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ORIGINAL ARTICLE

Lipid Profile Levels in Smokers

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ABSTRACT

Objective: The objective of this study was to record mean lipid profiles in smokers

Methodology: All 200 (Two hundred) smokers were enrolled from Cardiology Department, Punjab Institute of cardiology Lahore during 2015 to 2016, the demographic profile of the patients' was taken. Their ECG was done to ensure that they do not have any cardiac disease. After 12 hours over night fasting, blood samples were withdrawn from a cubital vein into blood tubes. The blood samples were sent to hospital laboratory for the analysis of lipid profile. Serum triglyceride (TG) level by GPO – PAP enzymatic colorimetric method, Serum total cholesterol (TC) level by CHOD – PAP enzymatic colorimetric method, Serum HDL-c level by precipitation method, Serum LDL-c level by using Friedewald's formula, LDL-c = TC - (HDL + TG/5), Serum VLDL-c level by using Friedewald's formula VLDL-c = TG/5.

Results: Of 200 cases 45%(n=90) were between 18-40 years of age while 55%(n=110) were between 41-60 years of age, mean+sd was calculated as 41.98+11.02 years, 89.5%(n=179) were male and 10.5%(n=21) were females, mean lipid profiles in smokers was calculated as 188.38+16 55 for total cholesterol, 157.33+15.93 triglycerides, 42.47+4.88 HDL, 114.44+13.43 LDL and 31.47+3.19 for VLDL.

Conclusion: the smokers are at higher risk of developing dyslipidemia. Our results are encouraging for smokers to quit their habits to live a healthy life.

Keywords: Smokers, lipid profile, mean

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death worldwide, with CVD mortality being higher in low- and middle-income nations.[1] In Pakistan prevalence of CVD is estimated up to 6.2%.[2] Dyslipidemia is an abnormal level of lipids in the blood, referred to increase in the levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides, and a decrease in high-density lipoprotein (HDL) cholesterol. Many previous studies reported that dyslipidemia increases the risk of cardiovascular disease.[3] Dyslipidemia is an established risk factor for CVD, is strikingly common in patients with type 2 diabetes, affecting almost 50% of this population.[4] The prevalence of dyslipidemia has increased globally with lifestyle changes over the past decades.[5] Dyslipidemia is a well recognized and modifiable risk factor for cardiovascular diseases which is currently a leading cause of morbidity and mortality worldwide in both developed and under-developed regions.[6,7]

In addition, atherosclerotic cardiovascular disease has been linked to cigarette smoking. Multiple processes, including vascular damage, thrombosis, vascular dysfunction, and lipid peroxidation, account for the impact of smoking on atherosclerosis development. It has been found that aberrant lipid metabolism caused by smoking contributes to atherogenesis. [3,8]

A study reported significant difference of lipid profile in smokers i.e. mean cholesterol [mg/dl] in smokers 191.29±46.3 with p-value ≤ 0.05, the mean triglyceride [mg/dl] in smokers was 147.8±62.4 with p-value < 0.05. Mean HDLC [mg/dl] in smokers was 43.2±7.4, with p-value < 0.05. Moreover LDL [mg/dl] was also statistically significant i.e. mean LDL was 117.6±41.9 in smokers.[9]

One more study demonstrated, there is no difference in lipid profiles among smokers[10] that is a clear controversy with above cited study.[9] They reported that TG (mmol/L) in smokers was 1.21 ± 0.56; p-value > 0.05, mean TC (mmol/L) in smokers was 4.05 ± 0.81 and with p-value > 0.05. Mean HDL (mmol/L) was 1.54 ± 0.62 in smokers with p-value > 0.05. Moreover mean in smokers was LDL (mmol/L) 2.21 ± 0.55, p-value > 0.05.[10]

One more study reported significant difference in lipid profiles in smokers for total cholesterol and LDL (p-value > 0.05)[11], that is another controversy with above study.[9] The mean total cholesterol (mg/dL) was 210.74 ± 39.28 in smokers, p-value 0.192. Moreover mean LDL-cholesterol (mg/dL) in smokers was 132.36 ± 36.75 with p-value = 0.827.[11]

The rational of this study is to find mean lipid levels in smokers. Although a wide range of researches have been done to compare lipid profiles in the population, but still there is a controversy between smoking and lipid profiles.[9-11] So after this study, we will be able to rule out this controversy. If we find abnormal lipid profiles in smokers then in future high risk population (smokers) will be screened for possible prevention from dyslipidemia (abnormal lipid profile) and then from CVD. Moreover smoking is a modifiable risk factor for dyslipidemia and we can encourage smokers to quit their habits to live a healthy life.

METHODOLOGY

The study was done at Cardiology department Punjab Institute of cardiology Lahore during the year 2015 to 2016, a total of 200 Smokers (A person who came as an attendant with patients at PIC smoking presentably. They were labeled as smokers if they smoke ≥ 10 cigarettes/day ≥ 1 year) irrespective of gender status and aged 18-60 years were enrolled in this study, whereas the patients who already had history of CAD or cerebrovascular accident (CVA), taking lipid-lowering drugs, positive family history of dyslipidemia, having diabetes mellitus type II (BSF>126mg/dl on previous medical record, Obese with BMI > 25 kg/m^2, history of chronic liver disease, renal insufficiency (serum creatinine>1.4mg/dl) or on hemodialysis and pregnant females were excluded from the study. Demographic profile (name, age, sex, contact no.) was also taken. Their ECG was done to ensure that they do not have any cardiac disease. After 12 hours over night fasting, blood samples were withdrawn from a cubital vein into blood tubes. The blood samples were sent to hospital laboratory for the analysis of lipid profile. All data was collected by researcher himself on a predefined proforma. Following parameters were estimated: Serum triglyceride (TG) level by GPO – PAP enzymatic colorimetric method, Serum total cholesterol (TC) level by CHOD – PAP enzymatic colorimetric method, serum HDL-c level by precipitation method, serum LDL-c level by using Friedewald's formula: LDL-c = TC - (HDL + TG/5), Serum VLDL-c level by using Friedewald's formula=VLDL-c = TG/5. We used 20th version of SPSS for evaluation of collected data.

RESULTS

Age distribution of the patients shows that 45%(n=90) were between 18-40 years of age while 55%(n=110) were between 41-60 years of age, mean+sd was calculated as 41.98+11.02 years. Gender distribution shows that 89.5%(n=179) were male and 10.5%(n=21) were females.

Mean lipid profiles in smokers was calculated as 188.38+16 55 for total cholesterol, 157.33+15.93 triglycerides, 42.47+4.88 HDL, 114.44+13.43 LDL and 31.47+3.19 for VLDL. (Table No. 1)

Table 1: Lipid Profile of the Cases in Smokers (n=200)

Age(in years)	18-40 years		41-60 years		P value
	Mean	SD	Mean	SD	
Total cholesterol	154.55	11.79	159.6	10.66	0.001
Triglyceride	187.61	10.44	189.00	9.31	0.32
HDL	42.08	3.82	42.78	3.87	0.19
LDL	114.62	10.92	114.30	10.65	0.83
VLDL	30.91	2.36	31.92	2.13	0.001
GENDER	Male		Female		
Total cholesterol	157.38	11.52	156.90	10.87	0.85
Triglyceride	188.42	9.79	187.95	10.40	0.83
HDL	42.51	3.84	42.05	3.96	0.60
LDL	114.43	10.81	114.52	10.24	0.97
VLDL	31.48	2.30	31.38	2.17	0.85
Smoking duration (in years)	1-5		>5		
Total cholesterol	156.62	10.98	158.29	12.00	0.30
Triglyceride	187.90	10.05	189.01	9.54	0.43
HDL	42.08	4.09	42.99	3.45	0.09
LDL	114.50	10.97	114.37	10.44	0.92
VLDL	31.32	2.19	31.66	2.40	0.30

DISCUSSION

In our study, 45%(n=90) were between 18-40 years of age while 55%(n=110) were between 41-60 years of age, mean+sd was calculated as 41.98+11.02 years, 89.5%(n=179) were male and 10.5%(n=21) were females, mean lipid profiles in smokers was calculated as 188.38+16 55 for total cholesterol, 157.33+15.93 triglycerides, 42.47+4.88 HDL, 114.44+13.43 LDL and 31.47+3.19 for VLDL.

The findings of our study are in agreement with a previous study who reported significant difference of lipid profile in smokers i.e. mean cholesterol [mg/dl] in smokers 191.29±46.3 with p-value ≤ 0.05, the mean triglyceride [mg/dl] in smokers was 147.8±62.4 with p-value < 0.05. Mean HDLC [mg/dl] in smokers was 43.2±7.4, with p-value < 0.05. Moreover LDL [mg/dl] was also statistically significant i.e. mean LDL was 117.6±41.9 in smokers.[9] These findings are in agreement with our results.

Another study demonstrated, that there is no difference in lipid profiles among smokers[10] that is a clear controversy with above cited study.[9] They reported that TG (mmol/L) in smokers was 1.21±0.56; p-value > 0.05, mean TC (mmol/L) in smokers was 4.05±0.81 and with p-value > 0.05. Mean HDL (mmol/L) was 1.54±0.62 in smokers with p-value > 0.05. Moreover mean in smokers was LDL (mmol/L) 2.21±0.55, p-value > 0.05.[10]

One more study reported significant difference in lipid profiles in smokers for total cholesterol and LDL (p-value > 0.05)[11], that is another controversy with above study.[9] The mean total cholesterol (mg/dL) was 210.74 ± 39.28 in smokers, p-value 0.192. Moreover mean LDL-cholesterol (mg/dL) in smokers was 132.36 ± 36.75 with p-value = 0.827.[11]

The overwhelming body of epidemiological data points to smoking as a cause of cardiovascular disease. Many observational epidemiologic studies conducted in numerous nations have discovered a strong and reliable dose-response relationship between CHD and the length and intensity of smoking. 12 Men and women who smoke are at an increased risk of developing cardiovascular diseases such myocardial infarction, sudden death, stroke, peripheral vascular disease, and aortic aneurysm.[12] When compared to nonsmokers, smokers have a two- to four-fold higher risk of sudden mortality and a 70% higher risk of fatal CHD. Smoking can impact how common antianginal drugs like betablockers and calcium-channel blockers are metabolised, which can make therapy more difficult for smokers with established CHD.[13] Smoking cigarettes interacts with the other two in a positive way.

In accordance with other studies, we found abnormal lipid profiles in smokers, however, in future high risk population (smokers) may be screened for possible prevention from dyslipidemia (abnormal lipid profile) and then from CVD. Moreover smoking is a modifiable risk factor for dyslipidemia and these results may encourage smokers to quit their habits to live a healthy life.

CONCLUSION

We concluded that the smokers are at higher risk of developing dyslipidemia. Our results are encouraging for smokers to quit their habits to live a healthy life.

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