Effects of smoking on serum lipid profile in Iraqi subjects

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ABSTRACT

Objectives: (a) To compare serum lipid profile in smokers with nonsmokers. (b) To determine the magnitude of dyslipidaemia in the smoker subjects.

Methods: This descriptive study was conducted in Al-Zahrawi Private Hospital in Mosul, from January to December 2004. Fasting blood samples were collected from 179 apparently healthy smokers who attended the Outpatient Department and 205 apparently healthy nonsmokers matched age and sex. Serum lipid profile was compared between smokers and nonsmokers. In smokers, Comparison was also done according to the duration of smoking and the number of cigarettes smoked per day. The collected data were analyzed by Chi-square, Z, ANOVA and Duncan tests.

Results: Body mass indexes (BMIs) were significantly lower in smokers than in nonsmokers ($P<0.001$). Serum triglycerides (TG), total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C) were significantly higher in smokers than in nonsmokers ($P<0.001$) while high density lipoprotein (HDL-C) was significantly lower ($P<0.001$) in smokers than in nonsmokers. Serum TC and LDL-C were significantly higher in smokers of long duration. Heavy smokers had low HDL-C and high TG, TC, LDL-C and VLDL-C compared with light smokers. The prevalence of hypercholesterolaemia, hypertriglyceridaemia, hyperLDL-cholesterolaemia and low HDL-cholesterolaemia among the studied subjects according to the recommendation of British Hyperlipidaemia Association (1998) were 25.6%, 24.5%, 26.2 % and 37.4% respectively.

Conclusion: Smoking is associated with a change in serum lipid profile. The number of cigarettes smoked per day and the duration of smoking play an important role in lipid profile change. The results document a high prevalence of dyslipidaemia among Iraqi smokers.

الخلاصـــة

اهداف الدراسة: مقارنة واجهة شحوم مصل الدم بين المدخنين و غير المدخنين ودراسة نمط اضطراب شحمانية الدم لدى المدخنين.

طرق العمل: دراسة وصفية أنجزت في مستشفى الزهراوى الأهلي في الموصل أثناء الفترة من كانون الثاني ولغاية كانون الأول عام ٢٠٠٤ وتم جمع نماذج الدم في حالة الصوم لكافة القياسات من ١٧٩شخصا من المدخنين الأصحاء و٢٠٥ أشـخاص من الأصحاء غير المدخنين مع مطابقة العمر والجنس. وتمت مقارنة واجهة الشحوم بين المدخنين وغير المدخنين. وتمت المقارنة كذلك حسب فترة التدخين وعدد السكائر المدخنة فى اليوم الواحد. وتم تحليل البيانات بأستعمال مربع كاى، اختبار Z ، انوفا واختبار دنكن..

النتائج: كانت مؤشرات كتلة الجسم لدى المدخنين أقل مما هي لدى غير المدخنين (پ<٠٠١ ر٠). ولوحظ أن هنالك ارتفاعا معتدا إحصائيا في مستويات الكولسترول الكلي والشحوم الثلاثية والبروتين ألشحمي خفيض الكثافة والبروتين ألشحمي وضيع الكثافة لدى المدخنين (پ<٠٠١ ر٠) مع انخفاض معتد إحصائيا في البروتين ألشحمي رفيع الكثافة (پ<٠٠١ ر٠) لدى المدخنين. كما لوحظ عدم وجود فروق ملحوظة بين المدخنين الذكور والإناث. كذلك لوحظ وجود ارتفاع معتد إحصائيا في مستوى الكولسترول الكلى والبروتين ألشحمي خفيض الكثافة لدى المدخنين لفترة طويلة من السنين. أما الأشخاص الذين يدخنون عددا كبيرا من السكائر يوميا فلقد كانت مستويات الكولسترول الكلي والشحوم الثلاثية والبروتين الشحمي خفيض الكثافة والبروتين ألشحمي وضيع الكثافة أعلى مما هي لدى الأشخاص الذين يدخنون عددا قليلا من السكائر يوميا. كذلك كان مستوى البروتين ألشحمي رفيع الكثافة اقل لدى الأشخاص الذين يدخنون عددا كبيرا من السكائر يوميا مما هو عليه لدى الأشخاص الذين يدخنون عددا قليلا من السكائر يوميا. ولقد

صنف ارتفاع شحوم مصل الدم حسب توصيات الجمعية البريطانية لفرط شحوم الدم لعام ١٩٩٨ حيث لوحظ ارتفاع مستويات الكولسترول الكلي و الشحوم الثلاثية والبروتين الشحمي خفيض الكثافة بالنسب 25.6%، ٢٤,٥% و26.5% على التوالى مع نقصان في مستوى البروتين ألشحمي رفيع الكثافة بنسبة ٣٧,٤%.
الاستنتاج: إن تدخين السكائر يصحبه تغيير في واجهة شحوم مصل الدم، كما أن عدد السكائر المدخنة في اليوم الواحد والمدة الزمنية للتدخين يلعبان دورا في تغيير واجهة شحوم مصل الدم. وهذه النتائج تثبت النسبة المرتفعة لاضطراب شحمانية الدم لدى مدخني السكائر العراقيين.

Cigarettesmoking is a well recognized risk factor for coronary heart disease (CHD)[1] and hypertension.[2] Furthermore, there is a dose-response relationship between the number of cigarettes smoked per day and cardiovascular morbidity and mortality.[3] Certain components of cigarette smoke, such as nicotine and carbon monoxide, have been reported to be responsible for the development of CHD by increasing plasma catecholamines level and producing hypoxia.[4]

Cigarette smoking increases serum level of total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C) while the level of serum high density lipoprotein cholesterol (HDL-C) is decreased.[5-7] Multiple endocrine and neuro-humeral effects induced by smoking including elevations of catecholamines, growth hormone, cortisol and insulin levels might cause changes in lipolytic enzymes and lipoproteins metabolism in the liver[8] However, the detailed mechanisms of most of these interactions are unknown.[9]

The present study has been undertaken to evaluate the effect of smoking on serum lipid components of Iraqi subjects and also to explore the relationship between the duration of smoking and serum lipids.

Subjects and methods

This descriptive study was conducted during a period from January through December 2004 in the Outpatient Department of Al-Zahrawi Private hospital in Mosul. One hundred seventy nine apparently healthy smokers (95 males and 84 females), their ages ranged between 38-72 years (mean ± SD: 49.2 ± 6.4 years), and 205 apparently healthy nonsmokers as a control group (87 males and 118 females) their ages ranged between 38-72 years (mean ± SD: 47.6 ± 6.7 years) were enrolled in the study. The enrolled nonsmoker subjects had never smoked in the past. Subjects with hepatic, renal or cardiovascular diseases and diabetes mellitus were excluded from the two groups, but body weight value was not a preclusion criterion. None of the subjects were taking any drug that might influence serum lipid levels.

The smokers were divided into group 1, group 2 and group 3 according to the number of cigarettes smoked per day; 1-10, 11-20, and >20, respectively. Furthermore, the smokers were divided into group I, group II and group III according to the duration of smoking; 5 years, 6-10 years and >10 years, respectively.

Limited anthropometric physical examination has been done to each subject. This involved the measurement of height, weight. Body mass index (BMI) was calculated as the ratio of weight in Kg divided by the square of hight in meter.[10]

Blood samples (10 ml for each) were taken after an overnight fasting from the subjects, and transferred into plain tubes for measurement of different serum lipoproteins. Determination of TC, HDL-C and TG were performed using enzymatic methods[11]. LDL-C was calculated by using Friedewald formula: "LDL-C (mmol/L) = TC − [HDL-C + (TG ×0.455)]", for those who are having TG level of <4.5 mmol/L.[12]

Classification of dyslipidaemia was based on the recommendation of British Hyperlipidaemia Association (1998) using lipoprotein thresholds of: ≥ 2.4 mmol/L for TG, ≥ 5.0 mmol/L for TC, 3.0 mmol/L for LDL-C, ≤1.15 mmol/L for HDL-C, ≥5.0 for TC/HDL-C ratio, ≥2.5 for LDL-C/HDL-C ratio, and ≥3.0 for TG/HDL-C ratio as a cut-off limits for dyslipidemia.[13] A subject was considered to be dyslipidaemic when one or more of his serum lipoprotein was beyond the above mentioned levels.

Data were represented as mean ± SD. Analysis of variance (ANOVA), Duncan test, Z test and Chi-square test were used for comparison of the biochemical measurements. Pvalue of <0.05 was considered to be significant.

Table 1: *Serum lipid profile in smokers and nonsmokers represented as mean ± SD.*

Parameters (mmol/L)	Smokers (n=179)	Non-smokers (n=205)	P value
Total cholesterol	5.38 ± 0.77	4.49 ± 0.76	P< 0.001
Triglycerides	1.78 ± 0.91	1.45 ± 0.75	P< 0.001
HDL-C	1.10 ± 0.26	1.20 ± 0.24	P< 0.001
LDL-C	3.46 ± 0.82	2.60 ± 0.73	P< 0.001
VLDL-C	0.82 ± 0.34	0.69 ± 0.15	P< 0.001

Table 2: *Lipid profile in relation to the number of cigarettes smoked per day represented as mean ± SD.*

Parameters (mmol/L)	Number of cigarettes smoked per day		
	Group 1 1-10 cigarettes/ day (n=105)	Group 2 11-20 cigarettes/ day (n=59)	Group 3: >20 cigarettes / day (n=15)
Total cholesterol	5.21 ± 0.68 a	5.69 ± 0.68 a	6.23 ± 1.96 b
Triglycerides	1.74 ± 0.95 a	1.77 ± 0.86 a	1.86 ± 0.84 b
HDL-C	1.19 ± 0.24 a	1.07 ± 0.27 b	0.96 ± 0.22 b
LDL-C	3.26 ± 0.7 a	3.84 ± 0.82 ab	4.35 ± 1.5 b
VLDL-C	0.76 ± 0.43 a	0.78 ± 0.39 a	0.92 ± 0.38 a

Different letters horizontally mean significant difference at P< 0.05 level according to Duncan test (ab: not significant with either a or b).

Results

The BMI in smokers group (23.66 ± 2.64 kg/m^2) was significantly lower (P<0.001) than in nonsmoker group (24.71 ± 2.78 kg/m^2). Serum TG, TC, LDL-C, and VLDL-C were significantly higher (P <0.001) in smokers than in nonsmokers, while HDL-C level was lower (P<0.001) as shown in Table 1.

Furthermore, no significant difference in the values of lipid components was seen between smoker male and female subjects (data not shown).

Heavy smokers (greater than 20 cigarettes/ day) had significantly high LDL-C and TC levels (P< 0.05) and significantly lower HDL-C levels (P < 0.05) than the light smokers (less than 10 cigarettes/ day) (Table 2).

Serum TC, TG and LDL-C were significantly higher in smokers of long duration of smoking (> 10 years) than in smokers of short duration (less than 5 years). However, there were no significant differences in the levels of other lipoproteins in regard to the duration of smoking, as shown in Table 3.

Considering the criteria of British Hyperlipidaemia Association (1998) to diagnose dyslipidaemia, the prevalence of hypercholesterolemia, hypertriglyceridemia, hyperLDL-cholesterolemia and lower HDL-cholesterolemia were 25.6%, 24.5%, 26.2% and 37.4% respectively (Table 4).

Table 3: *Serum lipid profile in smokers and nonsmokers according to the duration of smoking represented as mean ± SD.*

Parameters (mmol/L)	Duration of smoking in years		
	Group I: < 5 years (n=43)	Group II: 6-10 years (n=108)	Group III: >10 years (n=28)
Total cholesterol	5.09 ± 0.70 a	5.43 ± 0.73 b	5.7 ± 0.87 b
Triglycerides	1.57 ± 0.69 a	1.79 ± 0.99 ab	1.98 ± 0.88 b
HDL-C	1.13 ± 0.20 a	1.07 ± 0.27 a	1.00 ± 0.33 a
LDL-C	3.12 ± 0.74 a	3.53 ± 0.77 b	3.77 ± 0.96 b
VLDL-C	0.84 ± 0.31 a	0.83 ± 0.45 a	0.93 ± 0.4 a

Different letters horizontally mean significant difference at P<0.05 level according to Duncan test (ab= not significant with either a or b).

Table 4: *Prevalence of dyslipidemia in the smoker subjects according to the British Hyperlipidemia Association. Results are expressed as number (%) of subjects.*

Number of smokers	Hyper-cholesterolemia TC ≥ 5mmol/L	Hyper-triglyceridemia ≥2.4 mmol/L	HDL-C ≥1.15 mmol/L	LDL-C ≥ 3 mmol/ L	TC:HDL ratio ≥5. 0	LDL-C: HDL-C ratio ≥2. 5	TG: HDL ratio ≥3. 0
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Males (95)	23 (12.8)	23 (12.8)	35(19.5)	22(12.3)	52(29)	18 (10)	11 (6.0)
Females (84)	23 (12.8)	21 (11.7)	32(17.9)	25(13.9)	44(24.6)	15 (8.4)	6 (3.4)
Total (179)	46 (25.6)	44 (24.5)	67(37.4)	47(26.2)	96(53.6)	33 (18.4)	17(9.5)
P value	NS	NS	NS	NS	NS	NS	NS

Discussion

When considering the deleterious effect of smoking, it is important to consider the impact of low BMI on smokers. Smokers, in this study, had lower BMIs than nonsmokers. This is conforming other studies.[14-16] However, this benefit does not seem to be dominant as it is overwhelmed by many unfavorable effects of smoking that jeopardize the coronary and the peripheral vessels including dyslipidaemia, platelet abnormalities; particularly higher concentration of glycoprotein IIb/IIIa[17,18], endothelial dysfunction, and thrombosis abnormalities.[19] Dyslipidaemia seems to be very prominent among these risks as high levels of LDL-C, VLDL-C and TG are strongly associated with the development of CHD, while a low level of HDL-C remains a significant independent predictor of CHD.[20]

In this study, serum levels of TC, TG, LDL-C, and VLDL-C were significantly higher in smokers than in nonsmokers. This is consistent with the results that have been found by other workers.[13-16] Furthermore, serum HDL-C level was lower in smokers which is consistent with what other investigators have found.[19,21] However, this finding of lower level of HDL-C among smokers was inconsistent with Seikmeier et al.[22], and Dirican et al.[23] who reported no difference of HDL-C concentrations between smokers and non-smokers. It has been proved that smoking stimulates oxidation of LDL-C particles resulting in a significant increase in TG and fall in HDL-C,[16, 24]

The levels of serum TG, TC, LDL-C were significantly higher in heavy smokers (>20 cigarettes/ day) than in the light smokers (<10 cigarettes/ day), while HDL-C level was significantly lower. This is consistent with comparable studies.[3,4,25,26] The change in HDL-C level associated with starting or quitting smoking has been estimated to be equal to 0.15 mmol/L.[27] Regarding the duration of smoking, in this study, serum levels of TC,TG and LDL-C were significantly higher in the longer duration groups, whereas HDL-C and VLDL-C levels showed no change. This is consistent with the finding of Mjos.[4]

The present study suggests that smoking adversely and progressively alters serum lipid profile producing dyslipidaemic state, a state that together with other untoward factors represent a significant risk factor of CHD. Moreover, the dose dependent dyslipidaemic response among smokers rationalizes the relationship between the number of cigarettes smoked per day and cardiovascular morbidity and mortality.[9] However, it is not clear whether smoking affects lipid components of the serum as a result of direct physiological changes of lipid metabolism or of a dietary changes induced by smoking.[3,28] In this study, the prevalence of dyslipidemia was in agreement with another study done by Cade et al.[28]

In ***conclusion***, smoking produces changes in serum lipid profile. The number of smoked cigarettes per day and duration of smoking also play a role in change of lipid profile. High prevalence of dyslipidemia among Iraqi smokers predisposes them to CHD.

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