I. The present study also showed significant decrease in level of HDL-C ($p<0.05$) in chronic smokers than in non-smokers (refer Table 1). Similar results have been observed in the study on Lipid and lipoprotein profiles among middle aged male smokers of southern India in which level of Total Cholesterol, Triglyceride, LDL-C was significantly elevated and level of HDL-C was significantly reduced in smokers[21]. In another study[22] the same association is reported between smoking and level of HDL-C, however level of Triglyceride and LDL-C were not significantly high in smokers but in meta-analysis there was considerable increase in level of Triglyceride and LDL-C. In contrast our study showed significant increase in level of Total Cholesterol, Triglyceride and LDL-C. The rise in lipid levels in smokers may be explained by following mechanism: Cigarette smoking causes absorption of nicotine into the body which leads to lipolysis and release of free fatty acids into the bloodstream via activation of adenylcyclase in adipose tissue by nicotine stimulated secretion of catecholamines. These increased free fatty acids in liver give rise to increased hepatic Triglyceride and VLDL synthesis, thus increasing the concentration of Triglyceride and VLDL-C in blood.

The present study also showed significant decrease in level of HDL-C ($p<0.05$) in chronic smokers than in non-smokers (refer Table 1). Several studies reported high levels of plasma Homocysteine in chronic smokers (23). Plasma Homocysteine is negatively correlated with HDL-C and Apo A-I. Increase levels of Homocysteine may lead to decrease level of HDL-C by several mechanisms. Further decrease in HDL-C in chronic smokers may also be explained by smoking induced increase catecholamine release, causing increase in VLDL-C and decrease in HDL-C concentrations. Thus smoking promotes Coronary Heart Disease and atherosclerosis by lowering the anti-atherogenic factor HDL-C and increasing the potentially atherogenic lipoproteins LDL-C which further may lead to vascular endothelium damage.

Conclusion

This study clearly reveals a strong relationship between cigarette smoking and increase in serum lipids. In chronic smokers the risk of increase in serum Cholesterol with an increase in LDL-Cholesterol and decrease in HDL-Cholesterol reflects a great significance since this is the finding associated with Coronary Heart Disease.

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