Influences of smoking on lipid profile in Iraqi men

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Abstract

Cigarette smoking is generally considered as associated with increased risk of a variety of medical disorders. Several studies provide the evidence that a smoking is strongly associated with change the normal status of the lipid profile. The present study aimed to compare the lipid profile between smokers and nonsmokers and to determine the influence smoking on the lipid profile in our community. Forty smoking males and thirty non-smoking males from different areas of Babylon governorate were included in this study. Their age was ranged between 20 and 40 years. Fasting blood was withdrawn from all groups and used for determination of lipid profile [Cholesterol, Triglyceride (TG), High density lipoprotein (HDL) and low density lipoprotein (LDL)]. Significantly increased of serum Cholesterol, Triglyceride and low-density lipoprotein with significant decreased in serum High density lipoprotein level in smokers as compared to nonsmokers. These results showed that the mean levels of TC, TG and LDL were increased with duration of smoking while HDL showed a decreased with the increase duration of smoking. This study concluded that smoking made alteration in lipid profile, related in the alteration lipid levels associated with increases risk of coronary artery disease.

Keywords: smoking, Lipid profile, Coronary artery disease

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Introduction

Smoking is the single largest preventable cause of disease and premature death in the world. Smoking found to harm nearly every organ and system in the body and diminishes a person's overall health. Smoking-related deaths are mainly due to breathing problems, chronic obstructive pulmonary disease (COPD) and heart disease. Smoking is the most leading cause of cancer and death from cancer. It causes cancers of the lung, esophagus, larynx, throat, kidney, mouth, bladder, liver, pancreas, stomach, colon, cervix and rectum, as well as acute myeloid leukemia [1, 2]. Smoking is one of the environmental factors which can alter the normal lipid profile. It 's one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease [3]. In addition, smoking causes inflammation and impairs immune

function [1]. Second hand smoke also causes disease and premature death in nonsmoking adults and children[2, 4]. Lipids play an important role in all aspects of biological life. Some of these roles include serving as hormones or hormone precursors, helping in digestion, storage function, providing energy, and metabolic fuels; cting as functional and structural compounds in biomembranes and forming insulation to allow nerve conduction or to prevent heat loss [5]. Cigarette smoking (CS) is generally considered as associated with increased risk of a variety of medical disorders. Cigarette smoke is a complex mixture of over 4800 identified constituents that include high concentrations of free radicals, reactive oxygen and nitrogen species, reactive aldehydes, and diverse metals [6]. Several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile [7]. Cigarette smoking continues to be a major health hazard, and it contributes significantly to cardiovascular morbidity and mortality. CS impacts all phases of atherosclerosis from endothelial dysfunction to acute clinical events, the latter being largely thrombotic. The mechanism by which smoking increases the cardiovascular diseases are unclear. Recently it has been suggested that smoking adversely affects the concentration of plasma lipids and lipoprotein levels. The toxic components of cigarette smoke and the mechanisms involved in CS-related cardiovascular dysfunction are largely unknown, while CS increases inflammation, thrombosis, and oxidation of LDL. Recent studies support the hypothesis that cigarette smoke exposure increases oxidative stress as a potential mechanism forainitiating cardiovascular dysfunction [5]. Nicotine and other toxic components 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 such as TC, TG and LDL circulating in the blood vessels and decreases the amount of good fat such as HDL. The toxic components of cigarette smoke damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a nonsmoker [8]. Nicotine induces oxidative stress, which leads to the formation of free radicals, and they attack the membrane of lipids and this consequently leads to the formation of malondialdehyde (MDA) which causes tissue damage [9]. Smoking is now increasing rapidly throughout the developing world and it is one of the biggest threats to current and future health so the purpose of our study was undertaken to clarify further the influence smoking on the lipid profile in our community.

Materials and Methods

This study had been conducted between September 2015 and April 2016. The present study included 70 healthy men volunteers from Babylon University (40 smoking male subjects and

30 nonsmoking male subjects). They were free of diabetes mellitus, hypertension, cardiovascular, and immune diseases and they have received no medications. The smokers in this study was those smoking 4-10 cigarette/day. Their age was ranged between (20-40) years. Ten ml of blood was withdrawn from each volunteer, and then it was collected in a container with no anticoagulant. The blood sample was placed at room temperature for 10 minutes. The serum was then being separated by centrifuge at 3000 rpm for 10 minutes. The sera was stored at -4C° till analysis. Serum Cholesterol, Triglyceride, HDL and LDL was measured by using the enzymatic method [10].

Statistical analysis

Results are presented as mean ± standard deviation (SD). The statistical significance was accepted when p value at <0.05. All analyses were carried out using the Statistical Package for the Social Sciences (SPSS) 15.0 software**.**

Result and Discussion

Lipid profiles of smokers and non-smokers were shown in able 1. All the components of lipid profile studied (Cholesterol, TG, LDL) were found significantly increased for smokers compared to the healthy control non-smoking subjects, while the HDL were decreased in smoker group compared to the non smoker group. The values of significance for various comparisons given in the table 1.

Table1.
Serum Lipid profile of smokers and non-smokers represented as mean ± SD.

Levels of Lipid profile (mg/dL)	**Smokers n= 40**	**Non mokers n= 30**	**p value**
T C	200.32±33.62	172.0±12.06	< 0.01*
T G	179.55 ±61.52	94.30±11.85	< 0.001*
H DL	29.76±3.31	55.80±4.44	< 0.001*
LDL	116.35±41.64	64.60±18.50	0.000*

*Statistically significant (p<0.05).

Table 2.
Heart disease markers in smokers and non-smokers

Marker of heart disease	Smokers n=n40	Nonsmokers n=n30
LDL/HDL	3.9+1.2	1.15+0.41
TC/HDL	6.73±1.01	3.08+0.27
TG/HDL	6.03±1.8	1.68+0.266

*Statistically significant (p<0.05).

The results in table 1 showed that smoking is significantly increased with serum TC, TG and LDL and significantly reduction in level of HDL in smokers when compared to nonsmokers at ($p<0.05$). These results were analogous with the other study [11].

Table 2 shows the ratio of lipid fractions between smokers and nonsmokers. There were significant differences in all the ratios of lipid fractions in smokers when compared with nonsmokers at p-values (<0.05). CS is one of the leading causes of preventable morbidity and mortality that usually starts in adolescence and continues in adult life [12].

In the developing world, [13].The smoking is responsible for premature development of cardiovascular disease. The results in this study showed a statistically significant different in the total cholesterol level (TC) of smokers ($p<0.05$) when compared with non-smokers, this indicate that the smoking have increased serum concentration of cholesterol than non-smokers. The result of our study is in the same with study of Guedes *et al*. where high concentration of cholesterol was recorded in smokers when compared with the nonsmokers [14-17]. Increased cholesterol is a causative factor in the etiology of atherosclerotic disease. In the present study, serum TC, TG and LDL were significantly higher in smokers as compared to non-smokers. The serum HDL level was significantly lower in smokers as compared without-smokers. Our results are in accordance with the results of many research workers. The change in the serum cholesterol and lipoprotein levels become more marked with duration of smoking in years. Diricana et al [15] did 't find significant differences in serum TG, LDL and HDL levels between smokers and nonsmokers. High density lipoprotein (HDL) of smokers was significant lower when compared with non-smokers' in this work that mean that smoking are caused to developing coronary heart disease earlier in smokers than their non-smoking groups, also reported high level of LDL in smokers, suggesting that there is increased LDL-Cholestarol synthesis in smokers which is dangerous to their health. LDL/HDL ratio was significantly higher in smokers as compared to that of nonsmokers. The result agrees with that study of other study [18]. This ratio is an index of possibility of developing coronary heart disease (CHD) in smokers. In addition, the TG/HDL and TC/HDL ratio were significantly higher in smokers ($p<0.05$) than in nonsmokers. These ratios are useful as markers of disease risk and indicate very high risk of CHD [19, 20]. The main objectives of this study are that important factors which may contribute to the cardiovascular risk factors among youth.

Conclusion

The findings in this study suggest that smoking might be related in the alteration in lipid profile adversely causing dyslipidemia in smokers. Smoking plays the main role for atherosclerotic process and with coronary artery disease. This study is helpful for future

studies in understanding the underlying mechanism causing series of changes influenced by smoking activity. The duration of smoking play an important role in lipid profile change. The results documented a high prevalence of dyslipidemia among Iraqi smokers. The results of this study show that smokers are at much greater risk of developing atherosclerotic plaques and different heart diseases than nonsmokers among Iraqi young.

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