The Correlation between Lipid Profile and Smoking

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Abstract

The objectives were to find out the alterations in serum lipid status among adult male smokers and non-smokers in Maysan Province, south of Iraq, as well as to evaluate the relationship between the heaviness of smoking and dyslipidemia in smoker subjects. A comparative study was achieved on 160 men with aged 30-63 years which were grouped to smokers and nonsmokers, among them 120 individuals were smokers and the other 40 were nonsmokers. According to the intensity of smoking, the smoker group is divided again into three subgroups: mild (A), moderate (B), and heavy (C) smokers. The concentrations of total cholesterol (TC), high density lipoprotein-c (HDL-c), triglycerides (TG), low density lipoprotein-c (LDL-c) and very low density lipoprotein-c (LDL-c) were measured and calculated. The results revealed that concentrations of serum lipid profiles in smokers were significantly higher than that of nonsmokers except in case of high density lipoprotein-c which was significantly reduced among smokers. In addition to that the lipid profile had a significant correlation with the cigarette numbers of that smoked daily; the mean values of all lipid fractions except HDL-c were noticeably increased from group A to group C, while they were decreased in the case of HDL-C. There were significantly different between nonsmokers and different groups of the smoker, the values of TC of groups A, B, and group C were (178.6, 181.7, and 183.5) mg/dl respectively. According to similar arrangement of above smoker groups, the mean values of TG were (238.4, 245.2, and 248.7) mg/dl. In case of HDL- c, they were (31.5, 28.4, and 26.8) mg/dl. Among LDL-c level, they were (194.8, 203.8, and 204.9) mg/dl. In VLDL- c level, they were (47.7, 48.2, and 50.5) mg/dl. The findings of the study showed that smokers (especially heavy smokers) are at much greater risk of initiating atherosclerotic plaques and various heart diseases than non-smokers.

Keywords*: Lipid Profile, smokers, non-smokers, TC, HDL-c, TG, LDL-c, and LDL-c, Maysan.*

Introduction

The lipids are playing a crucial role in all parts of biological life. Some roles involve acting as hormones and hormone precursor, using in digestion, producing energy, storing functions, and metabolic fuel; serving as structural and functional substances in biological membranes and forming padding to permit neuron transfer or to prevent the losing of heat [1]. The blood test that used to investigate the levels of serum Tc, TGs, LDL-c, HDL-c, and VLDL-c is called lipid profiles [2]. The smoking is an essential risk factor causing atherosclerosis, and the disease of coronary artery [3]. Several studies have recorded increase in blood TC levels among people who repeatedly having smoking and decrease in TC levels among individuals leaving smoking. Further researches also showed that smoking decreases HDL-c levels, causing increased risks of heart disease. Smoking leads to an elevate in LDL-c and TG levels. Several studies have shown a dose-dependent relationship between smoking and serum lipid profile [4]. The mechanism by which the smoking changes the metabolism of lipoprotein is not completely understood. Numerous mechanisms assumed are stimulation of the sympathoadrenal system by nicotine leading to lipolysis and raised serum free fatty acid level which is causing an increase the synthesis of VLDL from the liver. A high intake of free fatty acid by heart leads to increased myocardial oxygen demand. Depressive acting of smoking on estrogen levels leads to decreased HDL-c. Smokers are thought to eat a diet that rich in fat and

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lipids and lesser in fibers as well as cereals [5]. Nicotine stimulates the releasing of catecholamine leading to activate the adenyl cyclase of the adipose tissue causing an increase in lipolysis, elevated concentration of free fatty acids of plasma, secreting hepatic triglycerides and hepatic free fatty acids along with VLDL-c in bloodstream [6]. Although the exact mechanism of tobacco smoke that having role in the process of atherosclerotic and coronary artery disease remains not wholly clear, many chemicals that is found in tobacco smoke cause risky effects on the health[7]. In the World Health Organization's Western Pacific Region (WHO WPR), being born males is the unique highest risky indicator for tobacco usage [8]. The prevalence of male smoking was (62.3%) and the rates of increasing are the greatest in world [9]. Atherosclerotic alterations that found in the middle of age initiate in childhood and the mechanisms may linked to irregular levels of risky factors such as serum lipid profiles and smoking, which are supposed to be associated with initially stages of atherosclerosis and coronary artery disease. In adults, increased level of LDL-c and decreased level of HDL-c and its main subfractions are correlated with myocardial infarction or risk factors for coronary heart disease (MI)[10,17&19]. Although many researches have been achieved globally to investigate the lipid profile status in smokers, there were a little data concerning the influence of smoking on lipid profiles in Iraqi smokers [11]. The current study has been achieved to reveal the correlation between smoking and lipid profiles among smoker persons in Maysan Province, south of Iraq and to estimate the p-value between lipid profile tests of smokers and nonsmokers based on the intensity of smoking.

Materials and Method

The present study was a comparative study, carried out from October 2019 to February 2020 at Al. Sadr Teaching Hospital in Maysan Province. The total samples of this study were consisting of 160 adult males; out of them 40 individuals were non-smokers and the other 120 were smokers since at least 15 years duration, aged between 30-63 years. The smokers were divided into three groups: (mild, moderate, and heavy smokers) depending on heaviness (intensity) of their smoking.

Group A: Mild smokers = 20-34 cigarettes per day (N =40)

Group B: Moderate smokers = 35-49 cigarettes / day (N =40)

Group C: Heavy smokers = 50-64 cigarettes / day (N =40)

Under the aseptic condition, about five ml of fasting (12-14 hours) venous antecubital blood samples were collected from each individual in a test tube and waiting for at least 30 minute until blood were clotting. Then the sera were centrifuged up to 4000 round per minute for 10 minutes. Sera were stored at -20°C until tested. Total cholesterol, triglycerides and high density lipoprotein-c were tested by standard enzymatic kit of Human Ltd while low density lipoprotein-c and very low density lipoprotein-c were estimation by calculated method of Friedewald. All data were analyzed by using (SPSS) version (V. 18.0).

Results

Table 1 demonstrates the difference in means ± standard deviation (SD) of lipid levels between smokers and nonsmokers. All the levels of lipid profile were found to be higher with significant values among the smokers compared to the nonsmokers ($p < 0.05$) except HDL-c levels, it was lower among smokers with significant p-value ($p < 0.05$).

Table 1: Difference in lipid levels between smokers and nonsmokers

parameters N.	Lipid Levels (*)				
	Total cholesterol	Triglyceride	HDL-c	LDL-c	VLDL-c
Smokers (N = 120)	181.3±46.7	244.1±67.5	28.9 ±7.8	201.2±49.5	48.8±14.2
Nonsmokers (N = 40)	150.4±30.6	163.4±63.9	50.6 ±7.8	132.5±35.6	32.7±12. 8
t-value	3.9	6.6	13.2	8.1	6.4
p-value	0.000	0.000	0.000	0.000	0.000

*The units of all types of lipids are measured with mg/dl

The table 2 show the degree of smoking was inversely proportional to HDL- c levels i.e. the level of HDL- c decreased as the smoking degree increased.

Table 2: Difference between nonsmokers and the groups of smokers in lipid profile

Lipid profile parameters	Nonsmokers (N = 40)	smokers			
		Group (A) N = 40	Group (B) N = 40	Group (C) N = 40	Total N = 120
TC mg/dl	150.4±30.6	178.6±36.5	181.7±55.1	183.5±47.9	181.3±46.7
TG mg/dl	163.4±63.9	238.4±64.9	245.2±69.3	248.7±69.7	244.1±70.8
HDL- c mg/dl	50.6 ±11.8	31.5±4.9	28.4±9.3	26.8±7.8	28.9 ±7.8
LDL- c mg/dl	132.5±35.6	194.8±40.9	203.8±58.8	204.9±54.7	201.2±49.5
VLDL- c mg/dl	32.7±12. 8	47.7±12.9	48.2±14.9	50.5±14.6	48.8±14.2

The Analysis of variance (ANOVA) in table 3 was done between the different groups for TC. It was found that the F value was significantly more for comparison between smokers and nonsmokers.

Table 3: Difference between various groups of smokers and nonsmokers in total cholesterol

The Groups	Total Cholesterol		Group of Smoker		
	The range	Mean±SD	Group A	Group B	Group C
Nonsmokers	140.6-160.2	150.4±30.6	P <.004	P <.002	P <.001
Group A	166.9-190.3	178.6±36.5	-	Ns	Ns
Group B	164.1-199.3	181.7±55.1	-	-	Ns
Group C	168.2-198.8	183.5±47.9	-	-	-

One way ANOVA (F = 5.12, P <0.002), Ns: Non significant

One way ANOVA in table 4 was done between the different groups for TG. It was found that the F value was significantly more for comparison between smokers and nonsmokers but not across the degrees of the smokers.

Table 4: Difference between various groups of smokers and nonsmokers in Triglycerides

The Groups	Triglycerides		Group of Smoker		
	The range	Mean±SD	Group A	Group B	Group C
Nonsmokers	142.9-183.8	163.4±63.9	P <.000	P <.000	P <.000
Group A (mild)	217.7-259.1	238.4±64.9	-	Ns	Ns
Group B (Moderate)	217.3-265.1	241.2±74.9	-	-	Ns
Group C (Heavy)	229.2-276.1	252.7±73.2	-	-	-

One way ANOVA (F = 13.8, P <0.000), Ns: Non significant

One way ANOVA in table 5 was done between the different groups for HDL- c. It was found that the F value was significantly more for comparison between smokers and nonsmokers and It was found to be statically significant difference between group A and group C, while it was not significant between group A and group B and between group A and group C.

Table 5: Difference between various groups of smokers and nonsmokers in HDL- c

The Groups	HDL-c		Group of Smoker		
	The range	Mean±SD	Group A	Group B	Group C
Nonsmokers	46.8-54.3	50.6±11.8	P <.000	P <.000	P <.000
Group A (mild)	29.9-33	31.5±4.9	-	Ns	P <.01
Group B (Moderate)	25.4-31.4	28.4±9.3	-	-	Ns
Group C (Heavy)	24.3-29.3	26.8±7.8	-	-	-

One way ANOVA (F = 62.1, P <0.000), Ns: Non significant

The table 6 the Show that the LDL- c and VLDL- c value was significantly different between nonsmokers and all groups of smokers. But it was not different between the groups of smokers.

Table 6:Demonstrates difference between smoker groups and nonsmokers in LDL-c and VLDL- c.

Groups	LDL-c		VLDL-c		Group of Smoker		
	The range	Mean±SD	The range	Mean±SD	Group A	Group B	Group C
Nonsmokers	121.1-143.9	132.5±35.6	28.6-36.8	32.7±12. 8	P <.000	P <.000	P <.000
Group A	181.9-207.7	194.8±40.2	43.5-51.8	47.7±12.9	-	Ns	Ns
Group B	183.7-219.3	201.5±55.6	43.5-53	48.2±14.9	-	-	Ns
Group C	190.7-223.8	207.3±51.8	45.8-55.2	50.5±14.6	-	-	-

LDL-c: One way ANOVA (F = 22.3, P <0.000), Ns: Non significant

VLDL-c: One way ANOVA (F = 13.9, P <0.000), Ns: Non significant

Discussion

The present study has shown that the mean values of total cholesterol, triglycerides, low density lipoprotein-c, and, very low density lipoprotein-c were expressively higher in smokers when compared to nonsmokers. Simultaneously the levels of HDL-c were considerably lower in smokers comparing to nonsmokers (Table 1), these findings in accordance with this study [1, 11, & 12]. Among the various groups of total cholesterol in nonsmokers, the mean values of groups A, B, and C were in the range of (150.4, 178.6, 181.7, and 183.5) respectively. The values of TG were in the range of (163.4, 238.4, 245.2, and 248.7) among nonsmokers, group A, B and group C respectively. Further parallel raise is seen in LDL-c level from nonsmokers, group A, B and C in the range of (132.5, 194.8, 203.8, and 204.9) respectively. VLDL-c level also increased substantially in nonsmokers, group A, group B and group C, in the range of (32.7, 47.7, 48.2, and 50.5) respectively. Inversely, mean value of HDL-c level was decreased in the range: (50.6, 31.5, 28.4, and 26.8) mg/dl from nonsmoking groups A, B and C respectively. The decrease in HDL-c in smokers explained by the smoking-induced increase

catecholamine release, causing an increase in VLDL-C and a decrease in HDL-c concentrations also promotes Coronary Heart Disease and atherosclerosis by lowering HDL-c and increasing the LDL-C which further may lead to vascular endothelium damage[18]. As above results, there was a significant elevation in the level of TC, TG, LDL-c, VLDL-c and decline in HDL-c level among different groups of smoking in regarding to increase in the intensity of the smoking (Table 2). These findings were agreed with the several studies [1, 12, 14 &15]. From table3 to table 6, p-value was with highly significant(P <.000) between nonsmoker and various groups of smokers in all lipid values, while there was no significant within the smoking groups except in HDL-c levels, It was found to be significant between group A and group C (P <.01). These results were in line with the findings of many studies [1, 11, 12, & 16]. The mechanisms by which smoking causes the above observed dyslipidemic alterations are still not fully understood. High serum low density lipoprotein-c and low serum High density lipoprotein-c are correlated with an increased risk of atherosclerosis. Raised levels of HDL-c are indicative of decrease efflux of cholesterol from arterial wall thus favoring atherosclerosis and Chronic Heart Diseases (CHD)[12]. The rise in lipid levels in smokers explained by the mechanism: Cigarette smoking causes absorption of nicotine into the body which leads to lipolysis and release of free fatty acids into the bloodstream via activation of adenyl cyclase in adipose tissue by nicotine stimulated secretion of catecholamines, increased of free fatty acids in the liver give rise to increased hepatic Triglyceride and VLDL synthesis, so thus increasing the concentration of Triglyceride and VLDL-C in blood[18].

Conclusion

The study clearly showed a considerable correlation between the elevation of lipid levels and smoking. It has been found that the alterations in the serum lipid profiles tend to be elevated with the increase in the heaviness of smoking. The risk of an increase in serum levels of TC with an increase in low-density lipoprotein-c and a reduced in levels of high-density lipoprotein-c assumes great importance since it has been the patterns correlated with CHD. The declined levels of high-density lipoprotein-c in smokers and the raised exposure of the vascular endothelium to actively atherogenic lipids as a result of impairment of clearance of triglyceride-rich lipoproteins could produce a mechanism whereby smoking predisposes to a larger risk of developing atherosclerotic plaques and CHD.

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Ethical Clearance: Permission to conduct this study was issued by the Health institutional and the collection of Blood samples of individuals was carried out by under public health technician supervision.

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