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A COMPARATIVE STUDY ON ALTERATIONS OF LIPID PROFILE AMONG SMOKERS AND NON SMOKERS

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ABSTRACT:

Smoking is one of the important etiological and independent risk factor for coronary artery disease. Increased carbon monoxide in the blood may damage the endothelium this accelerate the entry of cholesterol into the wall of the artery, carboxyhemoglobin which will be formed creates relative anoxemia in the tissue, including the myocardium and smoking enhances platelets aggregation. The study subjects were divided into 2 groups i.e. Group-I (Smokers, n=50) and Group-II (Controls, n=50). Both cases and controls were in age group of 18 to 55 years. Serum lipid profile was analyzed in all the subjects using the end-point method by Mindray - BS-380 autoanalyzer. The present study shown statistically significant decreased serum levels of High Density Lipoprotein Cholesterol (HDL-C) in group-I compared with group-II. The increase of serum Total Cholesterol (TC), Triglycerides (TG) and Low Density Lipoprotein Cholesterol (LDL-C) and Very Low Density Lipoprotein Cholesterol (VLDL-C) levels in group-I when compared with group-II. And this increase leads to the dyslipidemia. Smoking causes various complications including, Cardiovascular- Ischemic Heart Disease. Respiratory-COPD, Asthma, Pneumonia. Others- Cataracts, Periodontitis, Low bone density, Hip fractures. Reproduction-Fetal death, Stillbirths, Delay conception. Cancer - Oropharynx ,Laryngeal, Esophageal, Lung, Cervical, Kidney, Bladder, Leukemia, Pancreatic, Stomach. The alteration of lipid profile in tobacco smokers have raised serious medical concern with respect to atherogenic risk and recommendation for counseling the tobacco smokers to quit tobacco smoking and routine evaluation of serum lipid profile is suggested.

Keywords: Tobacco Smoking, Lipid Profile, Dyslipidemia, Ischemic Heart Disease.

Introduction

Cigarette smoking is generally considered as associated with increased risk of a variety of medical disorders. Several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile [1]. Cigarette smoking increases plasma catecholamine which induces lipolysis and release of free fatty acid, which will be taken up by the liver[2].

Nicotine and other toxic substances from tobacco smoke are absorbed through the lungs into the blood stream and are circulated throughout the body. These substances narrow or damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a nonsmoker. Nicotine increases the amount of bad fats, they are Total Cholesterol (TC), Low Density Lipoprotein Cholesterol (LDL-C), and Triglycerides (TG) circulating in the blood vessels and decreases the amount of good fat is

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High Density Lipoprotein Cholesterol (HDL-C) availability. The tobacco smoking is responsible for premature development of CAD Cardiovascular disease and abnormal levels of risk factors such as serum lipid and lipoprotein levels, hypertension, and smoking are related to the earliest stages of atherosclerotic CAD [3].

Nicotine induces oxidative stress, generates free radicals that attack on the membrane lipids resulting in the formation of malondialdehyde (MDA), which causes peroxidative, tissue damage [4].

Low Density Lipoprotein-Cholesterol (LDL-C) and Very Low Density Lipoprotein-Cholesterol (VLDL-C) are atherogenic and the High Density Lipoprotein-Cholesterol (HDL-C) is a protective factor against coronary atherosclerosis [5].

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 in the blood stream [6]

(b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL-Cholesterol [7]

(c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL and TG due to decreased activity of lipoprotein lipase [8]

MATERIALS & METHODS

The study was conducted in the Department of Physiology (2011-2013) at Kamineni Institute of Medical Sciences and Hospital, Narketpally, Nalgonda district, Telangana, India. The study protocol was approved by the institutional ethics committee. A written informed consent was obtained from each subject.

Inclusion criteria:

1. Age between 18-55 years
2. The study subjects were divided into 2 groups i.e. Group-I (Smokers) and Group-II (Controls)
3. Group-I (Smokers) with history of tobacco smoking (1 year to 11 years +) and n=50
4. Group-II (Controls) were age and sex-matched and healthy volunteers and n=50

Exclusion criteria:

Subjects suffering from Hypertension, Diabetes Mellitus, Alcoholism, Nephrotic Syndrome, HMG-CoA Reductase Inhibitors, Fibric Acid Derivatives, Beta Blockers, Nicotine acid, Diuretics and Subjects who are on diet restriction.

Blood samples were obtained following an overnight fasting. 5 ml of blood withdrawn from a cubital vein into blood tubes. The serum was then separated from the cells by centrifugation at 3000 rpm for 10 min and immediately stored at 4°C. Serum Total Cholesterol, Triglyceride, HDL-C, LDL-C, VLDL-C was measured by using the end-point method using Mindray - BS-380 autoanalyzer.

And the reference value are

Total cholesterol: <200 mg/dl, Triglycerides: 0-150 mg/dl, HDL-C: 40-60 mg/dl,

LDL-C: 0-100 mg/dl, VLDL-C: 12-40 mg/dl.

RESULTS

The results of this study showed that the decrease in serum levels of HDL-C in Group-I when compared with Group-II. Increase in serum levels of TC, TG, LDL-C, VLDL-C in Group-I when compared to Group-II.

The statistical analysis was performed using SPSS software version 11.0. The descriptive results were expressed as Mean and Standard deviation. Significance of the difference between the patient and control groups observed was assessed by using the student t-test.

Table 1: Comparison of serum lipid profile in Group-I (Smokers) with Group-II (Controls)

Lipid Profile In Serum	Group-I (Smokers)	Group-II (Controls)	p-Value
Total Cholesterol (<200 mg/dl)	235.4±8.80	164.46±6.44	<0.05
Triglycerides (0-150 mg/dl)	199.76±24.54	108.77±18.60	<0.05
HDL Cholesterol (40-60 mg/dl)	46.48±5.97	60.17±3.63	<0.05
LDL Cholesterol (0-100 mg/dl)	132.98±30.36	94.25±13.82	<0.05
VLDL Cholesterol (0-40 mg/dl)	39.95±4.90	21.75±3.72	<0.05

p-Value ≤ 0.05 was considered statistically significant.

Table-I & Figure-I: Shows that there is statistically significant difference in between both the groups in Total Cholesterol (mg/dl) levels (p<0.05) with 235.4±8.80 in group-I and 164.46±6.44 for group-II. In Triglycerides (mg/dl) levels (p<0.05) with 199.76±24.54 for group-I and 108.77±18.60 for group-II. In HDL-Cholesterol (mg/dl) levels (p<0.05) with 46.48±5.97 for group-I and 60.17±3.63 for group-II. In LDL-Cholesterol (mg/dl) levels (p<0.05) with 132.98±30.36 for group-I and 94.25±13.82 for group-II. In VLDL-Cholesterol (mg/dl) levels (p<0.05) with 39.95±4.90 for group-I and 21.75±3.72 for group-II.

Table-I & Figure-I: Also describes that the increase in serum levels of Total Cholesterol, Triglycerides, LDL Cholesterol, VLDL Cholesterol and decrease in serum levels of HDL Cholesterol in group-I when compared to group-II.

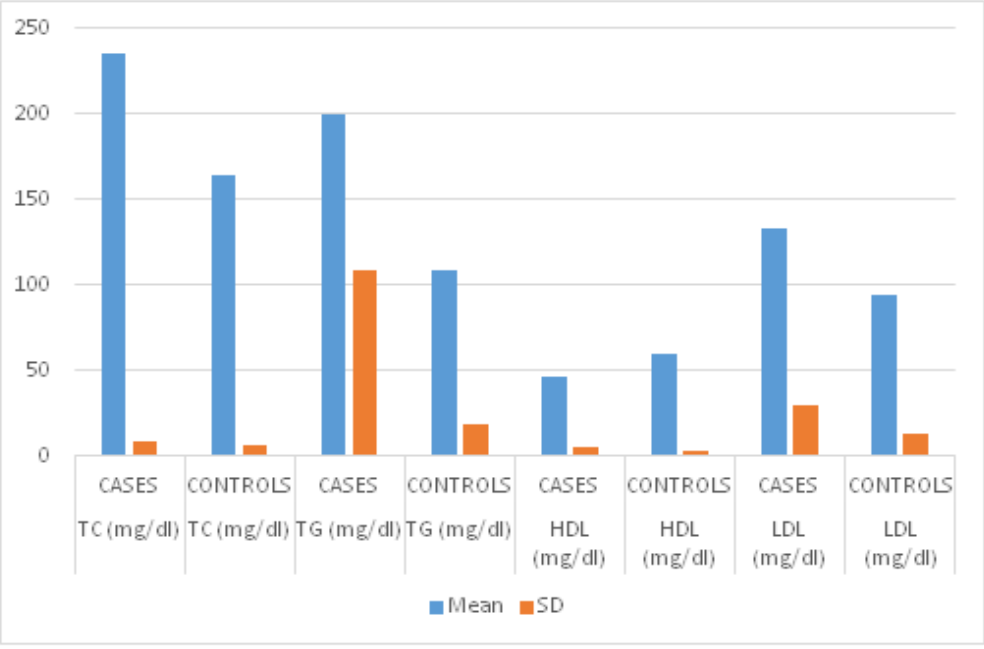

Figure 1: Comparison of Lipid Profile in Group-I (Smokers) with Group-II (Controls)

DISCUSSION

Cigarette smoking is associated with reduced HDL-C level by alteration of the critical enzymes of lipid transport lowering Lecithin-Cholesterol Transferase (LCAT) activity and altering cholesterol ester transfer protein (CETP) and hepatic lipase activity [9]. Previous research workers have reported that tobacco smoking is associated with increased levels of total cholesterol, triglyceride, LDL–C, VLDL-C and decreased level of HDL-C [10, 11, 12]

The present study selected 50 study subjects (cases) were chronic tobacco smokers and 50 healthy subjects (controls) in the comparable age group. The study population comprised fully of males which may be attributed to the cultural background of the local population. The present study to assess the impact of smoking on lipid profile in cases when compared to controls.

In our study Serum TC, TG, LDL-C and VLDL-C were significantly higher in Group-I as compared to Group-II and furthermore observed that the serum HDL-C levels was significantly lower in Group-I as compared to Group-II. Our findings are in accordance with the findings of many research workers like Sinha AK et Al [13]. It seemed that cigarette smoking could promote atherosclerosis, in part, by its effect on lipid profile [14, 15, 16]. It is also found that the risk of development of CVD is directly related to the number of cigarettes smoked [17, 18]. Dyslipidemia is a well-established risk factor for the development of coronary artery disease. Our study demonstrated presence of dyslipidemia in chronic smokers.

CONCLUSION

Chronic tobacco smoking related in the alteration in lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with increased number of years smoked. Tobacco smoking plays the key role for atherosclerotic process and with coronary artery disease. It is strongly recommended to avoid tobacco smoking for the benefit of cardiac health.

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