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

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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: Smoking; Total Cholesterol (TC); Triglyceride (TG); Low Density Lipoprotein Cholesterol (LDL-C); Very Low Density Lipoprotein Cholesterol (VLDL-C); High Density Lipoprotein Cholesterol (HDL-C)

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-Colesterol 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

A total number of 300 subjects were evaluated at Arth Diagnostic Private Limited, Udaipur. 150 male chronic smokers who were smoking for more than 20 years, 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 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. In case of total Cholesterol, the method is based on the determination of 4-cholestenone after enzymatic cleavage of the cholesterol ester by cholesterol esterase, conversion of cholesterol by cholesterol oxidase and subsequent measurement by the Trinder reaction of the hydrogen peroxide formed. The reference range of normal, borderline and high Total Cholesterol was <200, 200-250, >250 mg/dl respectively. The method of Triglyceride estimation is based on the work by Wahlefeld using a lipoprotein lipase from microorganisms for the rapid and complete hydrolysis of Triglycerides to glycerol followed by oxidation to dihydroxyacetone phosphate and hydrogen peroxide, This H_2O_2 is measured by Trinder endpoint reaction. The expected value for Triglyceride is <150 mg/dl. LDL-Cholesterol analysis depends on the cleavage of cholesterol ester into free cholesterol and fatty acids by cholesterol esterase. In presence of oxygen, cholesterol is oxidized by cholesterol oxidase to 4-cholestenone and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-aminoantipyrine and HSDA to form a purple-blue dye. The colour of this dye is directly proportional to the cholesterol concentration in LDL and is measured photometrically. The expected value of LDL-C is <150 mg/dl. The HDL-Cholesterol is determined enzymatically by cholesterol esterase and cholesterol oxidase coupled with polyethylene glycol to the amino groups (approximately 40%). Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by cholesterol esterase. In presence of oxygen, cholesterol is oxidized by cholesterol oxidase to 4-cholestenone and hydrogen peroxide. Hydrogen peroxide further forms dye, intensity of which determines HDL-Cholesterol concentration. 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.

Name of Parameter	Smokers Group I Mean ± SD	Non Smokers Group II (Control) Mean ± SD	P-value
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	<0.05
VLDL-Cholesterol mg/dl	38.42 ± 11.28	23.14 ± 6.42	<0.05
LDL-Cholesterol mg/dl	189.76 ± 15.74	107.68 ± 9.55	<0.05
HDL-Cholesterol mg/dl	40.7 ± 2.21	51.74 ± 5.36	<0.05

Table 1: Lipid profile parameters.

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 1. 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 blood stream via activation of adenyl cyclase 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 [21]. 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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