Original Research Article

Comparative Study of Lipid Profile in Young Smokers and Non- Smokers

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Received: 28-07-2021 / Revised: 22-08-2021 / Accepted: 08-10-2021

Abstract

Background and Objective: The present study provides a detailed profile of the plasma lipid and lipoprotein levels according to cigarette smoking status (smoker, ex-smoker and non-smoker) and dosage [number of cigarette(s) smoked per day] in this part of Rajasthan. **Materials and Methods:** The study is being carried out in 100 healthy male smokers and 100 healthy males non smokers selected from volunteers from general public, patient attendants and hospital staff of New Medical College and Hospital & MBS Hospital Kota. **Results:** Out of 100 patients in the present study the number of subjects in mild, moderate and high smokers group were 42 (42%), 44 (44%), 14 (14%) respectively. Smokers had higher total cholesterol, plasma triglycerides, serum LDL, serum VLDL and lower levels of serum HDL compared to non-smokers which was statistically significant. **Conclusion:** The present study provides a detailed profile of the plasma lipid and lipoprotein level according to cigarette/ beedis smoking status (smoker, ex-smoker and non-smoker) and dosage (number of cigarettes/ beedis smoked per day). Cigarette / beedi smoking is associated with significant lower levels of serum HDL and high levels of serum cholesterol, serum triglycerides, serum LDL levels.

Key words: Cigarette, Beedi,Smoking, Total Cholesterol, Plasma Triglycerides, Serum LDL, Serum VLDL.

Introduction

Tobacco is a serious threat to health[1] and a proven killer and ranks second as a cause of death[1] in the world. Worldwide, tobacco use causes more than 7 million deaths per year[1]. Tobacco use is an emerging pandemic marching forward relentlessly. Evidence accumulating since early 1950s indicate that more than 25 diseases are now known or strongly suspected to be causally related to smoking. WHO estimates that unless current smoking pattern is reversed, tobacco will be responsible for 10 million deaths per year, by the decade 2020–2030, with 70% of them occurring in developing countries.

In India tobacco kills 8–10 lakhs people each year and many of these deaths will occur in people who are very young. It has been estimated that an average of five-and-a-half minutes of life is lost for each cigarette smoked. Youth in general and adolescents in particular fall prey to this deadly habit with severe physical,psychological, and economic implications[2]. A large number of risk factors which predispose to atherosclerosis and coronary artery diseases have been identified. These include modifiable ones like hypertension, dyslipidemia, smoking, diabetes mellitus, changing lifestyle and non-modifiable ones like age and sex. As the number of risk factors in an individual increases, so does the risk of developing atherosclerosis and its complications mainly coronary artery diseases (CAD)[3]. In subject more than one of these risk factors the risk is more than additive[4. Although smoking has been established as an independent risk factor[5] for coronary heart disease, the mechanism by which it increases the risk of coronary heart disease are unclear.

However, studies to date have revealed incomplete, inconclusive or conflicting results about the association of smoking on the plasma lipid and lipoprotein levels. In some studies, smokers had increased plasma cholesterol levels, in others plasma cholesterol level have actually been lower[6]. Only a few studies have specifically examined the plasma lipoprotein according to smoking status or no. of cigarettes (dosage)[7]. Smokers are reported to have higher LDL and lower HDL cholesterol levels than non-smokers[4].

There is inadequate data on the association of smoking and dyslipidemia in India. Also there is wide spread habit of smoking cigarette and beed is and also increased prevalence of coronary artery disease among rural population in Kota district and surrounding areas. The present study provides a detailed profile of the plasma lipid and lipoprotein levels according to cigarette smoking status (smoker, ex-smoker and non-smoker) and dosage (number of cigarettes smoked per day) in this part of Rajasthan.

Materials & methods

The study is being carried out in 100 healthy male smokers and 100 healthy male non smokers selected from volunteers from general public, patient attendants and hospital staff of New medical college hospital & MBS hospital,Kota. After obtaining written consent, detailed history and physical examination was done in all subjects.

Inclusion criteria for smokers and non-smokers

1. The subjects were divided into 4 groups

a) Non- smokers: subjects who have never smoked or those who left smokingatleast 5 yrs before in the present study

b) Mild smokers: 1-10 cigarettes or 1-15 beedis / day for atleast 5 yrs or more

c) Moderate smokers: 11-20 cigarettes or 16- 30 beedis / day for atleast 5 yrs or more.

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d) Heavy smokers: more than 20 cigarettes or 30 beedis / day for atleast 5 yrs or more.

2. The subject's were chosen in age groups of 18 - 40 yrs of age

3. The subject's BMI were less than 28

4. The subjects were taking average Indian diet.

Exclusion criteria for smokers and non- smokers

1. Subjects having diseases mentioned below known to influence blood lipids were excluded from the study

- Diabetes mellitus
- Nephrotic syndrome
- Alcoholism
- Hypertension

2. Subjects who were on following drugs:

- HMG CoA reductase inhibitors
- Fibric acid derivatives
- Nicotinic acid
- Beta blockers
- Diuretics

3. Subjects who were on diet restriction

Methods

A detailed history was taken. Subjects were explained in detail about the study and written in formed consent was taken. Blood Sample was collected after overnight fasting under all aseptic precautions and sample was centrifuged at 2000rpmfor one minute. Lipid profile estimation which includes serum cholesterol, serum triglyceride, High density lipoprotein, low density lipoprotein, very low-density lipoprotein, fasting blood sugar (FBS), serum creatinine and urine for albumin, sugar and microscopic examination.

Statistical analysis

were measured by x^2 test (chi square test) with SPSS 21 version software. Statistical value P<0.05 were analyzed.

Results

The present study the number of Non-smokers in 18-20, 21-30 and 31-40 age group was 7, 77 and 16 respectively and number of Smokers in 18-20, 21-30 and 31-40 was 5, 28 and 67 respectively (table 1).

Table 1: Age distribution among non- smokers and smokers

Group	18-20	21-30	31-40
Non-smokers	7	77	16
Smokers	5	28	67

Smokers had higher total cholesterol levels compared to non-smokers (175.63 versus 140.12) this difference was statistically significant. Smokers had higher plasma triglyceride level compared to non-smokers (129.76 versus 101.8) this difference was statistically significant. Smokers had higher serum LDL levels compared to non-smokers, (118.11 versus 73.93) this difference was statistically significant. Smokers had higher VLDL levels compared to non-smokers (29.95 versus 20.36) this difference was statistically significant. Smokers had lower levels of serum HDL compared to non-smokers (31.56 versus 45.82) and this difference was statistically significant (table 2).

Table 2: Lipid profile in non-smokers and smokers.

Lipid profile	Non-smokers n = 100	Smokers n = 100	P value
Total cholesterol	140.12 ± 28.58	175.63 ±28.46	<0.05
Serum triglycerides	101.84 ± 24.23	129.76± 38.62	<0.05
Serum LDL	73.93 ± 28.35	118.11 ±29.08	<0.05
Serum VLDL	20.36 ± 4.84	29.95 ± 7.72	<0.05
Serum HDL	45.82 ± 7.65	31.56 ± 5.91	< 0.05

Smokers were further subdivided into mild, moderate, heavy group based on number of cigarette/beedis smoked and the values of each group were compared with non-smokers (table 3).

Table 3: Lipid profile in relation to number of cigarette/beedis smoked per dayin smokers as compared to non-smokers.

Lipid profile (mg/dl)	Non- smokers (n=100)	Mild smokers (n=42)	P Value	Moderate smokers (n=44)	P Value	Heavy Smokers (n=14)	P Value
Total cholesterol	140.12	149.85 ±12.32	<0.05	187.79 ±14.04	<0.05	214.71 ±27.30	<0.05
Serum triglycerides	101.84	117.78 ±28.71	<0.05	129.04 ±37.77	<0.05	167.92 ±44.77	<0.05
Serum LDL	73.93	91.60 ±12.61	<0.05	131.39 14.84	<0.05	155.91 ±28.37	<0.05
Serum VLDL	20.36	23.55 ±5.74	<0.05	25.80± 7.55	<0.05	33.58± 8.95	<0.05
Serum HDL	45.82	34.69 ±4.40	<0.05	30.59±5.29	<0.05	25.21 ± 5.87	<0.05

Total cholesterol value was highest in heavy smokers (214.71), less in moderate smokers (187.79) and least in mild smokers (149.85). The difference of these values with non-smokers was statistically significant.

The triglyceride levels were highest in heavy smokers (167.92), less in moderate smokers (129.04) and least in mild smokers (117.78). The difference of these values with non-smokers was statistically significant.

The serum LDL level were highest in heavy smokers (155.91), less in moderate smokers (131.39) and least in least in mild smokers (91.60). The difference of these values with non-smoker was statistically significant.

The serum VLDL level was highest in heavy smokers (33.58), less in moderate smokers (25.80) and least in mild smokers (23.55). The difference of these values with non-smokers was statistically significant.

The serum HDL level was lowest in heavy smoker group (25.21), higher in moderate smoker group (30.59) and highest in mild smoker group (34.69). The difference of these values compared to non-smoker group was statistically significant.

Discussion

Smoking is the major risk factor in the developing world but falling in developed nations. In the developing world, tobacco consumption is rising by 3.4% per year. Among young teenagers between the age of 13 to 15, about one in five smokes worldwide. Between 80,000 and 100,000 children worldwide start smoking every day - roughly half of whom live in Asia. Evidence shows that around 50% of those who start smoking in adolescent years go on to smoke in 15 to 20 years age[2].

Cigarette smokers have a higher risk of coronary artery disease than non-smokers. Several possible explanations have been offered for this association, including altered blood coagulation, impaired integrity of the arterial wall, and changes in blood lipid and lipoprotein

concentrations. Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease.

It is revealed that triglycerides, LDL, VLDL, HDL, and TC were statistically significantly higher in smokers as compared to non smokers. The mean serum total cholesterol in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum triglycerides were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum VLDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum LDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum HDL were higher in non smokers when compared to smokers, which was statistically significant ($p<0.05$).

Several studies have shown an association between cigarette smoking and altered serum lipid and lipoprotein concentrations, but many of these have lacked enough statistical power to establish a firm association. By combining the results of individual studies in the present analysis we have shown conclusively that smoking is associated with significantly higher serum concentrations of total cholesterol, triglycerides, very low-density lipoprotein cholesterol, and low-density lipoprotein cholesterol and lower serum concentrations of high density lipoprotein cholesterol and apolipoprotein AI and that this association is dose dependent. To our knowledge the data relevant to changes in serum lipid and lipoprotein concentrations associated with degree of exposure to cigarette smoke have not previously been compiled and reviewed. The dose dependent relation that we found may provide new evidence for a causal relation.

In support of these clinical observations Brischetto et al[7] proposed a mechanism to explain the link between smoking and some of the observed changes in serum lipid and lipoprotein concentrations: (a) nicotine stimulates the release of adrenaline by the adrenal cortex, leading to the increased serum concentrations of free fatty acid observed in smokers"; (b) free fatty acid is a well known stimulant of hepatic secretion of very low density lipoprotein and hence triglycerides; and (c) high density lipoprotein concentrations vary inversely with very low density lipoprotein concentrations in serum. Complementary to this mechanism is the finding that free fatty acid also stimulates hepatic synthesis and secretion of cholesterol.

Cigarette smoking is reported to be associated with an average 70% increase in the risk of death from coronary artery disease. In calculating this figure the surgeon general took into account all age groups. The excess risk of coronary artery diseaseassociated with smoking is, however, relatively higher among younger than older adults. To date sufficient data on prospective risk to allow the calculation of meaningful risk estimates are available only for serum cholesterol: a 1% increase in serum cholesterol concentration is associated with at least a 2% increase in the risk of coronary artery disease. The 3.0% mean increase in serum cholesterol concentrations identified among all current smokers in the present study would therefore be associated with an estimated 6.0% higher absolute risk of coronary artery disease with a range of 3- 6-9.0% higher absolute risk for light to heavy cigarette smoking. According to the present estimates, at least 9% of the total excess relative risk of coronary heart disease in current smokers could be accounted for by increased serum cholesterol concentrations. A further proportion of this excess risk is likely to be accounted for by changes in the other lipid and lipoprotein variables, but this cannot be quantified until their association with the risk of coronary artery disease is better defined prospectively[8].

In NS Neki et al[9] serum total cholesterol was significantly higher ($p<0.05$) when compared to non smokers. The mean triglyceride levels were higher in smokers than non smokers and this difference was statistically highly significant ($p<0.01$). The mean LDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean VLDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically highly significant ($p<0.01$).

In OA Odedeji et al[4] serum total cholesterol was significantly higher ($p<0.05$) when compared to non smokers. The mean triglycerides levels difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean LDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean VLDL difference between smokers and non-smokerswere not statistically significant ($p>0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically highly significant ($p<0.01$).

In Mokoto et al[10]found that the mean triglycerides levels difference between smokers and non smokers was statistically significant ($p<0.05$). The mean total cholesterol levels difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean VLDL difference between smokers and non smokers were not statistically significant ($p>0.05$). The mean LDL difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically significant ($p<0.05$).

In Aneela et al[11] found that the mean serum total cholesterol in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum triglycerides were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum VLDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum LDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum HDL were higher in non smokers when compared to smokers, which was statistically significant ($p<0.05$).

There is inverse relationship between smoking and serum HDL level. Also, this inverse relationship is dose dependent i.e. it is dependent on number of cigarettes /beedis smoked per day as in the present study. Serum HDL was lowest among heavy smoker group in present study (25.21±5.87), in Imamura et al (55.2) compared to moderate smokers in present study (30.59), in Imamura et al (55.5), and mild smoker group in present study group (34.69), in Imamura et al (62.2),which was statistically significant ($p<0.05$).

Serum TG was highest among heavy smoker group in present study (167.9), in Imamura et al (105.7) compared to moderate smokers in present study (129.0), in Imamura et al[12] (100.1), and mild smoker group in present study group (117.7), in Imamura et al[12] (90.7),which was statistically significant ($p<0.05$).

Serum LDL was highest among heavy smoker group in present study (155.9), in Imamura et al (118.2) compared to moderate smokers in present study (131.3), in Imamura et al[12] (123.0), and mild smoker group in present study group (91.60), in Imamura et al[12] (112.4),which was statistically significant ($p<0.05$).

Serum TC was highest among heavy smoker group in present study (214.7), in Imamura et al[12] (194.6) compared to moderate smokers in present study (187.7), in Imamura et al[12] (198.5), and mild smoker group in present study group (149.8), in Imamura et al[12] (192.7),which was statistically significant ($p<0.05$).

As serum HDL is a protective risk factor against coronary heart disease, this greater risk to smokers of coronary heart disease development may result from this HDL lowering effect of smoking.

Conclusion

From the results of the present study, it may be concluded that, cigarette smoking in young adults induces dyslipidemic state in the direction of increased risk for coronary artery disease. So it is strongly recommended to avoid smoking for the benefit of cardiac health.

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Conflict of Interest: None; **Financial Support:** Nil