Effect of Smoking on Lipid Profile in Men in Ramadi Municipality

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Summary:

Background: Tobacco smoking is one of the major modifiable cardiovascular risk factors. Nicotine and other toxic substances from tobacco smoke are absorbed through the lungs into the blood stream and are circulated throughout the body. Smoking increases the amount of bad fats circulating in the blood vessels and decreases the amount of good fat availability.

Objectives: The aim of the study was to find out differences in the serum lipid profile between men smokers and non-smokers in the fasting state.

Patients and Methods: This descriptive study included two hundred and forty healthy non- obese males, categorized into two groups. One hundred (100) and forty (40) healthy men were included in group I (smokers) with in the age of 19-48 years (31.76±6.93) and one hundred apparently healthy men were included in group II (non-smokers) of the same age and weight matched as a control group. Group I was classified as those who stated smoking number of cigarette per day for at least three consecutive months into three different categories; group A included 38 males smoked 1-10 cigarette per day, group B included 78 males smoked 11-20 cigarette per day, and group C included 24 healthy males smoked more than 20 cigarette per day. Lipid profile was measured for all these groups.

Results: This study showed that total cholesterol(TC), low-density lipoprotein cholesterol (LDL-C), very-low density lipoprotein cholesterol (VLDL-C), LDL-C/HDL-C (atherogenic index), and triglyceride (TG) were significantly higher in smokers as compared with non-smokers (control group) ($P<0.001$, $P<0.001$, $P=0.001$, $P<0.001$, $P=0.001$ respectively) while low-density lipoprotein cholesterol is smoking group was significantly lower in non-smoker ($P=0.04$). total cholesterol, low-density lipoprotein cholesterol, and atherogenic index were statistically affected by number of cigarette/day.

Conclusions: Smoking produces adverse effects on lipid profile and the changes become more marked with the number of cigarette/day smoked

Keywords: lipid profile, nicotine, male.

Introduction:

Tobacco smoking is one of the major modifiable cardiovascular disorders(1), pulmonary diseases(2), and oxidant stress(3). Nicotine increases the amount of bad fats (total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG)) circulating in the blood vessels and decreases the amount of good fat (high-density lipoprotein cholesterol(HDL-C) availability (4). These observations are clinically important because of the widespread use of smokeless tobacco products such as nicotine containing chewing gum and nicotine lozenge in smoking cessation therapy(5). In fact, smoking 1-5 cigarettes per day presents a significant risk for a heart attack(4).

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 damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a non-smoker(4). Nicotine induces oxidative stress, generates free radicals that attack on the membrane lipids resulting in the

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formation of malondialdehyde (MDA), which causes peroxidative, tissue damage(6).

Lipoprotein oxidation is presumed to occur in the artery that may generate superoxide radicals, hydrogen peroxide or lipid peroxides outside the cell may contribute to the oxidation of LDL (7). These silent effects begin immediately and greatly which increase the risk for heart disease and stroke(8).An increased level of MDA has been documented in smokers by several authors (9-11), an evidence of intensification of lipid peroxidation processes which may cause chronic stress for endothelial cells. On the other hand, it can also reorientate enzymatic systems of the arachidonic acid cascade towards intensified TXA2 synthesis (9-11). In this way, cigarette smoking substantially hastening the risk of coronary heart disease and ischemic stroke(12-14). In addition, a study in Japan showed a measurable decrease in the elasticity of the coronary arteries of non-smokers after just 30 minutes of exposure to second hand smoke(15). There is a dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality(16).

To date, no statistical data are available on tobacco smoking among Iraqi people, a country with the highest cigarette smoking incidence. Moreover, there have been no studies showing the relationships of tobacco smoking with cardiovascular disorders.

The present work is an attempt to determine the alterations in the serum lipid profile (total cholesterol, LDL-C, HDL-C, VLDL-C, atherogenic indices, and triglyceride) between men smokers and non-smokers in the fasting state.

Materials and methods:

This study had been conducted between June 2007 and December 2007. The present descriptive study included two hundred (100) and forty healthy non obese males, categorized into two groups were recruited for the study after obtaining written informed consent. One hundred and forty healthy male subjects with similar dietary habits were selected for this study group I (smokers) in the age of 19-48 years (31.76±6.93) and one hundred apparently healthy males were included in group II (non-smokers) of age and weight matched as a control group. Group I was classified as those who stated smoking number of cigarette per day for at least three consecutive months into three different categories; group A was included 38 males in the age 19-45 years (30.12±6.52) and smoked 1-10 cigarette per day, group B was included 78 males in the age 20-46 years (31.45±6.92) and smoked 11-20 cigarette per day, and group C was included 24 healthy males in the age 24-48 years (35.42± 6.55) and smoked more than 20 cigarette per day.

From both groups (smokers and non smokers), 5 ml of early morning venous blood samples were drawn aseptically by venepuncture for lipid analysis, following a 12 hour overnight fast. Blood samples were collected into white tubes, allowed to clot and the serum collected for analysis. TC, TG, and HDL-C were estimated using the enzymatic commercial endpoint (kit) method. TC was estimated by Biomaghreb, its reference value 3.6-7 mmol/l. TG, and HDL-C were estimated by Linear chemicals, S.L, their reference values 1.70-2.25 mmol/l and 0.90-1.42 mmol/l respectively. Very low-density lipoprotein cholesterol (VLDL-C) and LDL-C were estimated by calculation. VLDL-C was calculated as TG/5 and LDL-C as follows: LDL-C = TC – (HDL-C + VLDL). The ratio of LDL-C to HDL-C (atherogenic index) was also calculated. A detailed history and physical examination of the subjects of both groups were done. Those excluded from the study were persons abusing alcohol, diabetes mellitus, hypertension, renal disease, hepatic impairment, obesity, and on drugs like β-blockers, lipid lowering drugs, and thiazide diuretics in both groups (smokers and non smokers), by assessing blood glucose, blood urea, and serum creatinine were also estimated to exclude the mentioned cases.

Blood pressure was assessed to exclude hypertensive patients.

The data from both groups were compared using independent student's "t" test. Values were expressed as mean ± SD. Statistical analysis was done using the SPSS, version 12.0. (ANOOVA and LSD), "P" value <0.05 was considered to indicate statistical significance.

Results:

Table 1: Relationship between cigarette smoking/day and serum lipids and lipoproteins

Lipid profile	1-10 Cig/d N=38	11-20 Cig/d N=78	> 20 Cig/d N=24	P-value
Age/ year (mean±SD)	30.12±6.52	31.45±6.92	35.42±6.55	
TC mmol/l (mean ±SD)	4.62 ±0.54	4.88 ±0.50	5.25±0.42	A=0.009** b=0.000** c=0.002**
LDL-C mmol/l (mean ±SD)	3.04±0.54	3.30±0.50	3.66±0.43	A=0.010** b=0.000** c=0.002**
HDL-C mmol/l (mean ±SD)	1.22±0.04	1.21±0.04	1.21±0.05	A=0.391NS b=0.335NS c=0.725NS
VLDL-C mmol/l (mean ±SD)	0.36±0.08	0.37±0.03	0.38±0.05	A=0.320NS b=0.215NS c=0.585NS
LDL-C / HDL-C (mean ±SD)	2.51±0.51	2.72±0.43	3.07±0.44	A=0.017** b=0.000** c=0.002**
TG mmol/l (mean ±SD)	1.81±0.41	1.86±0.13	1.89 ±0.18	A=0.320NS b=0.215NS c=0.585NS

*significant, **highly significant, NS non significant

a: P-value comparison 1-10 cig/d versus 11-20 cig/d, b: P-value comparison 1-10 cig/d versus >20 cig/d, c: P-value comparison 11-20 cig/d versus >20 cig/d,

TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, VLDL-C: very low density lipoprotein cholesterol, TG: triglyceride

Table 1 was showing that the number of cigarette/day increased with advanced age. There was significant increase in the mean levels of total cholesterol, low density lipoprotein, and atherogenic index (LDL-C/HDL-C) among smoking group (1-10 cig/d versus11-20 cig/d,1-10 cig/d versus >20 cig/d, and 11-20 cig/d versus >20 cig/d).

Table 2: Relationship of lipid profile in both control and smoking group

variable	Control group N=100		Smoking group N=140		P-value
	mean ±SD	SE	mean ±SD	SE	
TC mmol/l	4.32±0.43	0.43	4.87±0.54	0.45	P<0.001**
LDL-C mmol/l	2.74±0.48	0.48	3.29±0.53	0.05	P<0.001**
HDL-C mmol/l	1.23±0.07	0.01	1.21±0.04	0.01	P=0.04*
VLDL-C mmol/l	0.35±0.01	0.00	0.37±0.05	0.00	P=0.001**
LDL-C / HDL-C	2.26±0.51	0.05	2.73±0.49	0.04	P<0.001**
TG mmol/l	1.77±0.05	0.01	1.85±0.24	0.02	P=0.001**

*significant, **highly significant

In table 2, there was significant increase in the mean levels of total cholesterol, LDL-C, VLDL-C, triglyceride, and atherogenic index (LDL-C/HDL-C); while there was significant fall in the mean level of HDL-C in the smokers as compared to that in the control group.

Table 3: Distribution of lipoproteins in both control and smoking groups

Variable	Control group n=100	Smoking group n=140		
		1-10 cig/d (n=38)	11-20 cig/d(n=78)	>20 cig/d (n=24)
% LDL-C	63.43	65.79	67.55	69.77
% HDL-C	28.47	26.38	24.84	23.04
% VLDL-C	8.10	7.83	7.61	7.19

Table 3 showed the percentage of LDL-C in control group is lesser than of smoking group and increased with increasing cigarette/day**;** while the percentage of both HDL-C and VLDL-C were higher in control group than smoking group and decreased with increasing cigarette/day.

Discussion:

Almost 3000 years after the ancient Greek saying: ('prevent rather than treat'), prevention and health promotion remain as the foundation of medical practice and research. In this respect, physicians are expected not only to provide their patients with medical care and advice but also to set an example for them.

Our data showed that total cholesterol, LDL-C, VLDL-C, and LDL-C/HDL-C (atherogenic index) were significantly higher in smokers as compared with non-smokers (control group) (table 2). It is revealed that the mean serum total cholesterol in smokers (4.87±0.54 mmol/l) was significantly higher than in non-smokers (4.32±0.43 mmol/l) were

P<0.001. It was also revealed that the mean of total cholesterol in subjects smoking >20 cig/d (5.25±0.42 mmol/l) was significantly

higher than subjects smoking 11-20 cig/d (4.88 ±0.50 mmol/l) and 1-10 cig/day (4.62 ±0.54 mmol/l)(P=0.002, P=0.000 respectively), and in subjects smoking 11-20 cig/d was significantly higher than subjects smoking 1-10 cig/d (P=0.009)(table 1).

In the present study, showed that the mean of LDL-C and VLDL-C in smokers (3.29±0.53 mmol/l and 0.37±0.05mml/l respectively) were significantly higher than in non-smokers (2.74±0.48mmol/l and 0.35±0.01 mmol/l respectively) (P<0.001 and P=0.001 respectively, table 2). But LDL-C was significantly higher in subjects smoking > 20 cig/d as compared to those smoking 11-20 cig/d and those 1-10 cig/d (P=0.002, P=0.000 respectively), and in subjects smoking 11-20 cig/d was significantly higher than subjects smoking 1-10 cig/d (P=0.01), while VLD-C was not significant (see table 1). These data are in tune with the findings of Abbassi et al(17), TC, TG, and LDL-C were significantly increased in smokers, when compared with non-smokers.

This study revealed that the mean serum triglyceride in smoking group (1.85±0.24 mmol/l) was significantly higher than in non-smokers (1.77±0.05 mmol/l)(P=0.001, table 2), but these values were not significant depending on the cigarette smoking/d. It also revealed that the mean serum HDL-C in smoking group (1.21±0.04 mmol/l) was significantly lower than in non-smokers (1.23±0.07 mmol/l)(P=0.04, table 2), but these values were not significant depending on the cigarette smoking/d. These data agree with the findings of Abbassi A which showed LDL-C significantly decreased in smokers as compared with non smokers and TC, TG, LDL-C were significantly increased while HDL-C was significantly decreased with increased number of cigarette/day(17).according to our data, the atherogenic index (LDL-C/HDL) was significantly higher in smokers (2.73±0.49) than in control group (2.26±0.51)(P<0.001, table 2). These values were significantly affected by cigarette smoking/day (see table 1).

Epidemiological studies have shown that long-term morbidity and mortality in coronary heart disease (CHD), manifest over years, is directly related to circulating levels of atherogenic lipoproteins, in particular LDL-C(18). 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- C in the blood stream (19); (b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL – cholesterol(20); (c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL-C, VLDL-C, and TG due to decreased activity of lipoprotein lipase(21).

Our study showed that the percentage of LDL-C was lower in control group than that in smokers and increased with increasing cigarette/day, while the percentage of HDL-C and VLDL-C were higher in control group than that in smokers and decreased with increasing cigarette/day (table 3). It has been suggested that smoking even of short duration and quite moderate consumption of cigarettes is associated with adverse lipoprotein profiles(22).

These findings suggest that smoking alters the lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with the number of cigarette/day smoked. Smoking plays the key role for atherosclerotic process and with

coronary artery disease (23). It should be noted, however, that no case was symptomatic of cardiovascular disease at the time of the examination.

Many studies have reported similar adverse changes in lipids associated with smoking. These observations are in tune with the findings of Cullen *et al*(24) and Neki(25). Venkatesan et al found that total cholesterol and LDL-C were significant increase in smokers while HDL, VLDL, and triglyceride were not vary between two groups(11). Contrary report to this has been documented by Sirisali *et al* who found that total and LDL cholesterol did not vary between smokers and non smokers (26), Mammas et al (27) showed significant increasing in serum triglyceride and fall in LDL-C, while others were non significant. This difference in observation can be due to ethnic differences in the study population(28). These values were affected by the number of cigarette/d; total cholesterol and LDL-C were significantly higher in groups where more cigarette smoking. Adedeji and Etukudo (29) showed significant increasing in total cholesterol and LDL-C among smokers and others were not significant .

We Conclude that Smoking produces adverse effects on lipid profile, therefore increasing the cardiovascular disease risk. The high prevalence of an atherogenic lipid profile in smokers makes them prone to develop premature atherosclerosis and the changes become more marked with the number of cigarette/day smoked and the changes become more marked with the number of cigarette/day smoked.

We recommend It is important to establish a special anti-smoking programmes by the ministry of health through visible and audible communication aids and through schools and colleges explaining risks of smoking on the cardiovascular system and other systems.

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