

The Impact of Cigarette Smoking on Lipid Profile among Iraqi Smokers

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Abstract

Background: Cigarette smoking is a risk factor for peripheral vascular disorders and heart disease. The monitoring of lipid profile is very important to give an estimation of the future cardiovascular diseases among smokers.

Objectives: The objective of this study is to describe the effects of smoking on the lipid profile status among Iraqi smokers as well as to identify the morbidity risk among smokers and non-smokers.

Methods: A cross-sectional study was carried out in the outpatient clinic of the Tikrit General Hospital, Tikrit, Iraq. A total of 143 patients who performed their lipid profile tests in fasting condition were purposively selected.

Results: The findings showed that the mean value of total cholesterol in the smokers group (5.23 ± 1.41 mmol/l) was higher than in non-smokers group (4.55 ± 0.90 mmol/l). There was a significant higher level of total cholesterol, triglycerides, low density lipoprotein (LDL) and very low density lipoprotein (VLDL) in the smokers group compared to non-smokers ($P < 0.001$). While the high density lipoprotein (HDL) was lower in the smokers group compared to non-smokers group. In addition, total cholesterol and LDL were significantly associated with the number of cigarettes smoked per day ($P < 0.001$).

Conclusions: Cigarette smoking is associated with dyslipidaemia among Iraqi smokers. Total cholesterol and LDL may be considered as the main parameters that are affected by the heaviness of smoking. However, preventive strategies are needed to avoid the future cardiovascular diseases and in supporting the benefits of quit smoking.

Keywords: Cigarette, Dyslipidaemia, Lipid profile, Smokers

Introduction

Cigarette smoking is one of the most important modifiable risk factor for atherosclerosis and increasing morbidity and mortality of Chronic Heart Diseases (CHD).^{1,2} Although the precise mechanism of tobacco smoke role in the atherosclerotic process remains not fully understood, several chemicals among thousands that exist within tobacco smoke produce harmful and toxic effects on health.³ Many changes which could promote atherosclerosis in chronic tobacco smokers have been reported by many research

workers, whereby these changes include; alternation of lipid profile,^{4,5} increased oxidative LDL-C,⁶ decreased nitric oxide (NO) availability,^{7,8} endothelial dysfunction,⁹ increased insulin resistance,¹⁰ alternation in fibrinolysis,¹¹ platelet dysfunction,^{12,13} high blood viscosity, on-going inflammation with increasing inflammatory markers,^{14,15} and more recently free radicals-mediated oxidative stress appear to play an important role in mediation of athero-thrombotic disease in chronic smokers.¹⁶⁻¹⁸ Cigarette smoking increases plasma catecholamine which induces lipolysis and release of free fatty acid,¹⁹ which will be taken up by the liver. Atherosclerosis has been described as a lipid driven inflammatory disorder of the arterial wall.^{20,21} 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.²² Previous research workers have reported that tobacco smoking is associated with increased levels of total cholesterol, triglyceride, LDL-C, VLDL and decreased level of HDL-C.^{5,23-25} However, other studies were reported with conflicting results.^{26,27} It seemed that cigarette smoking could promote atherosclerosis, in part, by its effect on lipid profile.^{4,28,29} It is also found that the risk of development of CVD is directly related to the number of cigarettes smoked.^{30,31} The non-HDL-C is a progressing parameter which includes all potentially atherogenic apoB -containing lipoprotein particles including LDL-C, IDL-C and VLDL. It is more strongly related to risk for atherosclerosis of coronary heart diseases.^{32,33} Although many studies have been done worldwide to identify the lipid profile among smokers, but we have little evidence about the impact of smoking on lipid profile among Iraqi smokers. The present study aims to demonstrate the effects of smoking on lipid profile among Iraqi smokers and to identify the differences between the lipid profiles of smokers based on the heaviness of smoking.

Methods

Study design

A cross-sectional descriptive study was conducted at the outpatient clinic of the Tikrit General Hospital, Tikrit, Iraq. This outpatient clinic is the main general clinic in the city, where a considerably large population is attending. The study was carried out from April–May 2014.

Study sample

Adult smokers and non-smokers in both genders were invited to participate in this study. Smokers who reported smoking cigarettes everyday were eligible for this study. Smokers and non-smokers who attended the outpatient clinic as a patient, attendant, volunteer and staff were selected to participate in the study. However, those who reported having hypertension, diabetes, renal or hepatic diseases were excluded. In addition, people who are using alcohol, drug therapy altered lipid profile and ex-smokers were excluded from this study. A convenience sampling with purposive method was used to select the participants from different age groups. In total, 143 participants were approached consecutively as being eligible for the inclusion criteria in the study.

Procedure

Approval for this study was obtained from the Medical Committee of Tikrit Teaching Hospital. The interview approach was done to participants who were present at the outpatient clinic of Tikrit Teaching Hospital, Tikrit. After explaining the aims of the study to all of the participants, a written informed consent was obtained. A self-administered questionnaire was used to obtain the demographic data of the participants.

The smoking history as well as their medical history were also obtained in case if they are suffering from chronic diseases or taking medications for lipid lowering. In addition, all participants were asked to perform their blood test in fasting condition for at least 12 hours before collecting the blood sample. All blood samples were tested at the hospital laboratory in Tikrit Teaching Hospital. The LDL-cholesterol was calculated using Friedewald's equation.

$LDL-C = TC - HDL-C - Triglyceride / 2.2$ in mmol/l

$Non-HDL-C = TC - HDL-C$

Blood samples with Triglyceride concentrations above 4.5 mmol/l were excluded. The normal ranges of lipid profiles and the morbidity risk was determined based on the Report of the National Cholesterol Education Program (NCEP).³⁴

Data analysis

The results were analyzed using the Statistical Package for Social Sciences (SPSS, version 18). Data were described using the mean and standard deviation for the significant differences between groups. T-test was used to compare the differences of lipid profiles between smokers and non-smokers. In addition, ANOVA test was performed to find the differences of lipid profiles among three groups of smoking intensity. Furthermore, the Chi-square test was used to evaluate the association between variables. All statistical tests were considered significant in p-value of <0.05 with a confidence level of 95%.

Results

From Table 1, the results showed that there was a significant difference between smokers and non-smokers in the mean values of total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol and non-HDL cholesterol. Smokers had higher levels of total cholesterol, triglycerides, LDL-cholesterol and VLDL-cholesterol and lower levels of HDL-cholesterol and non-HDL-cholesterol compared to the non-smokers group.

According to Table 2, the heaviness of smoking was associated with an increasing in the levels of total cholesterol, LDL-cholesterol and non-HDL-cholesterol. Smokers who reported smoking more than 20 cigarettes per day had higher levels of total cholesterol, LDL-cholesterol and non-HDL-cholesterol.

From Table 3, it can be seen that there is a significant association between smokers and non-smokers according to the normal levels of serum lipid profiles. Smokers were more likely to have high morbidity risk in their lipid profiles compared to the non-smokers group.

Discussion

Cigarette smoking is associated with reduced HDL-C level by alteration of the critical enzymes of lipid transport lowering lecithin-cholesterol acyltransferase (LCAT) activity and altering cholesterol ester transfer protein (CETP) and hepatic lipase activity.³⁵ {McCall, 1994 #84} In the present study, the results showed that the serum level of total cholesterol, triglyceride, LDL-C, and VLDL-C were significantly higher in smoker as compared to non-smokers. The serum level of HDL-C was significantly lower in smokers compared to nonsmokers. Previous studies have reported the same findings that smokers have a higher risk lipid profile than non-smokers.^{24,25,36} On the contrary, a

study by Dirican et al. reported that there was no significant difference in lipid profile among smokers and non-smokers.²⁶

However, others studies indicated that smoking is associated with an increasing in the levels of total cholesterol, triglyceride, LDL-C, VLDL and decreasing in the level of HDL-C.^{5,36} Chronic smokers might be the cause of the increased levels of total cholesterol, triglyceride, LDL-C and VLDL-C due to a decrease in lipoprotein lipase activity.^{10,37} This is being expected to increase the synthesis of VLDL-C which is consistent with the changes reported above in lipid profile of cigarette smokers. In addition, HDL-C became susceptible to oxidative modifications by cigarette smoking and loses its atheroprotective properties. Therefore, other studies showed that smoking cessation leads to normal values of HDL-C to be as alike in non-smokers.^{38,39} The non-HDL fraction which is considered as a strong predictor for risk of CHD has increased in smokers as compared to non-smokers with a highly significant difference.⁴⁰⁻⁴² This indicates the presence of higher level of atherogenic lipoprotein particles in smokers as compared to non-smokers. This indicator can be used to evaluate the morbidity risk among cigarette smokers especially because it can be estimated at any time without the fasting samples.

According to the differences between lipid profile level and the number of cigarette smoked per day, our results reported that the level of total cholesterol, LDL-C and non HDL-C were associated with the increase of smoking intensity. These findings are in accordance with observation of Afroz et al. who reported that the increasing levels of atherogenic lipoproteins notably LDL and IDL (estimated within the non-HDL-C) in association with increased intensity of smoking is most probably leading to produce higher levels of oxidized LDL-C through an increased in oxidative modification of LDL-C reported in smokers.^{6,29,43} This seems to play an important role in increasing the risk of atherosclerosis and CHD.⁴⁴ Another study on hyperlipidmic rabbit model reported that the injection of cigarette smoke extract accelerated atherosclerosis through oxidative modification of LDL.⁴⁵

In addition, this study showed that there was no significant difference in the level of VLDL-C and HDL-C accompanied with increased smoking intensity. Besides that, the findings indicated that the significantly lower level of HDL-C among smokers reaching the morbidity level is related to light smoking and there is no further significant decrease by increasing the smoking intensity. The lower level of HDL-C (anti-atherogenic lipoprotein) in light smokers could explain the early sign of cardiovascular diseases reported in people who smoke fewer than five cigarettes per day.^{3,40,41} This study also showed a significant percentage of smokers are within the level of high morbidity risk of total cholesterol, triglyceride, low density lipoprotein and high density lipoprotein with statistically significant difference. This is more likely to explain the role of tobacco smoking in increasing the risk of atherosclerosis and CHD among smokers.

Conclusion

Cigarette smoking causes an alternation of lipid profile which includes increasing the levels of total cholesterol, triglyceride, LDL-C, VLDL-C and non-HDL-C with a decrease in HDL-C level. The increasing intensity of smoking has led to more increase in atherogenic lipoprotein without a further decrease in anti-atherogenic lipoprotein (HDL-C).

Light smoking is enough to decrease the anti-atherogenic lipoprotein to the level of morbidity risk. Non-HDL-C is a valuable indicator to predict the risk of CHD among smokers. All of the above findings support a need of an educational program about the risks of cigarette smoking. In addition, the health policy-makers should take an active

role in planning strategies to increase the awareness of the health risk of smoking and to illustrate the role and risk of smoking in atherosclerosis and applying the suitable smoker quitting programs in Iraq. Applying an effective strategy for quitting of smoking is important for reducing the risk of CHD among Iraqi smokers.

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Conflict of Interest

No

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Table 1: Comparison of serum lipid profiles among smokers and non-smokers in (mean \pm SD).

Lipid profile	non-smokers (N=72)	Smokers (N=71)	P-value
Total cholesterol mmol/l	4.55 \pm 0.90	5.23 \pm 1.41	0.001*
Triglycerides mmol/l	1.72 \pm 0.75	2.29 \pm 1.12	0.001*
HDL-cholesterol mmol/l	1.34 \pm 0.37	1.04 \pm 0.22	<0.001**
LDL- cholesterol mmol/l	2.42 \pm 1.11	3.14 \pm 1.36	0.001*
VLDL- cholesterol mmol/l	0.38 \pm 0.18	0.45 \pm 0.22	0.001*
Non-HDL cholesterol	3.20 \pm 1.08	4.19 \pm 1.50	<0.001**

*P value <0.01=very significant difference.

**P value <0.001=highly significant difference.

Table 2: Serum lipid profiles among smokers according to the intensity of smoking.

Smokers (Daily Smoke)

N=71

Lipid profile	Mild smokers	Moderate smokers	Heavy smokers	P-value
	1-10 cigarettes	11-20 cigarettes	>20 cigarettes	
Total cholesterol mmol/l	4.45 ± 0.94	4.91 ± 1.07	6.18 ± 1.52	<0.001*
Triglycerides mmol/l	2.17 ± 0.78	2.04 ± 1.19	2.62 ± 1.27	0.168
HDL-cholesterol mmol/l	1.00 ± 0.19	1.07 ± 0.23	1.04 ± 0.24	0.506
LDL- cholesterol mmol/l	2.46 ± 0.99	2.90 ± 1.25	3.94 ± 1.37	<0.001*
VLDL-cholesterol mmol/l	0.43 ± 0.15	0.40 ± 0.23	0.52 ± 0.25	0.168
Non-HDL-cholesterol mmol/l	3.45 ± 1.00	3.83 ± on-HDL- cholesterol L(HDL- 1.10	5.13 ± 1.69	<0.001*

*P value <0.001=highly significant difference.

**P value <0.01=very significant difference.

Table 3: The risk of morbidity level among smokers and non-smokers classified according to ATP III of NCEP.

Lipid profile	Non-smokers	Smokers	P-value
	N (%)	N (%)	
Total cholesterol mmol/l			
Normal value	52 (72.2%)	41 (57.7%)	0.003*
Moderate morbidity risk	18 (25.0%)	15 (21.1%)	
High morbidity risk	2 (2.8%)	15 (21.1%)	

Triglycerides mmol/l			
Normal value	54 (75.0%)	44 (62.0%)	0.088*
Moderate morbidity risk	18 (25.0%)	24 (33.8%)	
High morbidity risk	-	3 (4.2%)	
HDL-cholesterol mmol/l			
Normal level	54 (75.0%)	34 (47.9%)	0.001*
Low level	18 (25.0%)	37 (52.1%)	
LDL-cholesterol mmol/l			
Normal value	49 (68.1%)	47 (66.2%)	0.020*
Moderate morbidity risk	17 (23.6%)	8 (11.3%)	
High morbidity risk	6 (8.3%)	16 (22.5%)	

*P value <0.05=significant difference.