Original Article

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Study of Lipid Profile in Young Smokers and Non-Smokers

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Abstract

Background: Study of lipid profile in young smokers provides opportunity to explain the physiological consequences of the cigarette smoking activity. Cigarette smoking is the second cause of death in the world. The addictive liability and pharmacological effects of smoking are primarily mediated by the major tobacco alkaloid nicotine. Cigarette smoke may promote atherogenesis by producing oxygen-derived free radicals that damage lipids. Cigarette smoking is associated with impaired endothelium-dependent vasodilatation and cardiovascular disease (CVD). As the current report concerns solely to the study of lipid profile in normal healthy controls and smoking male subjects, the results of this study can be correlated with other biochemical, physiological and clinical aspects. *Aim:* The Aim of our study was to determine and compare lipid profile in smokers and non-smokers. *Method:* A Total No. of 100 subjects were selected, out of which 50 were healthy individual and 50 were young smokers less than 35 years of age. The lipid parameter were analyzed using kits on automated biochemistry analyzer. *Result:* The levels of total cholesterol, Triglyceride, LDL-C and VLDL-C were significantly higher while the levels of HDL was significantly lower in smokers as compared to their levels in non smokers.

Keywords: Lipid Profile; Smokers; Non-Smokers.

Introduction

Smoking may be explained as habit of inhalation of smoke arising from the burning of tobacco in a pipe or in the form of a cigar or cigarette. Nowadays the cigarette smoking is fashion but it causes many illnesses. Smoking is the major preventable cause of death in many areas of the world today. The World Health organization estimates that worldwide 5 million deaths are cause prematurely by smoking every year [1].

In India around, 5,500 adolescents start smoking cigarettes every day, by joining the 4 million young people, under the age of 15, who already smoke regularly. Like other developing countries, the most susceptible time for initiate smoking in India is during adolescence and early adulthood i.e 15 – 24 years [2].

A large number of risk factor which predispose to atherosclerosis and Coronary Artery Disease (CAD) have been identified. These include modifiable ones like hypertension, dyslipidemia, smoking, diabetes mellitus, changing life style and non modifiable ones like age and sex. As the number of risk factor in an individual increases, so does the risk of developing atherosclerosis and its complication mainly coronary artery disease (CAD). In subject with more than one of these risk factor the risk is more additive [3]. Although smoking has been established as an independent risk factor for Coronary Heart Disease [4], the mechanism by which it increases the risk of coronary heart diseases is unclear. Some

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explanations have been postulated: the increased carbon monoxide in the blood of cigarette smokers may damage the endothelium and accelerate the entry of cholesterol into the wall of the artery [5]. The formation of carboxyhemoglobin creates relative anoxemia in the tissue, including the myocardium [6].

Smoking enhances platelets aggregation, and the nicotine absorbed from cigarette smoke may induce cardiac arrhythmia through its pharmacologic action. Cigarette smoke contains numerous compounds, many of which are oxidants and prooxidants, capable of producing free radicals and enhancing the oxidative stress [7].

Cigarette smoke is a complex mixture of over 7000 chemical compounds [8,9]. Cigarette smoking predisposes the individual to several different clinical to several different atherosclerotic syndromes, including stable angina, acute coronary syndromes, asthma [9], sudden death and stroke.

It is associated with average 70 % increase in risk of death from coronary artery disease [10]. An additional mechanism has been recently suggested that smoking adversely affect the concentration of the plasma lipids and lipoproteins. However, studies to date have revealed incomplete, inconclusive or conflicting results about the association of smoking on the plasma lipid and lipoprotein levels. In some studies, smokers had increased plasma cholesterol in other plasma cholesterol level have actually been lower only a few studies have specifically examined the plasma lipoprotein according to smoking status or number of cigarettes smoked (dosage) Smokers are reported to have higher low density lipoprotein (LDL) and lower high density cholesterol levels than non smokers [11].

Aim & Objectives

1. Present study has been taken up to find out the alteration of serum lipid profile between young smokers & non smokers & also to see any dose related changes in serum lipid among the smoking population.
2. To create awareness among young smoker population as well as young non-smoker population decrease the burden of adverse effects of smoking inn society.

Material & Methods

Study Design: The current Prospective study was undertaken from Aurangabad; Maharashtra.

Study Period: Dcember 2016 to December 2017.

Ethical Approval: The study was approved by the Institutional Ethical Committee of IIMSR Medical College Jalna.

Inclusion

We labeled smoker as the one who smokes atleast 10 cigarettes per day [12]. The study composed of 100 male smokers and nonsmokers between the age group of 20 to 35 years. All the subjects were consuming both vegetarian diet and non vegetarian diet, and belonging to different walks of community. The subjects were volunteer participants in the study and gave informed consent.

Exclusion

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.

Site of Sample Collection

Samples were collected Medicine and TB Chest OPD of IIMSR Jalna.

Site of Sample Study: Central Clinical laboratory (Biochemistry section), IIMSR Jalna.

Study Subjects

Group I Non Smokers (Control) n = 50

Group II Cigarette Smokers n=75.

Method

In order to ensure accurate and reproducible results overnight 12 hours fasting blood samples were collected from these subjects. Serum was separated by centrifugation at 3600 rpm for six minutes. The clear serum sample were employed on fully automated biochemistry analyzer for estimation of LDL , VLDL , Cholesterol , Triglycerides & HDL. Statistical analysis was done by using Chi square test by calculating p value with the help of SPSS software. Difference between the parameters of two groups was considered significant if $p < 0.001$.

Table 1:

Sr. No.	Parameters (mg/dl)	Normal Range	Non-Smokers (Mean+/- SD)	Smokers. (Mean+/- SD)	P value
1.	Mean Total Cholesterol	150-250 mg/dl	194.11+/-2.97	212.37+/-33.00	<0.001
2.	Mean HDL	40-60 mg/dl	37.86 +/-4.93	29.06 +/-2.98	<0.001
3.	Mean LDL	90-140 mg/dl	126.74 +/-14.54	153.54+/- 30.16	<0.001
4.	Mean VLDL	0-40 mg/dl	22.09+/-5.06	30.17 +/-7.13	<0.001
5.	Mean Triglycerides	60-170 mg/dl	116.37+/-29.02	142.14 +/-36.11	<0.001

Results

The levels of total cholesterol, Triglyceride, LDL-C and VLDL-C were significantly higher while the levels of HDL was significantly lower in smokers as compared to their levels in non smokers.

Discussion

In this study we found that total cholesterol and triglycerides levels in smokers were significantly higher than that in non-smokers. Similar findings were shared by some of the studies [13]. However in some other studies no significant changes were observed in cholesterol levels in smokers and non smokers [14].

The reason for increased serum total cholesterol and Triglyceride levels in smokers are increased catecholamine secretion leading to lipolysis , which in Turn elevates hepatic output of Triglycerides and VLDL [15], hyperinsulinemia leads to decreased activity of lipoprotein lipase [16], increased activity of hepatic lipase and decreased lecithin Cholesterol acyl transferase (LCAT) activity [17]. The mean levels of LDL–C and VLDL – C in this study among smokers were higher than non smokers. These findings were similar to other studies [13,18].

The mechanism of increased LDL- C and VLDL- C levels in chronic smokers are due to hyperinsulinemia leading to decreased activity of lipoprotein lipase [16] and increased activity of cholestryl ester transfer protein (CETP) and phospholipid transfer protein. Decrease in HDL- C level in smokers compared to non smokers was also significant.

This findings is similar to me other study findings as well[13] However some other studies have reported no significant difference in HDL – C level between smokers and non smokers. The explanations cited for low levels of HDL – C in smokers are due to low levels of estrogen leading to fall in HDL-C level and increased activity of cholestryl ester transfer protein (CETP) [19].

Conclusion

Finding of this study concludes that compare with non smokers the young smokers developed Dyslipidemia and are thus at higher risk of cardiovascular diseases although some more cardiovascular parameters needs to be studies and compared to confirm increased CVD risk in young smokers.

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