

Effect of Cigarette Smoking on Lipid Profile In Male in the City of Zawia –Libya

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Abstract

Objective

The present study aimed to compare the lipid profile between smoker and non smoker and to evaluate the durational significance on lipid profile in the smokers and to clarify the influence of daily cigarette smoking on the components of lipid profile in Libyan males.

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 (physician) a medical doctor.

Results:

A significantly increased of serum total Cholesterol, Triglyceride and Low density lipoprotein cholesterol with significant decreased in serum High density lipoprotein cholesterol level in smokers as compared to non smokers and same results were found in smoker group with > 15 cigarettes smoked per day while with increase duration of smoking the TC & LDL-C were increased, TG showed no difference while the HDL-C was decreased showing greater risk of these persons to atherosclerosis and coronary heart disease.

Conclusions:

This study concluded that cigarette smoking causes alteration in lipid profile. Increased duration of smoking and number of smoked cigarettes / day causes more dyslipidaemia. This smoking might be related in the alteration in serum lipid profile levels, and may be the major causes of increases risk for coronary artery and hence cardiovascular disease among cigarettes smokers.

Key words:

Cigarette Smoking, Coronary Heart Disease, Dyslipidaemia, Lipid profile and Tobacco consumption.

INTRODUCTION:

Cigarette smoking is generally considered as associated with increased risk of a variety of medical disorders. Such as chronic obstructive pulmonary diseases (chronic

bronchitis and emphysema), carcinogenesis and for cardiovascular disease^{1,2}. Several studies provide the evidence that cigarette smoking is strongly associated with altering

the normal status of the lipid profile
³.Cigarette smoking increases risk for death from all causes in men and women.⁴
The risk of dying from cigarette smoking has increased over the last 50 years in men and lung cancer.⁴Smoking is estimated to increase the risk for coronary heart disease by 2 to 4 times^{4,5} for stroke by 2 to 4 times,⁴ of men developing lung cancer by 25 times, of women developing lung cancer by 25.7 times⁴. Smoking causes diminished overall health, increased absenteeism from work, and increased health care utilization and cost.⁴There 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) among smokers ⁶. Nicotine and other toxic substances from Tobacco smoke are absorbed through the lungs into the blood stream and are

MATERIALS & METHODS:

The study was conducted from January 2016 to May 2016 at. Az Zawiyah Research Center –Libya. A total of 50 healthily male were enrolled in this study in the age of 30 – 60 Years, 50 were smokers [44.2±20Years] and 50 were non smokers [42.10±13.39Years].

The local ethics committee approved the study. Before participation, volunteers were

women in the United States.⁴Smokers are more likely than nonsmokers to develop heart disease, stroke,

circulated throughout the body. These substances damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a non smoker ⁷. Nicotine increases the amount of bad fats (total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), and triglycerides (TG)) circulating in the blood vessels and decreases the amount of good fat (high-density lipoprotein cholesterol (HDL-C) availability ⁷.

Nicotine induces oxidative stress, generates free radicals that attack on the membrane lipids resulting in the formation of malondialdehyde (MDA), which causes per oxidative, tissue damage

fully informed of the nature and purpose of the study and written consent was obtained from each.

The smoker group was sub classified according to smoking number of cigarette /day into two group smoking less than 20 cigarettes/ day and smoking more than 20 cigarettes/ day, and according to the duration of smoking sub classified to smoking for less

than 20 years and smoking for more than 20 years. Blood samples were obtained following an overnight fasting. Samples were withdrawn from a cubital vein into blood tubes (plain containers). The serum was then separated from the cells by centrifugation at 3000 r/min for 10 min and

STATISTICAL ANALYSIS:

Data were expressed as mean \pm standard deviation (SD). The means were compared using independent sample t.test. Analysis

RESULTS:

Baseline characteristics of the 100 participants (male), 50% of them were non smoker (n = 50) aged 42.10 ± 13.39 years and 50% were cigarettes smoker (n = 50) and they were 44.20 ± 20 years of age. The mean \pm SD values for serum cholesterol, triglycerides, LDL-C and HDL-C are given in Table 1. All the components of lipid profile studied (Cholesterol, triglycerides and LDL-C) were found significantly increased for smokers compared to the healthy control non-smoking subjects, while the HDL-C were decreased in smoker group compared to the non smoker group. The values of significance for various comparisons are given in Table 1. Table 2 In this set of data, the subjects were categorized according to average number of cigarettes [Less than 15 and above 15

immediately stored on ice at 4°C. Serum Cholesterol, Triglyceride, HDL-C and LDL-C was measured by using the enzymatic method using Cobas integra 400 plus - Roche, the reference value are [TC < 200mg/dl, TG < 200mg/dl, HDLC > 55mg/dl and LDL 49 -172mg/dl].

was two-tailed and a p-value ≤ 0.05 was considered as statistically significant.

cigarettes / day] table 2 showed that there was significant increased in the mean levels of cholesterol, Triglyceride while the number of cigarettes smoked per day has no effect on serum LDL-C. HDLC was significantly decreased among smoking group [>15 cigarettes/day versus <15 cigarettes/day]. Table 3 In this set of data, the subjects were categorized according to the duration of smoking [Less than 20 and above 20 Years] table 3 showed that the mean levels of total cholesterol and Triglyceride were increased with increase in the duration of smoking, HDLC shows a decreased with the increase duration of smoking, while the duration of smoking has no effect on serum LDL – C.

Table1: Comparison of lipid profile between smoker and non smoker

Serum Level	Smokers [n =50]	Non smokers [n =50 n]	P -Value
Cholesterol [mg/dl]	199.5	167	0.00
HDL –Cholesterol [mg/dl]	38.68±11.68	47.06±68	0.01
LDL –Cholesterol [mg/dl]	158.56±39.69	99.64±34.78	0.00
Triglyceride [mg/dl]	184.26±50.13	144.74±25	0.04

The table show the mean ± SD and probability (P). T- test was used for comparison.P value ≤ 0.05 was considered significant

Table 2: Influence of daily number of smoking cigarettes on lipid profile

Serum level	Smoking>20 cig/ day [N=14] Mean±	Smoking <20 cig./day [n=36] Mean ±	P -value
Total cholesterol [mg/dl]	218.04±24.68	198±26.39	0.00
LDL –cholesterol	139.02±19.42	149.53±21.51	0.11
HDL –cholesterol	37.50±5.27	40.32 ±5.277.70	0.03
Triglyceride [mg/dl]	185.04±40.53	155.43±54.36	0.00

The table show the mean ± SD and probability (P). T- test was used for comparison.P value ≤ 0.05 was considered significant.

Table3: Influence of duration of smoking on lipid profile

Serum level	smoking > 20 years [N=19] mean ±	smoking<20years [n=31] mean±	P- value
Total cholesterol	202.50±15.46	180.34 ±8.55	0.01
LDL – cholesterol	161.00 ±24.0	154.05 ±15.20	0.08
HDL – cholesterol	41.32 ±4.82	47.50 ±1.40	0.01
Triglyceride [mg/dl]	257.12 ±64.24	146.42 ± 7.78	0.00

The table show the mean ± SD and probability (P). T- test was used for comparison.P value ≤ 0.05 was considered significant.

Discussion:

and reduces the health of smokers in general.^{9,10}Cigarette smoking is the leading

Cigarette smoking harms nearly every organ of the body, causes many diseases,

adult males . In our study Serum TC, TG and LDL were significantly higher in smokers as compared to non-smokers and the serum HDL level was significantly lower in smokers as compared to non-smokers. Our findings are in accordance with the findings of many research workers. The change in the serum cholesterol & lipoprotein levels became more marked with the number of cigarettes smoked per day and duration of smoking in years. This finding has been substantiated by Imamura et al.⁵, N .S Neki⁶ Contrary to the above findings Diricana M et al⁷ did not find significant differences in serum TC, TG, LDL-C, and HDL-C levels between smokers and nonsmokers. Nesje LA. et al ⁸ also found no significant difference between smokers and non- smokers concerning triglycerides and total cholesterol .These differences may due to ethnic's variation in population in previous studies. Dyslipidemia is a well - established risk factor for the development of coronary artery disease. Our study demonstrated presence of Dyslipidemia in chronic Libyans smokers. The main limitation of this study is that important factors which may contribute to the cardiovascular risk factors among males Libyans such as dietary habits, physical activity and genetics were not included.

CONCLUSION:

increased duration / Years and number of cigarette/day smoked. Smoking plays the key role for atherosclerotic process and with coronary artery disease.

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preventable cause of death in the United States. Smoking causes more deaths each year than Human immunodeficiency virus (HIV), Illegal drug use, Alcohol use, Motor vehicle injuries, and Firearm-related incidents combined¹¹. The tobacco smoking is responsible for premature development of CAD Cardiovascular disease and abnormal levels of risk factors such as serum lipid and lipoprotein levels, hypertension, and smoking are related to the earliest stages of atherosclerotic CAD⁷. Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increase 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 in the blood stream¹² (b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL¹³; (c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL and TG due to decreased activity of lipoprotein lipase¹⁴. This study was conducted to assess the impact of smoking on lipid profile in Libyans males . In our study all subjects in both smokers and non smokers group were apparently healthy

These findings suggest that smoking might be related in the alteration in lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with

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