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Effects of cigarette smoking on lipids profile

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ABSTRACT:

Cigarette smoking is one of the major risk factors in the genesis of coronary atherosclerosis and heart disease. This study aims to investigate the effects of smoking on the lipid profile. 110 healthy male subjects participated in this study, 70 smokers and 40 non-smokers in the age range of 18 and 50 years at medical laboratory department in higher institute of sciences and medical techniques, Mesallata, Libya, total cholesterol (TC), triglycerides (TG), very low density lipoproteins (VLDL) , low density lipoproteins (LDL) and high density lipoproteins (HDL) were measured. The result showed that significant increase levels of TC, TG, VLDL and LDL in smokers group compared to non-smokers group ($P < 0.001$). While HDL significantly decrease in smokers compared to non-smokers group ($P < 0.001$). In conclusion cigarette smoking has severe adverse effects on lipids profile which includes increasing the levels of TC, TG, LDL and VLDL with a decrease in HDL level. Smoking whether light or heavy equally affects lipid profile levels, this dyslipidemic levels among smokers may provide a mechanism whereby smoking predisposes to greater risk of developing atherosclerotic plaques and chronic heart disease.

INTRODUCTION

Cigarette smoking is the most common type of tobacco use[1].

Cigarette smoking is one of the major risk factors of atherosclerosis, coronary artery disease [2]. Cigarette solid in developing countries tend to higher tar contents, and are less likely to be filtered, potentially increasing vulnerability to tobacco related disease in these countries. Cigarette contains carcinogens (polycyclic aromatic hydrocarbon etc.), tar, nicotine, carbon monoxide and other gases. Cigarette smoke contains many oxidants and free radicals which can harms lipids, carbohydrates and other biomolecules[3]. The free radical can damage the heart muscles and blood vessels. They react with cholesterol leading the buildup of fatty material on artery walls[4]. Smoking reduce the pulmonary function by narrowing the airway and also increases the risk of stroke and myocardial infarction by blocking the blood flow to brain and heart respectively[5]. Several studies provide the evidence that smoking is strongly associated with altering the normal status of the lipid profile [6-8]. Different results have been obtained by various investigator, some studies reported that tobacco smoking is associated with increased levels of TC,TG, LDL, VLDL and decreased level of HDL-C[9,10] . However, other studies were reported with conflicting results[11, 12]. It seemed that cigarette smoking could promote atherosclerosis, in part, by its effect on lipid profile[13]. This study was conducted to evaluate the effect of cigarette smoking on lipid profile and the effect of light and heavy smoking on lipid profile.

MATERIAL AND METHODS

The present study was done in 110 male volunteers divided into two groups. 70 male volunteers were smokers and 40 male volunteers were non-smokers and all were aged between 18 to 50 years attending to higher institute of medical sciences and techniques, Mesallata city, who had the history of smoking one or more cigarette per day, regularly for at least past one year. Institutional Ethical Committee permission and approval obtained. Informed consent was obtained from all the subjects.

Subjects with diabetes mellitus, ischemic heart disease or peripheral vascular disease, chronic renal disease, hypertension, any infectious or debilitating illness, taking any drugs that influence lipid level were excluded from the study.

The present study comprises of 2 groups, group I nonsmokers (control) n=40 and group II cigarette smokers n= 70. Group II were divided into 2

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subgroups depending upon intensity of smoking. Each group comprises about 35 volunteers.

Group II A, light smokers (n =35), smoking < 20 cigarettes / day)

Group II B, heavy smokers (n=35), smoking ≥ 20 cigarettes / day)

Overnight 12 hours fasting blood samples were collected from these subjects. Five millimeters of venous blood were withdrawn from the antecubital vein under sterile precaution. Serum was separated by centrifugation at 4000 rpm for five minutes. The clear serum samples were employed for the estimation of TC [14], TG [15] and HDL- C [16]. The of LDL-C and VLDL were calculated by using Friedewalds formula [17].

LDL-C (mg%) = TC – (HDL-C+ TG/5)

VLDL (mg%) = TG/5

Statistical analysis was performed by using the SPSS version 19 and statistical significance were analyzed by unpaired 't' test and ANOVA. P value < 0.001 was considered for statistically significance.

RESULTS

Table1. Compares the lipid profile between smokers and non-smokers which shows a statistically significant increase in TC, TG, LDL and VLDL levels (217.45, 218.71, 173.51, 45.51) in group II(cigarette smokers) when compared to group I (nonsmokers), (185.56, 115.56, 123.71, 23) respectively and significantly decrease in HDL-C of group II when compared to group I with P value < 0.001.

Table1. Lipid profile in non-smokers and smokers.

Parameters	Smoker Mean SD	Nonsmoker	t value	P value
TC (mg%)	217.45±23.55	185.56±16.44	3.573	0.001
TG(mg%)	217.49±19.29	115.56±12.088	5.303	0.000
HDL-C (mg%)	37.87±3.17	48.98±5.07	4.394	0.000
LDL-C (mg%)	158.93±14.34	123.71±11.56	6.917	0.000
VLDL (mg%)	45.59±3.69	23.0±2.4	5.123	0.000

Table2. Compares the lipid profile between light smokers (Group II A) and heavy smokers (Group II B) which shows no significant differences in the lipid profile between two groups (P value > 0.05).

Table 2 compression of lipid profile between smokers

Parameters	Light smokers	Heavy smokers	f value	P value
TC (mg%)	216.78±10.57	220.406± 11.54	0.068	0.795
TG(mg%)	209.27±32.73	241.70±24.6	0.979	0.326
HDL-C (mg%)	35.57±2.73	42.27± 2.781	3.693	0.059
LDL-C (mg%)	165.89± 17.5	155.91± 14.63	1.251	0.267
VLDL (mg%)	41.81± 4.76	52.70±3.65	2.022	0.160

DISCUSSION

Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease and peripheral vascular disorder[1]. In the present study the levels of TC, TG, LDL-C and VLDL were statistically significantly increase in smokers when compared to non-smokers and statistically significant reduction in levels of HDL-C in smokers when compared to non-smokers these result was analogous with Neki,[18] and Anandha, *et al* [19]. But some studies [20,21] did not find any statistically significant difference in the lipid levels.

The possible mechanisms for dyslipidemia among smokers were following.

(i)Nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol as well as hepatic secretion of very low density lipoprotein (VLDL) and increased TG.

(ii) HDL concentration was inversely related to VLDL concentration in serum[22].

(iii) Smoking increases insulin resistance and causes hyperinsulinemia. LDL, VLDL and TGL are elevated in hyperinsulinemic conditions due to decreased activity of lipoprotein lipase and hepatic lipase has been activated, which converts VLDL to LDL [23].

When the lipid levels were compared along with the duration of smoking, there was not statistically significant difference in TC, TG, LDL-C,VLDL and HDL-C between heavy and light smoking, there is no agreement with other study [24,25]. This differences may be due to *Light smoking* has the widest set of definitions, Cigarettes smoked per day is the natural measure of the magnitude of smoking. This is generally categorized as (1-9), (10-19), (20-29). Some investigators divide it arbitrarily into light and heavy. For example, Baird and Wilcox [26] considered 20 or less cigarettes per day as light, and 21 or more heavy, Petrauskaite et al. [27] used a cut-off of 7 cigarettes per day.

CONCLUSION

Cigarette smoking associated with dyslipideamia (increase TC, TG, LDL-C,VLDL and decrease HDL-C), this dyslipidemic levels among the smokers may expose the vascular endothelium to potentially atherogenic lipoproteins which predisposes the greater risk of developing atherosclerotic plaques and coronary heart disease among the smokers.

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