Serum Lipids and urate in Cigarette smokers

Altayeb Elazomi[1]., Sara E. Ibrahimm[2], Elrasheed I. Mohamed[1] , Salah B. Bahroun[4], H. I. Fahelbum[2] and Omar F. Idres3

1 Faculty of Medical Technology, University of Zawia, Libya.

2Sharq Elniel College, School of Medical Laboratory Science.

3Faculty of Science and Technology, University of Alneelain,Khartoum, Sudan.

4National Medical Research Center, Zawia, Libya.

Abstract

Objective :The present study was undertaken to evaluate lipid profile and urate in apparently healthy cigarette smokers and compare it with apparently healthy non- smokers in the fasting state. Also to demonstrate the possible effect of smoking on serum lipid profile and urate concentration

Methods: The study was conducted on 50 healthy cigarettes smokers and compared with 50 healthy age and weight matched non-obese non-smokers who served as controls. Subjects in both groups were in the age range of 30-60 yrs., having no history of alcohol abuse, or diseases like diabetes mellitus, hypertension, hepatic impairment, renal disease, gout, hypouricemia, and obesity, and were neither on drugs like β-blockers, lipid lowering drugs, or thiazide diuretics. Clinical data were obtained from the history and record on questionnaire sheet. The clinical assessment was done by a medical doctor.

Results: It was revealed that mean S.TC (275 ± 25 mg/dl), LDL-C (179 ± 44 mg/dl), and TG (116 ± 29 mg/dl), were significantly higher in smokers ($p < 0.001$) as compared to non-smokers, i.e., mean S.TC (139 ± 25 mg/dl), LDL-C (89 ± 25), TG (93 ± 25).

mg/dl ($p < 0.001$) as compared to non-smokers. Mean serum HDL-C (27 ± 6 mg/dl) was significantly lower in chronic smokers ($p < 0.001$) as compared to non-smokers, i.e., 40.0± 8 mg/dl. Mean serum Urate (5.2 ± 1.0 mg/dl) was significantly lower in chronic smokers ($p < 0.001$) as compared to non-smokers, i.e., 6.1. ± 1.1 mg/dl.

Conclusion: Smoking produces adverse effects on lipid profile and, therefore increasing the cardiovascular disease risk. On the other hand the significant low level of serum urate in smokers attributed in significant oxidative stress and hence cardiovascular disease. Cigarette smoke may be benefit in reducing hyperuricemia risk.

Key words

S.TC : Serum Total cholesterol; LDL-C : Low density lipoprotein cholesterol; HDL-C : High density lipoprotein cholesterol; TG : Triglycerides; urate.

Introduction:

Worldwide, the effect of smoking are estimated to kill 3 million people per year. This contracts with 0.2 million in 1950 and projections for 2025 of 10 million. [1] Epidemiological evidence suggest that cigarette smoking is the a major risk factor for chronic obstructive pulmonary disease such as chronic bronchitis and emphysema, carcinogenesis and cardiovascular disease [1,2,3]. Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL-C in the blood stream [4,5]; (b) Fall in estrogen levels occurs due to smoking which further leads to decreased HDL – cholesterol [35]; (c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL-C, VLDL-C, and TG due to decreased activity of lipoprotein lipase [6,7]; d) Consumption of a diet rich in fat and cholesterol as well as a diet low in fibre and cereal content by smokers [4,8.] Also recent evidence suggest that oxidants present in the gas phase of cigarette smoke are involved [8]. There is increasing experimental evidence that oxidation of low density lipoprotein cholesterol (LDL-C) plays a major role in the pathogenesis of coronary artery disease (CAD). However, results from clinical studies on LDL oxidation and CAD are not consistent [10]. Cigarette smokers have increased inflammatory responses that further enhance their oxidative stress.[11,12] Since in humans, uric acid is the most abundant aqueous antioxidant, accounting for up to 60% of serum free radical scavenging capacity[13] and is an important intracellular free radical scavenger during metabolic stress including smoking,[14,15] therefore, measurement of its serum level reflects the antioxidant capacity.[13]

Material and Methods

50 cigarette smokers and 50 normal healthy non smokers (control) were selected from the teaching staff of Sudan University for Science and Technology. None of them had past or present history of renal, gout, cardiac, hepatic, diabetes mellitus, hypertension endocrine disorders, obesity and on drugs like β-blockers, lipid lowering drugs, and thiazide diuretics.

The smokers were age matched to controls (30 – 60) and they were regular cigarette smokers for the last 8 years, consuming more 10 cigarette per day. Clinical data were obtained from the history and record on questionnaire sheet. Clinical assessment was done by a medical doctor. After overnight fasting, they were subjected to laboratory investigations including, lipid profile (serum cholesterol, triglycerides, HDL-C, LDL-C), and serum urate.

Results

Table (1) and fig. (1) show, significant difference between the means of serum total cholesterol in the control group (n =50) and the study group (smokers). (n =50). (139 ± 25 versus 275±51 mg/dl, p <0.001).Fig (1) shows comparison of serum total cholesterol levels between control and smokers. Table (1) and fig. (2) show, significant difference between the means of serum LDL in the control group (n =50) and the study group (smokers). (n =50). (89 ± 23 versus 179±44 mg/dl, p <0.001).Fig (2) shows comparison of serum LDL levels between control and smokers.

Table (1) and fig. (3) show, significant difference between the means of serum triglyceride in the control group (n =50) and the study group (smokers). (n =50). (93 ± 25 versus 116 ± 29 mg/dl, p <0.001).Fig (3) shows comparison of serum triglyceride levels between control and smokers.

Table (2) and fig. (4) show, significant difference between the means of serum HDL in the control group (n =50) and the study group (smokers). (n =50). (40 ± 6 versus 27 ± 8 mg/dl, p <0.001).Fig (4) shows comparison of serum HDL levels between control and smokers.

Table (2) and fig. (5) show, significant difference between the means of serum urate in the control group (n =50) and the study group (smokers). (n =50). (6.1 ± 1.1 versus 5.2 ± 0.98g/dl, p <0.001).Fig (4) shows comparison of serum urate levels in control and smokers.

Variable	**Control group n = 50**	**Study group n= 50**	***P***
Serum cholesterol (mg/dl)	139 ± 25 (77 – 189)	275 ± 51(177 – 415)	<0.001
Serum LDL	89 ± 23 (43 – 144)	179 ± 44(82 – 273)	<0.001
Serum triglyceride	93 ± 25 (54 – 141)	116 ± 29 (58 – 186)	<0.001

Table (1) :Comparison of the means of serum total cholesterol, LDL, and triglyceride in the control group and the study group (smokers).

Variable	Control group n = 50	Study group n= 50	*P*
Serum HDL (mg/dl)	40 ± 6 (24 – 56)	27 ± 8 (11- 51)	<0.001
Serum urate (mg/dl)	6.1± 1 (4.4 – 10.6)	5.2± 0.98 (3.4 – 7.1)	<0.001

Table (2): Comparison of the means of serum HDL, and urate in the control group and the study group (smokers).

- Table(1), and (2) show the mean ± S.D, range between brackets ( ) and probability (p)
-t -test was used for comparison.

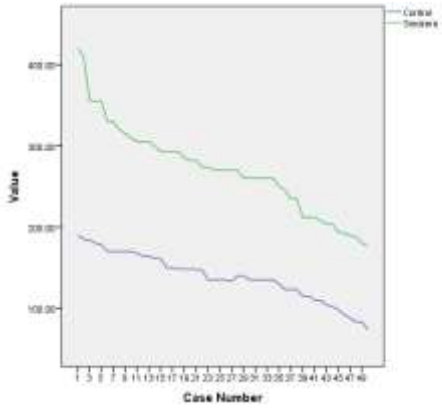

Figure (1): Comparison of levels of S. cholesterol (mg/dl) in control group and study group (smokers).

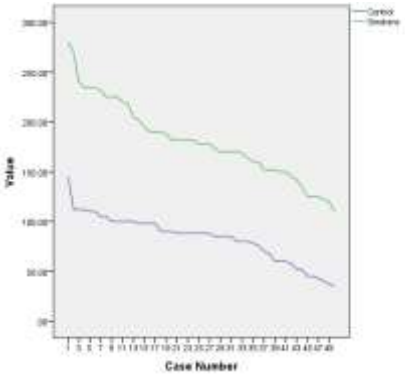

Figure (2): Comparison of levels of LDL- cholesterol (mg/dl) in control group and study group (smokers).

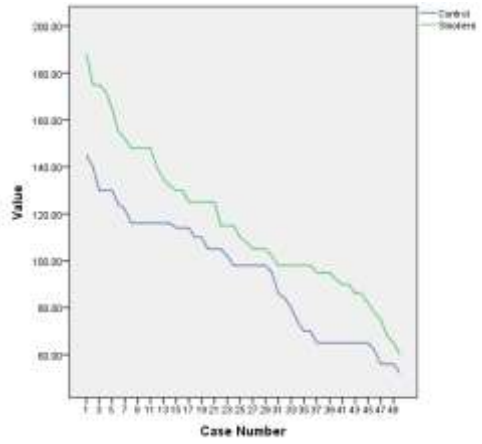

Figure (3): Comparison of levels of S. triglyceride (mg/dl) in control group and study group (smokers).

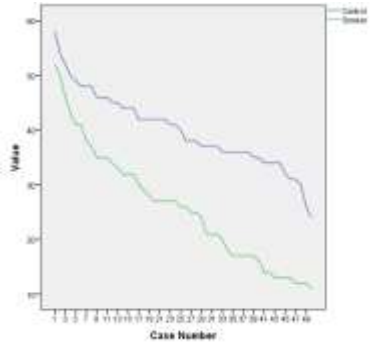

Figure (4): Comparison of levels of S. HDL- cholesterol (mg/dl) in control group and study group (smokers).

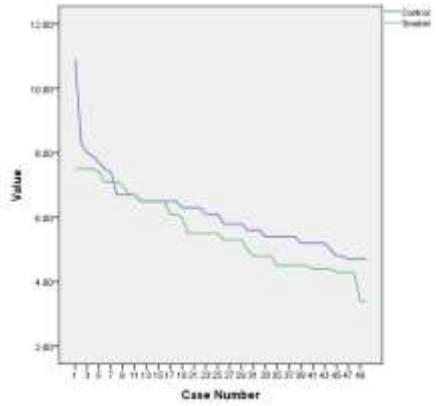

Figure (5): Comparison of levels of S. urate (mg/dl) in control group and study group (smokers).

Discussion

To our knowledge there appears to be no relevant published data on the effect of smoking on blood chemistry among Sudanese, so comparison will be made with studies made elsewhere. Prevalence of smoking varies considerably in different

countries and we think it is increasing in Sudan. And this resembles the situation in other parts of Africa.[16,17] Our study was done exclusively among the male teaching staff members of Sudan University for Science and Technology. In Sudan society female are reluctant to discuss the issue of smoking in an open manner, as smoking in young females is considered offending practice although it is accepted in old ones. It is revealed that triglycerides, LDL-C, TC were significantly higher in smokers as compared to non-smokers thereby revealing a direct dose response relationship [8,18],. The mean serum total cholesterol in non-smokers (control) was 139.1 ± 25 mg/dl while it was significantly higher in smokers, i.e., 275 ± 15 mg/dl. These observations are in tune with the findings of other workers [4,19,20] ,. Cigarette smoking substantially increases the risk of coronary heart disease and ischemic stroke [22,23]. Serum LDL – cholesterol showed 101% increase, when the control group was compared to the study group (89 ± 23 versus 179± 44 mg/dl, $p<0.001$). Serum LDL – cholesterol was highly significantly raised in the study group. This is in agreement with previous result published by many authors. [2,9,16,24]. The mean HDL-Cholesterol in non-smokers was 40. ± 6 and 27. ± 8 in smokers respectively (p value <0.001). This finding is similar to that of Rosenson [22] who reported that there is fall in HDL-C level by 3-5 mg/dl in smokers. The mean serum triglycerides levels in non-smokers and smokers were 93 ± 25 mg/dl and 116 ± 29 mg/dl, $p<0.001$ respectively, serum triglyceride was highly significantly raised in the study group (smokers). However, triglyceride levels in study group (smokers) were found to be within the normal reference range. [25,26] These findings suggest that smoking alters the lipid profile adversely causing dyslipidaemia in smokers and the changes become more marked with the number of cigarettes smoked [27] Smoking causes an increase in oxidized LDL-cholesterol level which plays the key role for atherosclerotic process[19]. A high level of LDL-C, VLDL-C and triglyceride are strongly associated with development of coronary artery disease while a low level of HDL-C remains a significant independent predictor of coronary artery disease [29,30]. Serum urate on average, showed 14.8% reduction when the control group was compared to the study group (6.1± 1.1 versus 5.2 ± 1.0 mg/dl, $p<0.001$), serum urate was highly reduced in the study group. This finding is in agreement with other studies that showed low serum uric acid in regular smokers [30,32].[34] and reduction of antioxidants including uric acid in smokers [33,34] indicating that oxidative stress increases every time a cigarette is smoked.[31]

Conclusion

Is obvious from these results, cigarette smoking has a hyperlipidemic effect, which is reflected as raised serum levels of total cholesterol, LDL – cholesterol and triglyceride with reduced HDL – cholesterol, that increase the risk for atherosclerosis and ischemic heart disease. Serum levels of urate were found to be clearly reduced in cigarette smokers, this may attributed in significant oxidative stress and hence cardiovascular disease. On other hand reduced serum urate may be benefit against hyperuricemia and gout.

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