ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 9, Issue 4, 2016

Online - 2455-3891
Print - 0974-2441

Research Article

DYSLIPIDEMIA AMONG SMOKERS

PREETI SHARMA[1]*, PRADEEP KUMAR[1], RACHNA SHARMA[2], KISHOR K[3], GUPTA G[1]

[1]Department of Biochemistry, Santosh Medical College & Hospital, Ghaziabad, Uttar Pradesh, India. [2]Department of Biochemistry, TSM Medical College & Hospital, Lucknow, Uttar Pradesh, India. [3]Department of Medicine, Santosh medical College & Hospital, Ghaziabad, Uttar Pradesh, India. Email: prcdri2003@yahoo.co.in

Received: 20 March 2016, Revised and Accepted: 12 April 2016

ABSTRACT

Objective: The objective of this study is to evaluate lipid profile among cigarette smokers and compare it with non-smokers.

Methods: About 125 subjects aged between 20 and 40 years including 100 smokers as case group and 25 non-smokers as control were taken into study. They did not having any history of any disease (i.e., diabetes, hypertension, liver diseases, renal diseases, or obesity) or alcohol intake. They were not taking any drug such as B-blockers, lipid lowering drugs, or thiazide diuretics.

Results: The mean serum total cholesterol, low-density lipoprotein cholesterol (LDL-C), very LDL-C (VLDL-C) were significantly raised ($p<0.05$) in all three groups of mild, moderate, and heavy smokers as compared to non-smokers control while mean serum high--density lipoproteins cholesterol (HDL-C) was significantly lower in all three above said groups.

Conclusion: Cigarette/beedi smoking is associated with lower level of god cholesterol, i.e., HDL, and higher level of cholesterol, triglycerides, and serum LDL and VLDL.

Keywords: Dyslipidemia, smokers

INTRODUCTION

Smoking has been one of the earliest vices that man gave himself up to now. The pleasure associated with smoking is far eclipses any notion of bodily harm that smoking might cause. Slow and insidious harm is caused by nicotine, the main constituent of tobacco filled in cigarette induces addiction among smokers [1]. Cigarette smoking is an escalating health concern especially in developed countries. According to WHO, by the year 2020, coronary artery diseases and stroke will be the leading cause of disability and mortality [2]. Incident of cardiovascular disease (CVD) would rise from 2.90 crore in 2002 to more than 6.40 crore in 2016 [3]. It is one of the most important risk factors for peripheral coronary and cerebral atherosclerotic vascular diseases [4]. There is shiny synergistic interaction exist between hypercholesterolemia and tobacco consumption in any form. Constituents of cigarette smoke that contribute to CVDs are nicotine, carbon monoxide and oxidant gases [5]. Nicotine is absorbed rapidly from cigarette smoke producing arterial blood level of 40-100 ng/ml after each cigarette. It is sympathomimetic drug that releases catecholamines both locally from neurons and systematically from adrenal glands. Enhanced hormonal circulation of epinephrine and norepinephrine would activate adenyl cyclise of adipose tissues which cause lipolysis of stored triglycerides (TG) and release of free fatty acids in blood [6]. Carbon monoxide binds avidly to hemoglobin impending oxygen supply to the tissues causing many disfunctions including ventricular disfunctions and arrthymias [7]. Moreover, high concentration of oxidizing chemicals in the smoke such as oxides of nitrogen and sulfur and a variety of free radicals. Oxidant stress is believed to contribute to a number of potential mechanisms of CVDs [8]. Well there is lack of sufficient studies on the relationship of lipid profile and smoking in the region. Therefore, we aim to study the lipid profile changes between smokers and non-smokers in the fasting state.

METHODS

The study was carried out in the Department of Biochemistry, Santosh Medical College and Hospital, Ghaziabad, from February 2013 to November 2014. About 125 patients between age group 20-40 years including 100 study subjects as smokers and 25 subjects as non-smokers control. Subjects gave their voluntary informed consent before start of the study. Cigarette smokers were divided into three groups depending upon the amount of smoking.

1. Mild smokers (1-10 cigarettes/beedies per day for at least 5 years)
2. Moderate smokers group (11-20 cigarettes/beedies per day for at least 5 years)
3. Heavy smokers (> 20 cigarettes/beedies per day for at least 5 years).

The subjects having body mass index (BMI) were less than 28, were included into study. They were all on average Indian diet.

Exclusion criteria

Subjects who chew tobacco, ex-smokers, having any disease influencing lipid profile (i.e., B-blockers, thiazide, statins, fibric acid derivatives, nicotinic acid, on any dietary restrictions, obese persons and ladies were excluded from the study. Patients in the study were subjected to complete medical and physical examination.

Fasting and post-prandial blood sugar (in order to rule out the diabetes), lipid profile including TG, very low-density lipoprotein cholesterol (VLDL-C), LDL-C, high-density lipoprotein cholesterol HDL-C), total cholesterol (TC) were done. Serum TC was estimated by cholesterol oxidase-peroxidase enzymatic colorimetric method [9], HDL by enzymatic clearance assay using phosphotungstate method [10], TG by GPO-PAP method [11], VLDL by Friedwald's WT formula TG/5. LDL by Friedwald's formula [10], blood glucose by GPO-PAP enzymatic colorimetric method [12], both in fasting and post-prandial state.

Statistical analysis

Statistical analysis was performed using Statistical Package for Social Sciences for PC (Window's).

RESULTS

In the above study, 100 smokers and 25 non-smokers were studied for lipid profile. Number of smokers in the mild smokers category were 24, in the moderate group 51 and in heavy smokers group were 25 of

Table 1: Lipid profile comparison between Heavy, moderate and mild smokers and non-smokers.

Lipid profile (mg/dl)	Non-smokers (n=25)	Heavy smokers (n=25)	p value	Moderate smokers (n=51)	p value	Mild smokers (n=24)	p value
TC	162.24	180.96±45.61	0.001	172.60±31.17	0.001	171.20±34.01	0.001
Serum TG	98.88	199.04±37.30	0.001	158.74±55.07	0.001	121.91±40.04	0.001
Serum LDL	95.88	139.35±24.01	0.001	128.47±23.89	0.001	116.35±22.76	0.001
Serum VLDL	22.86	45.88±10.94	0.001	35.77±5.95	0.001	34.32±4.15	0.001
Serum HDL	44.20	32.92±5.09	0.001	33.08±5.51	0.001	33.95±4.35	0.001

TG: Triglycerides, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein, HDL: High-density lipoproteins, TC: Total cholesterol

Table 2: Lipid Profile comparison between smokers (mean of all categories of smokers) and non-smokers

Lipid profile	Non-smokers (n=25)	Smokers (n=100)	p value
Total cholesterol	162.24±22.54	174.16±35.84	<0.001
Serum triglyceride	98.88±30.60	159.96±84.93	0.001
Serum LDL	95.88±14.65	127.51±24.35	<0.001
Serum VLDL	22.86±8.33	37.99±8.51	<0.001
Serum HDL	44.20±1.94	33.22±5.10	<0.001

LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein, HDL: High-density lipoprotein

the total 100 smokers. Mean of BMI of non-smokers was 22±1.06 and of smokers was 22.08±1.63. Smokers had high TC level compared to non-smokers (174.16 vs. 162.24) with p<0.001. Smokers had higher plasma TG compared to non-smokers (154.96 vs. 98.88). This difference was statistically significant. Smokers had high VLDL level compared to non-smokers (37.99 vs. 22.88). Smokers had lower level of serum HDL compared to non-smokers (33.25 vs. 44.2) and these differences were statistically significant (Tables 1 and 2).

DISCUSSION

Smoking is the major risk factor in the developing world. In present study 100 smokers and 25 non-smokers were studied for their lipid profile. It is revealed that TC, LDL, VLDL, HDL, and TC alterations were statistically significant in smokers as compared to non-smokers. Nearly 70% increase in the risk of death from coronary artery disease have been reported only due to cigarette smoke. Adedeji *et al*. explored that serum cholesterol and LDL were found to be raised significantly (p<0.05) when compared to non-smokers [13]. However, TG and VLDL level difference between smokers and non-smokers was not found to be significant (p<0.01). However, HDL levels were higher in non-smokers than in smokers (p<0.01). In another study conducted by Makoto *et al*., only mean TG level difference between smokers and non-smokers was statistically significant (p<0.05). Other parameters such as TC, VLDL, and LDL were insignificantly altered when compared in both the groups with p<0.05. Mean HDL levels were higher in non-smokers than smokers and this difference was statistically significant (p<0.05) [14]. Studies done by Neki [15] and Anila *et al.* [16] showed almost similar results. The mean serum TC in smokers when compared to non-smokers was statistically significant. Serum TG, VLDL, LDL showed the same trend, whereas the mean serum HDL was higher in non-smokers when compared to smokers. A study done by Rao and Subash in 2013 reported mean total serum cholesterol level in smoker subjects had an increase of about 16.94% (p<0.001). The mean serum VLDL and LDL were significantly raised in smokers with 27.54% (p<0.01) and 34.64% (p<0.001) values, respectively. Mean serum HDL level in smoker subjects was decreased by 9.78% (p<0.01) [2]. Similar results were found in our study.

CONCLUSION

From our study, it is apparent that cigarette/beedi smoking is associated with significant lower level of serum HDL and higher level of serum cholesterol, serum TG, serum LDL, and serum VLDL levels. Further, this association is dependent on number of cigarette/beedis smoked/day. The greater risk of smoking for the development of coronary heart diseases results from the HDL lowering effect of smoking. From the results of the above study, it may be concluded that cigarette smoking in young adults induces dyslipidemia in the direction of increased risk for coronary artety diseases. Hence, it is strongly recommended to avoid smoking for the benefit of cardiac health.

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