Original Research Article

A study of serum lipid profile in smokers and non-smokers: evaluation of role of smoking on lipid profile

Chandrashekar V. Kubihal, Hemalatha D. Naik*

Department of Biochemistry, Karnataka Institute of Medical Sciences, Hubli, Karnataka, India

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*Correspondence:
Dr. Hemalatha D. Naik,
E-mail: dr_hemanaik@yahoo.com

ABSTRACT

Background: Several studies have reported elevated blood cholesterol levels among persons who regularly smoke cigarettes and lowered blood cholesterol levels among persons quitting smoking. Other studies have also shown that smoking lowers high density lipoprotein level, resulting in an increased risk of coronary heart disease. Smoking also leads to increase in LDL cholesterol and triglyceride levels. The objective was to study serum lipid profile in smokers and non-smokers.

Methods: A cross sectional comparative study was carried in 100 subjects. The subjects were divided into two groups. First group consisted of 25 nonsmokers and second group of 75 smokers. The group of 75 smokers was again divided into three equal sub groups of 25 each depending upon the duration and intensity of smoking. Concentration of serum total cholesterol and HDL was determined by Zak's method. Concentration of serum LDL and VLDL cholesterol was determined by Friedwald's formula. Concentration of serum triglyceride level was determined by enzymatic end point peroxidase coupled method.

Results: All the values of lipid profile i.e., total cholesterol, triglycerides, LDL, VLDL were found to be significantly higher among the smokers compared to the non-smokers. HDL value was significantly lower among smokers. As the degree of smoking increased from mild to heavy smokers, the values of total cholesterol, triglycerides, LDL and VLDL increased. The degree of smoking was inversely proportional to HDL values i.e., the HDL value decreased as the smoking degree increased.

Conclusions: Thus, it can be said based on the present study that smoking affects and deranges the lipid profile of the person.

Keywords: HDL, LDL, Total cholesterol, Triglycerides, VLDL

INTRODUCTION

Cigarette smoking is the single most important preventable cause of illness and death. There is an increasing incidence of mortality due to cigarette smoking as compared to other causes of death like alcohol, addictive drugs and suicides. The first major study associating smoking with predicted risk of cardiovascular disease was published in the year 1958.

This was subsequently confirmed by numerous epidemiologic studies. The association has been so strong and so, consistent that smoking is now considered as a definitive cause of atherosclerosis. In the industrialized world, 50% of the deaths are attributed to atherosclerosis and its complications. In the United States, itself over four lakh individuals die prematurely each year from cigarette smoking representing approximately one out of every five deaths. Incidence of myocardial infarction in

heavy smokers is three times more common compared to nonsmokers. In India the effects of smoking could be even more harmful than western countries because of wide spread practice of smoking beedies which is more harmful than cigarettes.[1-3]

Several studies have reported elevated blood cholesterol levels among persons who regularly smoke cigarettes and lowered blood cholesterol levels among persons quitting smoking. Other studies have also shown that smoking lowers high density lipoprotein level, resulting in an increased risk of coronary heart disease. Smoking also leads to increase in LDL cholesterol and triglyceride levels. Even exposure to environmental tobacco smoke i.e. passive smoking results in decreased levels of HDL cholesterol demonstrated in children of smoker parents by Rosenses. Many studies have shown a dose dependent relationship between smoking and serum lipid profile.[4]

The mechanism by which smoking alters the lipoprotein metabolism is not clear. The various mechanisms postulated are stimulation of sympathoadrenal system by nicotine leading to lipolysis and increased serum free fatty acid level which leads to increased synthesis of VLDL from liver. Increased consumption of free fatty acid by heart leads to increased myocardial oxygen demand. Repressive action of smoking on the estrogen level which in turn lead to decreased HDL cholesterol. Smoker are thought to consume a diet rich in fat and cholesterol and poorer in fiber and cereals.[5]

Nicotine stimulates secretion of catecholamine leading to activation of adenyl cyclase of adipose tissue resulting in increased lipolysis, increased concentration of plasma free fatty acids, increased secretion of hepatic free fatty acids and hepatic triglycerides along with VLDL cholesterol in the blood stream.[6]

The plasma concentration of cholesterol and of its main component LDL cholesterol are established risk factor for the incidence of atherosclerotic vascular complications. Epidemiological studies have also consistently demonstrated that plasma concentration of HDL cholesterol is inversely correlated with the incidence of coronary artery disease. Human HDL is a heterogeneous mixture of lipoprotein particle comprising of two principal subtractions, HDL_2 and HDL_3. A widely held view is that the benefit of HDL is linked to HDL_2.

However, recent data from a case control study from the prospective physician's health study suggest that HDL_2 confers equal if not superior protection against myocardial infarction. In cross sectional studies, triglycerides show a univariate association with coronary artery disease and this association persists after cholesterol or LDL cholesterol taken into account. However, in prospective studies controlling also for HDL cholesterol concentration, triglycerides are eliminated as an independent risk factor in most studies.[7] With this background, present study has been undertaken to study the effect of smoking on lipid profile by comparing it with lipid profile between smokers and nonsmokers.

METHODS

This was a cross sectional comparative study carried out at JJM Medical College and hospital over a period of one year from November 2000 to October 2001.

The Ethical Committee of JJM Medical College had given the consent for conducting the present study. During the study, written informed consent was taken from each patient.

During the study period, it was possible to study a total of 100 subjects as per the inclusion and exclusion criteria set for the present study.

The subjects were divided into two groups. First group consisted of 25 nonsmokers and second group of 75 smokers. The group of 75 smokers was again divided into three equal sub groups of 25 each depending upon the duration and intensity of smoking. Mild smokers who had 1-5 years of smoking duration and smoking 10-15 cigarettes per day. Moderate smokers were those who had 6-10 years of smoking duration and were smoking 16-20 cigarettes per day. Heavy smokers were those who had more than 10 years of smoking duration and who were smoking more than 20 cigarettes per day.

Inclusion criteria includes the study group with history of smoking cigarettes and for controls, those who are free from habit of smoking.

Exclusion criteria were those who are eligible for the present study but not willing and subjects having diseases which influences lipid profile or subjects who are on lipid lowering drugs or diet restrictions and also persons chewing tobacco, ex-smokers, alcoholic, diabetic and hypertensive.

Procedure

After finding the person eligible for the present study, he was explained the nature of the study and consent was sought. Those willing to give the consent and able to cooperate brief history was taken from them.

Venous blood was collected by using sterile disposable syringe by taking all universal precautions. Later serum was separated from them. Biochemical analyses were made from the serum which are:

- Concentration of serum total cholesterol and HDL was determined by Zak's method.
- Concentration of serum LDL and VLDL cholesterol was determined by Friedwald's formula.
- Concentration of serum triglyceride level was determined by enzymatic end point peroxidase coupled method.

Statistical analysis

The data was recorded and analyzed using mean and standard deviation (+2SD). Students t-test was applied to compare the mean values between the two groups and if it was found that the p value was less than 0.05, it was taken as statistically significant i.e., the difference in the values of the two groups are present and the observation was not by chance.

RESULTS

Table 1 shows comparison of lipid profile between smokers and nonsmokers. All the values of lipid profile i.e. total cholesterol, triglycerides, LDL, VLDL were found to be significantly higher among the smokers compared to the nonsmokers (p <0.05). HDL value was significantly lower among smokers (p <0.05). Cholesterol value was 162.2±16.2 mg/dl in nonsmokers compared to 194.1±18.3 mg/dl in smokers. TG value was 121.9±32 mg/dl in nonsmokers compared to 180.8±32.2 mg/dl in smokers. HDLc value was 49.1±5.7 mg/dl in nonsmokers compared to 42.1±6.7 mg/dl in smokers. LDLc value was 88.8±21.1 mg/dl in nonsmokers compared to 116.2±25.1 in smokers.

Table 2 shows comparison of lipid profile between nonsmokers and different groups of smokers. It was found that as the degree of smoking increased from mild to heavy smokers, the values of total cholesterol, triglycerides, LDL and VLDL increased. The degree of smoking was inversely proportional to HDL values i.e. the HDL value decreased as the smoking degree increased. The value of cholesterol increased from 178.7±7.1 mg/dl in mild smokers to 210.7+16.1 in heavy smokers. The value of HDLc decreased from 47.2±2.2 mg/dl in mild smokers to 34.4±3.9 mg/dl in heavy smokers.

Table 1: Comparison of lipid profile between smokers and nonsmokers.

Groups	Tc (mg/dl)	TG (mg/dl)	HDLc (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
Non-Smokers (N = 25)	162.2+16.2	121.9+32	49.1+5.7	88.8+21.1	24.4+6.4
Smokers (N = 75)	194.1+18.3	180.8+32.2	42.1+6.7	116.2+25.1	36.2+6.5
t value	7.78	7.98	4.70	4.92	7.92
p value	<0.001	<0.001	<0.001	<0.001	<0.001

Table 2: Comparison of lipid profile between nonsmokers and different groups of smokers.

Lipid profile parameters	Non-smokers (N = 25)	Smokers			
		Mild smokers (N = 25)	Moderate smokers (N = 25)	Heavy smokers (N = 25)	Total smokers (N = 75)
Tc (mg/dl)	162.2+16.2	178.7+7.1	193+13.8	210.7+16.1	194.1+18.3
TG (mg/dl)	121.9+32	174.1+29.2	177.8+29.4	190.6+36.5	180.8+32.2
HDL-C (mg/dl)	49.1+5.7	47.2+2.2	44.8+4.7	34.4+3.9	42.1+6.7
LDL-C (mg/dl)	88.8+21.1	96.7+9.4	112.7+19.7	139.3+22.5	116.2+25.1
VLDL-C (mg/dl)	24.4+6.4	34.8+5.8	35.6+5.9	38.1+7.3	36.2+6.5

Table 3: Comparison of total cholesterol between non smokers and different groups of smokers.

Groups	Total cholesterol (mg/dl)		Group wise comparison		
	Range	Mean+SD	Mild smokers	Moderate smokers	Heavy smokers
Non-smokers (N = 25)	135.3-188.7	162.2+16.2	P <0.01	P <0.01	P <0.01
Mild smokers (N = 25)	162.5-190.8	178.7+7.1	-	P <0.01	P <0.01
Moderate smokers (N = 25)	176.8-221.2	193.0+13.8	-	-	P <0.01
Heavy smokers (N = 25)	180.7-236.8	210.7+16.1	-	-	-

One factor ANOVA (F = 55.9, P <0.001); Newman-Keul's Test (Min significance range = 12.6, p = 0.01), NS = Not significant.

Table 3 shows comparison of total cholesterol between nonsmokers and different groups of smokers. Analysis of variance was done between the different groups for total cholesterol. It was found that the F value was significantly more for comparison between smokers and nonsmokers as well as across the degrees of the smokers. The value of cholesterol increased from 178.7±7.1 mg/dl in mild smokers to 210.7±16.1 mg/dl in heavy smokers. It was 162.2±16.2 mg/dl in nonsmokers. Range in nonsmokers was 135.3-188.7 mg/dl which increased to 180.7-236.8 mg/dl in heavy smokers.

Table 4 shows comparison of triglyceride levels between nonsmokers and different groups of smokers. Analysis of variance was done between the different groups for triglycerides. It was found that the F value was significantly more for comparison between smokers and nonsmokers but not across the degrees of the smokers. The triglyceride value increased from 121.9±32.0 mg/dl in nonsmokers to 190.6±36.5 mg/dl in heavy smokers. The range in nonsmokers was 66-176 mg/dl which increased to 110-254 mg/dl in heavy smokers.

Table 4: Comparison of triglyceride levels between nonsmokers and different groups of smokers.

Groups	Triglycerides (mg/dl)		Group wise comparison		
	Range	Mean±SD	Mild smokers	Moderate smokers	Heavy smokers
Non-smokers (N = 25)	66-176	121.9±32.0	P <0.01	P <0.01	P <0.01
Mild smokers (N = 25)	130-234	174.1±29.2	-	NS	NS
Moderate smokers (N = 25)	116-230	177.8±29.4	-	-	NS
Heavy smokers (N = 25)	110-254	190.6±36.5	-	-	-

One factor ANOVA (F = 22.63, P <0.001), MSR = 29.0 (P = 0.01), NS = Not significant.

Table 5: Comparison of HDL-C levels between nonsmokers and different groups of smokers.

Groups	HDL-C (mg/dl)		Group wise comparison		
	Range	Mean±SD	Mild smokers	Moderate smokers	Heavy smokers
Non-smokers (N = 25)	38.7-62.9	49.1±5.7	NS	P <0.01	P <0.01
Mild smokers (N = 25)	41.9-63.1	47.2±2.2	-	NS	P <0.01
Moderate smokers (N = 25)	37.8-53.9	44.8±4.8	-	-	P <0.01
Heavy smokers (N = 25)	30.0-41.9	34.4±3.9	-	-	-

One factor ANOVA (F = 56.3, P < 0.001), MSR = 3.2 (P = 0.05), MSR = 4.0 (P = 0.01), NS = Not significant.

Table 5 shows comparison of HDL-C levels between nonsmokers and different groups of smokers. The HDL-C value was not significantly different between mild smokers and nonsmokers. Similarly, it was not found to be significant between mild and moderate smokers, but heavy smoking affected the HDL-C values significantly.

The value of HDLc decreased from 47.2±2.2 mg/dl in mild smokers to 34.4±3.9 mg/dl in heavy smokers. It was 49.1±5.7 mg/dl in nonsmokers.

The range was 38.7-62.9 in nonsmokers which decreased to 30.0-41.9 mg/dl in heavy smokers.

Table 6: Comparison of LDL-C levels between nonsmokers and different groups of smokers.

Groups	LDL-C (mg/dl)		Group wise comparison		
	Range	Mean±SD	Mild smokers	Moderate smokers	Heavy smokers
Non-smokers (N = 25)	54.8-120.6	88.8±21.1	NS	P <0.01	P <0.01
Mild smokers (N = 25)	72.5-113.7	96.7±9.4	-	P <0.05	P <0.01
Moderate smokers (N = 25)	83.71-146.6	112.7±19.7	-	-	P <0.01
Heavy smokers (N = 25)	99.8-175.6	139.3±22.5	-	-	-

One factor ANOVA (F =33.60, P < 0.001); MSR = 14 (P = 0.05); MSR = 17.2 (P = 0.01); NS = Not significant.

Table 6 shows comparison of LDL-C levels between nonsmokers and different groups of smokers. It was found that the LDL-C value was not significantly different between mild smokers and nonsmokers. But for all other groups, it was significantly different. The LDLc value was 88.8±21.1 mg/dl in nonsmokers which increased to 139.3±22.5 mg/dl in heavy smokers. The range in nonsmokers was 54.8-120.6 mg/dl which increased to 99.8-175.6 mg/dl in heavy smokers.

Table 7 shows comparison of VLDL-C levels between nonsmokers and different groups of smokers. It was found that the VLDL-C levels were not significantly different between mild and moderate smokers and also between mild and heavy smokers. It was also not significantly different between moderate and heavy smokers. But it was significantly different between smokers and nonsmokers. The value of VLDLc was 24.4±6.4 mg/dl which increased to 38.1±7.3 in heavy

smokers. The range in nonsmokers was 13.2-35.2 mg/dl which increased to 21.6-50.8 mg/dl in heavy smokers.

Table 7: Comparison of VLDL-C levels between nonsmokers and different groups of smokers.

Groups	VLDL-C (mg/dl)		Group wise comparison		
	Range	Mean±SD	Mild smokers	Moderate smokers	Heavy smokers
Non-smokers (N = 25)	13.2-35.2	24.4±6.4	P <0.01	P <0.01	P <0.01
Mild smokers (N = 25)	26.0-46.8	34.8±5.8	-	NS	NS
Moderate smokers (N = 25)	23.2-46.0	35.6±5.9	-	-	NS
Heavy smokers (N = 25)	21.6-50.8	38.1±7.3	-	-	-

One factor ANOVA (F =14.66, P < 0.001); MSR = 7.1 (P = 0.01); NS = Not significant.

DISCUSSION

In the present study, the mean levels of total cholesterol in control group and in total smokers are in the range of 162.2 and 194.1 mg/dl respectively. This difference was found to be highly significant (p <0.001). Among the groups, in controls, mild, moderate and heavy smokers it was in the range of 162.2 mg/dl, 178.7 mg/dl, 193 mg/dl and 210.7 mg/dl respectively. The difference among the groups was found to be highly significant (p <0.001). Analyzing the result with regard to intensity and duration of smoking, it was observed that on the whole, there was a significant increase in the level of serum total cholesterol with regard to an increase in duration and intensity of smoking. The association of total cholesterol level with smoking was indeed very prominent. The present study findings are in accordance with the study of Sinha AK et al, and others.[4,5,8-10]

The mean serum triglycerides levels in control group and in total smokers were in the range of 121.9 mg/dl and 180.8 mg/dl respectively. Among the groups, in controls, mild, moderate and heavy smokers, it was in the range of 121.9 mg/dl, 174.1 mg/dl, 177.8 mg/dl and 190.6 mg/dl respectively. The mean levels were increasing progressively from mild to heavy smokers showing a direct dose response relationship. These findings are in accordance with other studies.[5,7,11]

The mean levels of HDL-C in control group and in total smokers were in the range of 49.1 mg/dl and 42.1 mg/dl respectively. Among the groups, in controls mild, moderate and heavy smokers it was in the range of 49.1 mg/dl, 47.2 mg/dl, 44.8 mg/dl and 34.4 mg/dl respectively. The HDL-C values were found to be decreasing in smokers compared to nonsmokers. The values were decreasing progressively from mild to heavy smokers showing a dose dependent inverse relation with smoking. These findings are in accordance with other reports.[5,7,9]

The mean levels of LDL-C in the control group and in total smokers were in the range of 88.8 mg/dl and 116.2 mg/dl respectively. Among the groups in controls and, mild, moderate and heavy categories it was in the range of 88.8 mg/dl, 96.7 mg/dl, 112.7 mg/dl and 139.3 mg/dl respectively. In the present study, LDL-C values were found to be increasing progressively from mild to heavy smokers showing a dose dependent response. These findings are in accordance with other studies.[5,7,9,12]

The mean levels of VLDL-C in control group and in total smokers were in the range of 24.4 mg/dl and 36.2 mg/dl respectively. Among the different groups, in controls, mild, moderate and heavy smokers, it was in the range of 24.4 mg/dl, 34.8 mg/dl, 35.6 mg/dl, and 38.1 mg/dl respectively. The mean levels of VLDL-C were observed to be significantly higher in all groups of smokers more so in heavy smokers. These findings are in accordance with the findings of other reports.[6,10,11]

The mechanisms by which smoking causes the above noticed dyslipidemic changes are still not clear. It has been suggested that nicotine stimulates the secretion of catecholamine, leading to activation of adenyl cyclase of adipose tissue, resulting in increased secretion of hepatic triglycerides and VLDL into the blood stream. This repeated elevation of serum FFA over a prolonged period accelerates atherosclerosis.[11]

An increase in serum LDL-C and decrease in serum HDL-C is associated with an increased risk of atherosclerosis. Low levels of HDL-C are indicative of reduced efflux of cholesterol from the arterial wall thus favoring atherosclerosis. According to Mjøs OD et al, smoking has at least two lipid effects that might promote CHD and atherosclerosis.[9]

First nicotine in the cigarette smoke causes an increase in myocardial oxygen requirements by increasing the use of FFA by heart. Second, smoking by an unknown mechanism, lower the anti-atherogenic facto HDL_2.

According to Sinha AK et al, smoking represses the estrogen levels which may in turn leads to decreased HDL-C.[5] Difference in oral fat handling between smokers and nonsmokers along with nicotine induced lipolysis could be the cause for dyslipidemic changes in smokers.

CONCLUSION

The overall observation of the present study was that, there was an increase in concentration of total cholesterol, triglycerides, LDL-C and decrease in the concentration of HDL-C in smokers compared to nonsmokers. Thus, it can be said based on the present study that smoking affects and deranges the lipid profile of the person.

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