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RESEARCH ARTICLE

EFFECT OF CIGARETTE SMOKING ON LIPID PROFILE

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ABSTRACT

Tobacco smoking is one of the most potent and prevalent addictive, influencing behavior of human beings for over four centuries. Cigarette smoking is an important factor for the development of atherosderosis, coronary artery disease and peripheral vascular disorders. Cigarette smoking is known to alter the lipoprotein levels. Some authors have concluded that High density Lipoprotein (HDL) levels were same for smokers and non-smokers, while others have found conflicting results wherein significant variations (low levels of HDL in cigarette smokers) were obtained. The present study was done to compare the effects of cigarette smoking on lipid levels. This was a prospective study conducted at tertiary care hospital in South India from September May 2013 to August 2013. The subjects were selected from department of medicine who were undergoing master health check up. The smokers were divided into three groups. Mild smokers (Group I) = 1-10 cigarettes per day. Moderate smokers (Group II) = 11-20 cigarettes per day. Heavy smokers (Group III) = >20 cigarettes per day. The lipid levels were statistically significant in all the three groups. The HDL levels were moderately significantly ($P \leq 0.05$) in Group I. All other lipid levels were highly significant ($P \leq 0.01$) in all the three groups. When the lipid levels were compared along with the duration of smoking, there was statistically significant difference in total cholesterol and HDL levels, but moderately significant in triglycerides and Low density Lipoprotein (LDL), and no significant difference in Very Low density Lipoprotein (VLDL) levels. The results of this study show that smokers are at greater risk of developing atherosderosis than non-smokers. The changes become more marked with increased duration and number of cigarette smoked. Creating awareness regarding health consequences of smoking at young age, particularly in schools and colleges will help in reducing cardiovascular mortality.

Key words: Tobacco smoking, lipid profile, coronary artery disease, dyslipidemia

INTRODUCTION:

Tobacco smoking is one of the most potent and prevalent addictive, influencing behavior of human beings for over four centuries. Smoking is now increasing rapidly throughout the developing world and is one of the biggest threats to current and future health [1]. Cigarette smoking is the most common type of tobacco use. Tobacco continues to be the second major cause of death in the world [2]. Cigarette smoking is an important factor for the development of atherosderosis, coronary artery disease and peripheral vascular disorders. Cigarette smoking is known to alter the lipoprotein levels [3].

It is estimated that globally tobacco kills nearly 6 million people each year, of which more than 600 000 are non-smokers dying from breathing second-hand smoke. If the smoking epidemic continues it will kill more than 8 million people every year by 2030. More than 80% of these preventable deaths will be among people living in low- and middle-income countries [4]. There is a dose response relationship between number of cigarettes smoked and cardiovascular events [5]. The cardiovascular disease effects are mediated through multiple interrelated mechanisms, including oxidative stress, endothelial injury and dysfunction, altered blood coagulation, and derangements of lipid composition and metabolism [6].

Although several studies [7-9] provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile, there still is inconclusive evidence regarding the alteration of a particular lipoprotein, particularly to high density lipoprotein (HDL) levels. Some authors have concluded that HDL levels were same for smokers and non-smokers [10], while others have found conflicting results wherein significant variations (low levels of HDL in cigarette smokers) were obtained [11, 12]. The present study was done to compare the effects of cigarette smoking on lipid levels.

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MATERIAL AND METHODS:

This was a prospective study conducted at tertiary care hospital in South India from September May 2013 to August 2013. The subjects were selected from department of medicine who were undergoing master health check up. The study was carried out after an approval from Institutional ethical committee and informed consent was obtained from the study participants.

Inclusion criteria:

1) Male subjects aged between 20-50 years, non smokers and smokers (cigarette and beedi smoking) of >2 years duration.

Exclusion criteria:

1) Subjects with history of medical disorders such as diabetes, hypertension, hepatic, renal and cardiac disorders

2) Ex smokers, alcoholics, obese (BMI >30), family history of dyslipidemia

3) Subjects on medications such as beta blockers, steroids, vitamin supplementation, herbal medications and lipid lowering drugs.

The smokers were divided into three groups. Mild smokers (Group I) = 1-10 cigarettes per day. Moderate smokers (Group II) = 11-20 cigarettes per day. Heavy smokers (Group III) = >20 cigarettes per day.

Laboratory aspects:

Blood was drawn from the subjects after 12 hours fasting with staple food for two days. Enzymatic method was used to estimate total cholesterol (TC) and triglycerides (TGL) using commercial kits. High-density lipoprotein (HDL) was determined by precipitation of phosphotungstic acid MgCl [13]. The levels of low density lipoprotein cholesterol (LDL) and low density lipoprotein cholesterol (VLDL) were calculated using Friedewalds formula [14]. Adult Treatment Panel III (ATP III) criteria was used to classify plasma lipid levels [15]. Total cholesterol, triglyceride, LDL and VLDL levels exceeding 200, 150, 100 and 30 mg/dl respectively, and HDL levels below 45 mg/dl were considered as abnormal.

The data was recorded and analyzed using SPSS software (version 15). Differences between mean values were evaluated by student 't' test. Significance is assessed at 5% level of significance.

* Moderately significant (*P* value: $P \leq 0.05$)

** Strongly significant (*P* value: $P \leq 0.01$)

RESULTS:

Table 1: Demographic data of the subjects

Variable	Non-smokers	Group I	Group II	Group III
Number of subjects	60	63	66	64
Age	39.7±4.68	34.4±3.72	37.4±3.52	36.4±3.63
BMI	24.4±2.72	24.7±2.69	25.4±2.54	25.7±2.62
SBP	124.14±5.46	126.34±4.86	127.84±4.74	126.24±5.16
DBP	81.19±3.57	82.34±2.95	80.14±3.65	82.18±3.24

Values are expressed in Mean ± SD

BMI = Body mass index, SBP = Systolic blood pressure, DBP = Diastolic blood pressure

No statistically significant difference was seen among smokers and non-smokers

Table 2: Lipid profile of smokers and non-smokers

Lipid profile	Non-smokers	Group I	Group II	Group III
TC	153.18±23.19	183.18±14.84**	195.63±21.63**	212.18±24.69**
TGL	112.72±36.69	146.42±38.52**	167.22±45.19**	186.33±48.92**
HDL	49.58±8.96	44.15±7.32*	39.55±6.96**	32.45±6.54**
LDL	87.56±19.11	115.45±21.96**	145.32±22.31**	158.32±23.63**
VLDL	25.44±7.16	35.31±8.22*	39.12±7.12**	43.27±8.02**

TC = Total Cholesterol, TGL = Triglycerides, HDL = High density Lipoprotein, LDL = Low density Lipoprotein, VLDL = Very low density Lipoprotein cholesterol

**$P \leq 0.01$ = Strongly significant

*$P \leq 0.05$ = Moderately significant

All *P* values are in comparison with nonsmokers

Table 3: Lipid profile according to duration of smoking

Lipid profile	Smoking <10 years (n=109)	Smoking >10 years(n=84)
TC	187.7±36.6	207.7±47.4**
TGL	155.54±32.4	146.15±45.9*
HDL	44.32±5.1	36.27±4.9**
LDL	108.5±29.8	128.4±41.9*
VLDL	27.5±1.9	31.29±2.3 (NS)

TC = Total Cholesterol, TGL = Triglycerides, HDL = High density Lipoprotein, LDL = Low density Lipoprotein, VLDL = Very low density Lipoprotein

NS= not significant

**$P \leq 0.01$ = Strongly significant

*$P \leq 0.05$ = Moderately significant

DISCUSSION:

Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease and peripheral vascular disorders [16]. In the present study, the demographic characteristics were comparable between groups (table 1). The lipid levels were statistically significant in all the three groups (table 2). The HDL levels were moderately significantly ($P \leq 0.05$) in Group I. All other lipid levels were highly significant ($P \leq 0.01$) in all the three groups. The above findings are in accordance with the other studies [17-20]. But some studies [21,22] did not find any statistically significant difference in the lipid levels.

In another study conducted by Majos O. D. et al. [23] reported that there is significant decrease in HDL-C, but there is no change in total cholesterol and triglycerides. Another report shows lower but no significant HDL levels in smokers [24]. But a study conducted by Siekmeier et al [25] reported the HDL levels are same for smokers and non-smokers. These variations might be due to different demographics of the subjects, different laboratory methods used and geographical distribution of the subjects.

When the lipid levels were compared along with the duration of smoking, there was statistically significant difference in total cholesterol and HDL levels (table 3), but moderately significant in triglycerides and LDL, and no significant difference in VLDL levels. It emphasis that the changes in the serum lipids tends to be high with the increase in duration and intensity of smoking. The findings are accordance with other studies [26,27].

As the present study is concerned with only lipid profile in normal healthy controls and smoking male subjects, the results of this study should be correlated with other biochemical, physiological and clinical aspects. Creating awareness regarding health consequences of smoking at young age, particularly in schools and colleges will help in reducing cardiovascular mortality [28].

Limitations of the study:

The result of our study may not be generalized because of low sample size and same geographical distribution. We did not include apolipoprotein (Apolipoprotein A1 and Apolipoprotein B) concentration in lipid profile. Analysis was dependent on self reported smoking habits which sometimes may not be accurate. Further studies should be multicentic with large sample size and should cover wide geographical area.

CONCLUSION:

The results of this study show that smokers are at greater risk of developing atherosclerosis than non-smokers. The changes become more marked with increased duration and number of cigarette smoked. The policies that prevent and reduce smoking will have immediate and large benefits for reducing cardiovascular mortality.

Conflict of interest: None

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