Cigarette Smoking and the Accompanied Alterations in Lipid Profile in a Group of Students in Al-Mustansiriyah University

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Abstract

This study was carried out to assess further the association between cigarette smoking and the accompanied alterations in lipid profile. Forty smokers and thirty non smokers were included in our study; all volunteers were males of comparable ages. The enzymatic methods were employed to determine the concentrations of total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein and very low-density lipoprotein. The mean levels of total cholesterol, triglycerides and very low-density lipoprotein were significantly increased in smokers when compared to non smokers exhibiting a direct dose response relationship. Only insignificant elevation was noticed in the mean level of low-density lipoprotein. The anti atherogenic high-density lipoproteins mean level was significantly lower in smokers than in non smokers. Risk ratios were calculated as TC/HDL-C, LDL-C/ HDL-C and non- HDL-C/ HDL-C and were observed to be significantly higher in smokers than in non smokers. These findings confirm and extend those of various studies in other countries. The observed alterations in lipid profile in smokers along with high risk ratios do indicate that smoking produces adverse effects on lipid profile, therefore increasing the atherosclerotic disease risk.

الخلاصة

إن الهدف من هذا البحث هو دراسة تأثير تدخين السجائر على صورة الشحوم الكلية (lipid profile) في مصل الدم، ضمت الدراسة أربعين مدخنا وثلاثين شخصا من غير المدخنين وكانوا جميعا من الذكور و بأعمار متقاربة. تم استخدام الطرق الإنزيمية لقياس تركيز كل من الكوليستيرول الكلي TC، الدهون الثلاثية TG، البروتينـــــات

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الشحمية عالية الكثافة HDL ، البروتينات الشحمية واطئة الكثافة LDL، البروتينات الشحمية ذات الكثافة الواطئة جداVLDL في جميع العينات.

إن معدلات الكوليستيرول الكلي TC,TG,VLDL أظهرت ارتفاعا معنويا لدى المدخنين عند مقارنة نسبهم بغير المدخنين بينما كان الارتفاع غير معنوي في معدل LDL عند المدخنين إذا ما قورن بغير المدخنين. من جهة ثانية فقد لوحظ انخفاضا معنويا في تركيز HDL لدى الأشخاص المدخنين مقارنة بتركيزه عند غير المدخنين.

تم في هذا البحث حساب نسبة الخطورة وهي TC/HDL-C و LDL/HDL-C و TC-HDL-C/HDL-C وقد لوحظ أنها تكون أعلى بصورة معنوية لدى المدخنين مما هي عليه عند غير المدخنين.

إن التغيرات الملاحظة في صورة الشحوم الكلية بالإضافة إلى نسب الخطورة المرتفعة تشير إلى أن التدخين يعمل على إحداث تغيرات تؤثر سلبا في مستوى الشحوم العام وبالتالي فان هذا يرفع من خطر الإصابة بأمراض تصلب الشرايين.

Introduction

Cigarette smoking is a socially accepted health problem in various communities and it has drastically increased around the world [1]. Smoking has been identified as a one of the major causes of mortality and morbidity including respiratory and cardiovascular illness in developing and developed countries. It has also been estimated that smoking will kill about 10 millions by the year 2025 if the current trend toward cigarette consumption persists[2]. In Iraq, it has been reported that the ratio of smokers is about 7.5 millions and 30% of them is in the age ranging between 13-15 years[3].

Cigarette smoke is composed of more than 5000 chemicals; including approximately 70 carcinogens amongst them nicotine has received much attention because it triggers the immune system and alters the humoral and cellular immunity [4]. In addition, cigarette smoke is a dominant risk factor for premature and accelerated peripheral, coronary and cerebral atherosclerotic vascular diseases[5,6]. A one to three fold increase in risk of myocardial infarction has generally been noted among cigarette smokers[7]. The mechanism by which smoking cause myocardial infarction remains vague, but cigarette smoking has been found to alter the levels of lipoproteins[8, 9].

Serum lipoprotein abnormalities are said to be the underlying major risk factors and may even be essential for the common occurrence of atherosclerotic vascular diseases [10]. Clinical, genetic and epidemiological evidence indicates that elevated levels of low density lipoprotein are important risk factors for the disorder [11]. The present work was undertaken to investigate further the effect of smoking on lipid profile by employing the enzymatic methods to determine the alterations in triglycerides, total cholesterol, HDL, LDL and VLDL cholesterol in a group of smokers and to compare the resulting data to those obtained from non smokers.

Materials and methods

Forty male smokers between the ages of 25 to 35 years were included in this study. We included only males because in our society males smoke more frequently and openly compared with females. For the comparative assessment, thirty healthy individuals (non smokers) who were comparable in ages and sex were also enrolled. Smokers were included if they had a history of smoking 10 or more cigarettes per day and all have been smokers for at least 8 years. No volunteer had a history of diabetes mellitus or any other systematic disease predisposing them to endothelial dysfunction. Individuals receiving lipid lowering agents, those having renal, hepatic or endocrine disorders and persons who were taking non cardiac drugs that might affect the lipid profile were excluded from the study.

Venous blood samples were drawn from all volunteers after 10 hour overnight fast. Samples were centrifuged; serum was collected and stored at 20°C until analyzed.

Total cholesterol, triglycerides and HDL concentrations in sera of smokers and non smokers were estimated using the enzymatic methods by diagnostic kits obtained from BIOMERIEUX SA, France. VLDL and LDL levels were evaluated according to the method described by Friedewald et al[12].

VLDL=TG/5 , LDL=Total cholesterol-HDL-VLDL

Non HDL-cholesterol is defined as the difference between total cholesterol and HDL-cholesterol; consequently the resulting non HDL-cholesterol includes all the cholesterol present in lipoprotein particles considered to be atherogenic. Risk ratios were calculated as total cholesterol / HDL-cholesterol, LDL-cholesterol / HDL-cholesterol, Non-HDL-cholesterol / HDL-cholesterol.

Statistical Analysis

The data were analyzed by using SPSS version 10 software. All variables are shown as the mean ± standard deviation. The data between controls and smokers were compared by performing student's *t*-test. The "p" value less than 0.05 was considered to be statistically significant.

Results

This study was achieved to assess the potential relationship between cigarette smoking and the alterations in serum lipid profile.

The levels of total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein and very low-density lipoprotein were measured in serum samples from 40 smokers and 30 non smokers (controls) using the enzymatic methods. Both groups were comparable in age and sex.

The mean values ± SD of TC, TG, HDL-C, LDL-C and VLDL-C are summarized in table (1). The data obtained in this study indicate statistically significant elevation ($p< 0.01$) in the mean concentration of TC. Highly significant increase in the mean levels of TG and VLDL-C was noticed to be present in smokers group ($p<0.001$) when compared with controls. LDL-C mean level was higher in smokers (172.35±42.64 mg/dL) than in non smokers (164.60±11.50 mg/dL) but the increase was statistically insignificant ($p>0.05$).

Table(1): The levels of total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein and very low-density lipoprotein in smokers and non smokers.

Lipid profile(mg/dL)	Smokers n=40	Non smokers n=30	p value
TC	190.32±33.62	172.0±12.06	< 0.01*
TG	179.55±61.52	94.30±11.85	< 0.001**
HDL-C	29.76±3.31	33.80±4.44	< 0.001**
LDL-C	172.35±42.64	164.60±11.50	> 0.05
VLDL-C	34.41±13.84	19.20±2.20	< 0.001**
Mean age	28±5	27±6	-

*Statistically significant. **Highly significant

As can be seen in table (1), our results revealed a highly significant decrease ($p<0.001$) in HDL-C mean concentration in smokers as compared to control group.

The values of various risk ratios are demonstrated in table (2). The results showed that the ratios of TC/HDL-C, LDL-C/HDL-C and non-HDL-C/HDL-C were significantly higher in smokers than in non smokers ($p<0.01$).

Table(2): The atherogenic index as indicated by three risk ratios; TC/HDL-C, LDL-C/HDL-C and non HDL-C/HDL-C in smokers and non smokers.

Group	TC/HDL-C	LDL-C/HDL-C	Non HDL-C/HDL-C
Smokers	6.39±10.15	5.79±12.88	5.39±9.15
Non Smokers	5.08±2.71	4.86±2.59	4.08±1.71

Discussion

Cigarette smoking is the second cause of death in the world [13]. It is also responsible for 1 of 10 adult deaths or more than 4.9 million deaths each year[14].A number of adverse effects has been reported to be associated with smoking, affecting several physiological systems including cardiovascular, immunological systems and others[15].

The results show that smoking is significantly and positively associated with serum total cholesterol, triglycerides and VLDL-C. In other words, total cholesterol, triglyceride and VLDL-C levels are increased by smoking thereby revealing direct dose response relationship[16]. The mean serum total cholesterol in non smokers was 172±12.06 mg/dL while it was significantly higher in smokers i.e. 190.32±33.62 mg/dL ($p< 0.01$). These observations are in tune with the findings of other workers [17].

The mean serum triglycerides level showed a highly significant difference ($p<0.001$) between smokers and non smokers, the mean

values were 179.55±61.52 mg/dL and 94.30±11.85 mg/dL respectively. These findings are similar to those observed by Wyndr et al[18] and Rustogi et al[19].

The present investigation showed that VLDL-C mean level was 34.41±13.84 mg/dL in smokers group and it is significantly higher ($p<0.001$) than in non smokers, 19.20±2.20mg/dL.While only insignificant difference in the level of LDL-C was observed between smokers and non smokers. These findings are in accordance with those obtained by NS Neki[20] who also reported that the significance of LDL-C levels strongly depends on the number of cigarette smoked per day. Contrary report to this has also been demonstrated by Sirisali *et al* who found that total cholesterol and LDL-C did not vary between smokers and non smokers[21]. This difference in observations can be attributed to ethnic differences in the population studied. It has been reported that the stimulation of the sympathetic adrenal system caused by nicotine may lead to an increase in catecholamine secretion resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic triglycerides and VLDL-C in the blood stream[17]. The increased levels of LDL-C, VLDL-C and TG can also be attributed to the decreased activity of lipoprotein lipase as a result for the presence hyperinsulinaemia in smokers[22].

The most prominent feature in our assessment lies in the level of the anti atherogenic HDL-C, which showed a highly significant decrease ($p<0.001$) in smokers as compared to non smokers, 29.76±3.31 mg/dL *versus* 33.80±4.44 mg/dL respectively. In agreement with our finding, a fall in HDL-C level by 3-5 mg/dL in smokers has previously been reported by Rosenson[23]. The lowered HDL-C level can presumably be interpreted as a consequent for the decreased oestrogen level caused by smoking[24].

Our results suggest that cigarette smoking adversely alters the lipid profile resulting in dyslipidaemia in smokers. Moreover, some studies have shown that the changes get more marked as the number of cigarette smoked increases[25].Smoking causes an increase in oxidized LDL-C level which plays a key role in atherosclerotic process[26]. It has been suggested that the oxidation of LDL-C generates potent proatherogenic mediators[27, 28]. Lipoprotein oxidation is presumed to occur in the artery and the specific cell type(s) or mechanism(s), that may generates superoxide radicals, hydrogen peroxide or lipid peroxides outside the cell, may contribute to the oxidation of LDL-C[29]. Non HDL-C was found to be a better tool for screening and assessing the risk of atherosclerosis. In the present study the level of non HDL-C was significantly elevated in smokers when compared to controls.

The calculated risk ratios, table(2), that indicate the risk for the development of atherosclerosis were found to be significantly higher in smokers when compared with non smokers. The combination of these findings along with atherogenic ratios suggests that smokers are at high risk

for the development of coronary heart disease.

The present investigation implies both clinical as well as public health implications. In conclusion, measurements of lipid profile in smokers are good predictive tools in the assessment of cardiovascular diseases. A high level of LDL-C, VLDL-C and TG is strongly associated with development of coronary artery disease while a low level of HDL-C remains a significant independent predictor of cardiovascular disease.

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