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Comparative study to evaluate lipid profile in smoking and non-smoking individual at tertiary care centre

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Abstract---Background: Cigarette smoking, which is considered a major risk factor for atherosclerosis and coronary heart disease, may lead to changes in normal plasma lipid profile. The aim of the study was to evaluate the effect of cigarette smoking on lipid parameters in smokers and non-smokers. Material and Methods: A cross sectional comparative study was carried out in 200 subjects. The subjects were divided into two groups. Group A consisted of 100 smokers and Group B consisted of 100 Non-smokers. After overnight fasting following laboratory investigations were done in all subjects, Serum total cholesterol, Serum high density lipoprotein (HDL), Serum low density lipoprotein (LDL), Serum very low-density lipoprotein (VLDL), Serum triglyceride (TGL), Fasting blood sugar (FBS), Serum Creatinine, Urine for albumin, sugar and microscopic examination and the results of these tests were compared between the two groups. Results: All the values of lipid profile, TC, HDL, LDL, VLDL, TGL, were found to be significantly higher among the smokers compared to the non-smokers. The mean value of HDL was higher in non-smokers when compared to that of smokers and were statistically significant. The Total Cholesterol, Triglycerides, serum LDL, serum VLDL level was highest in

heavy smokers, less in moderate smokers and least in mild smokers, and was statistically significant with non-smokers. The serum HDL level was lowest in heavy smoker group (30.40 ± 3.49 mg/dl), higher in moderate smoker group (32.49 ± 3.49 mg/dl) and highest in mild smoker group (33.53 ± 3.40 mg/dl). The difference of these values compared to non-smoker group was statistically significant. The degree of smoking was inversely proportional to HDL values i.e., the HDL value decreased as the smoking degree increased. Conclusions: Cigarette /beedi smoking is associated with significant lower levels of serum HDL and high levels of serum cholesterol, serum triglycerides, serum LDL levels. Further this association is dependent on number of cigarette/beedis smoked per day. The greater risk to smokers for the development of coronary heart disease results from this HDL lowering effect of smoking.

Keywords---HDL, LDL, total cholesterol, triglycerides, VLDL.

Introduction

Tobacco smoking is the greatest public health burden in the world. ^[1] It contributes to 8 million deaths globally each year. Among these deaths, 7 million are caused by direct smoking, while another 1.2 million are caused by secondhand smoke. ^[2] The majority of the world's 1.3 billion smokers (80%) live in low/middle-income countries. ^[3] It is responsible for 1.6 million deaths in the WHO South-East Asia Region (SEAR), which is home to the world's largest tobacco manufacturers and users. ^[4]

India and Indonesia are two of the world's top five tobacco producers. Tobacco causes more deaths than AIDS, alcohol, cocaine, homicide, suicide and road traffic accidents. ^[5] Sri Lanka has an annual 20 000 death rate owing to tobacco use. ^[6] In regard to the cardiovascular diseases (CVD), South Asia saw a 73% rise in mortality attributable to ischaemic heart disease between 1990 and 2010, compared with a global increase of 30%. Additionally, South Asians (those from India, Pakistan, Bangladesh, Nepal and Sri Lanka) have been observed to suffer their first myocardial infarction (MI) about 10 years early than other ethnic groups. ^[7]

Cigarette smoking is widely known to be associated with CVDs. Cigarette smoking has another negative impact on lipid profile (LP) levels of blood. Nicotine has a significantly negative effect on lipid metabolism and regulation. ^[8] Smoking-induced lipid and lipoprotein abnormalities are hypothesised to have an influence on smoking-induced atherosclerosis. ^[9] Scientists have discovered that smoking produces considerably higher levels of total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C) and lower levels of high-density lipoprotein cholesterol (HDL-C). ^[10] The alterations mentioned above are linked to an increased risk of CVD. ^[11] However, the details related to the very LDL-C (VLDL) and TC/HDL ratio are poorly addressed.

The present study provides a detailed profile of the plasma lipid and lipoprotein levels according to cigarette smoking status (smoker, ex-smoker and nonsmoker) and dosage (number of cigarette smoked per day) in this part of area and to know if smoking has dyslipidemic potential in young individuals.

Material and Methods

This was a cross sectional comparative study carried out at Tertiary care teaching hospital over a period of one year. During the study period, it was possible to study a total of 240 subjects as per the inclusion and exclusion criteria set for the present study.

The subjects were divided into two groups. Study group consisted of 120 smokers and Control group of 120 non-smokers. The study group of 120 smokers was again divided into three subgroups depending upon the number of cigarettes smoked along with duration of smoking.

Table:1. Division of groups based on number of cigarettes smoked

Subgroups	Number of cigarettes smoked along with duration of smoking
Nonsmokers.	Never smoked or who left smoking atleast 5 years before the present study
Mild Smokers.	1-10 cigarettes or 1-15 beedis/day for atleast 5 years or more
Moderate smokers.	11-20 cigarettes or 16-30 beedis/day for atleast 5 years or more
Heavy smokers.	More than 20 Cigarettes or 30 beedis/day for atleast 5 years or more

Inclusion criteria includes the study group with history of smoking cigarettes and for controls, those who are free from habit of smoking, between the age groups of 18 - 40 yrs of age with BMI less than 28.4, and who were taking average Indian diet. Exclusion criteria were those who are eligible for the present study but not willing and subjects having following diseases like, Diabetes mellitus, Nephrotic syndrome, Alcoholism, Hypertension which can alter lipid profile and who are on the following drugs like HMG CoA reductase inhibitor, Fibric acid derivatives, Nicotinic acid, Beta blocker, Diuretics and if any subjects who were on diet restriction.

Methods

After overnight fasting, following laboratory investigations were done in all subjects, Serum total cholesterol, Serum high density lipoprotein (HDL), Serum low density lipoprotein (LDL), Serum very low-density lipoprotein (VLDL), Serum triglyceride (TGL), Fasting blood sugar (FBS), Serum Creatinine, Urine for albumin, sugar and microscopic examination.

Statistical analysis

The data was recorded and analyzed using mean, standard deviation, Z test and chi square test.

Results

The number of subjects in mild smokers group were 47 (39.1%), the number of subjects in moderate smokers group were 54 (45%), the number of subjects in heavy smokers were 19 (15.8%) of the total 120 smokers.

Table 2: Distribution of smokers based on no. of cigarettes/beedis smoked per day

Group	No. of subjects	Percentage
Mild smokers	47	39.1%
Moderate smokers	54	45%
Heavy Smokers	19	15.8%

Table 3: Age distribution among non- smokers and smokers

Group	18-20 years	21-30 years	31-40 years
Non-smokers	13	83	24
Smokers	11	33	76

There were 13 non-smokers and 11 smokers from 18-20 years, 83 non-smokers and 33 smokers from 21-30 years and 24 non-smokers and 76 smokers from 31-40 years age.

Table 4: Lipid profile in non-smokers and smokers

Lipid profile	Non-smokers	Smokers	P value
Total cholesterol	152.53 ± 16.63	221.47 ± 18.54	<0.05
Serum triglycerides	139.65 ± 14.63	209.63 ± 17.75	<0.05
Serum LDL	79.85 ± 7.87	148.68 ± 5.97	<0.05
Serum VLDL	27.93 ± 2.92	41.95 ± 7.72	<0.05
Serum HDL	44.75 ± 5.84	30.84 ± 4.85	< 0.05

Values are mean +/- standard deviation in mg/dl

P value are derived from analysis of variance

P value > 0.05 not significant.

P value < 0.05 significant

Smokers had higher total cholesterol levels compared to non-smokers (221.47 versus 152.53 mg/dl) this difference was statistically significant. Smokers had higher plasma triglyceride level compared to non-smokers (209.63 versus 139.65 mg/dl) this difference was statistically significant. Smokers had higher serum LDL levels compared to non-smokers, (148.68 versus 79.85 mg/dl) this difference was statistically significant. Smokers had higher VLDL levels compared to non-

smokers (41.95 versus 27.93 mg/dl) this difference was statistically significant. Smokers had lower levels of serum HDL compared to non-smokers (30.84 versus 44.75 mg/dl) and this difference was statistically significant.

Table 5: Lipid profile in relation to number of cigarette/beedis smoked per day in smokers as compared to non-smokers

Lipid profile (mg/dl)	Nonsmokers (n=120)	Mild smokers (n=47)	P Value	Moderate smokers (n=54)	P Value	Heavy Smokers (n=19)	P Value
Total cholesterol	152.53±16.63	155.54±14.64	<0.05	178.50±14.04	<0.05	221.53±24.52	<0.05
Serum triglycerides	139.65 ±14.63	157.64±15.65	<0.05	177.86±15.76	<0.05	198.59±25.85	<0.05
Serum LDL	79.85 ±7.87	94.53±9.54	<0.05	121.39 ±12.84	<0.05	145.91±14.37	<0.05
Serum VLDL	27.93 ±2.92	33.38±5.74	<0.05	38.80±3.49	<0.05	41.49±4.37	<0.05
Serum HDL	44.75 ±5.84	33.53±3.40	<0.05	32.49 ±3.49	<0.05	30.40 ±3.49	<0.05

Values are mean +/- standard error in mg/dl.

Values are from analysis of variance.

Values > 0.05 are not significant.

Values < 0.05 are significant.

Total cholesterol value was highest in heavy smokers (221.53±24.52 mg/dl), less in moderate smokers (178.50±14.04 mg/dl) and least in mild smokers (155.54±14.64 mg/dl). The difference of these values with non-smokers was statistically significant. The triglyceride levels were highest in heavy smokers (198.59±25.85 mg/dl), less in moderate smokers (177.86±15.76mg/dl) and least in mild smokers (157.64±15.65 mg/dl). The difference of these values with non-smokers was statistically significant. The serum LDL level were highest in heavy smokers (145.91±14.37 mg/dl), less in moderate smokers (121.39 ±12.84 mg/dl) and least in least in mild smokers (94.53±9.54mg/dl), the difference of these values with non-smoker was statistically significant. The serum VLDL level was highest in heavy smokers (41.49±4.37 mg/dl), less in moderate smokers (38.80±3.49mg/dl) and least in mild smokers (33.38±5.74mg/dl). The difference of these values with non-smokers was statistically significant. The serum HDL level was lowest in heavy smoker group (30.40 ±3.49 mg/dl), higher in moderate smoker group (32.49 ±3.49 mg/dl) and highest in mild smoker group (33.53±3.40 mg/dl). The difference of these values compared to non-smoker group was statistically significant.

Discussion

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 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 smoking have not previously been compiled and reviewed. The dose dependent relation that we found may provide new evidence for a causal relation.

In the present study, Total cholesterol value was highest in heavy smokers (221.53 ± 24.52 mg/dl), less in moderate smokers (178.50 ± 14.04 mg/dl) and least in mild smokers (155.54 ± 14.64 mg/dl). The difference of these values with non-smokers was statistically significant. The triglyceride levels were highest in heavy smokers (198.59 ± 25.85 mg/dl), less in moderate smokers (177.86 ± 15.76 mg/dl) and least in mild smokers (157.64 ± 15.65 mg/dl). The difference of these values with non-smokers was statistically significant. The serum LDL level were highest in heavy smokers (145.91 ± 14.37 mg/dl), less in moderate smokers (121.39 ± 12.84 mg/dl) and least in least in mild smokers (94.53 ± 9.54 mg/dl), the difference of these values with non-smoker was statistically significant.

The serum VLDL level was highest in heavy smokers (41.49 ± 4.37 mg/dl), less in moderate smokers (38.80 ± 3.49 mg/dl) and least in mild smokers (33.38 ± 5.74 mg/dl). The difference of these values with non-smokers was statistically significant. The serum HDL level was lowest in heavy smoker group (30.40 ± 3.49 mg/dl), higher in moderate smoker group (32.49 ± 3.49 mg/dl) and highest in mild smoker group (33.53 ± 3.40 mg/dl). The difference of these values compared to non-smoker group was statistically significant.

Blood lipids play an important role in the human body, contributing to various biochemical functions and acting as a structural component but unstable lipid fractions are hazardous to human health. ^[12] This study also confirmed that continued smoking is significantly associated with dyslipidaemia. Smoking-related dyslipidaemia can be explained by a variety of processes. Our findings revealed that smokers had considerably higher TC levels than non-smokers in accordance with previous investigations. ^[13] TG, LDL and VLDL of smokers were also high compared with the non-smokers ($p < 0.001$) and our findings are compatible with several studies. ^[13] Furthermore, smokers had lower HDL values ($p = 0.016$) than anthropometrically matched non-smokers and the finding is consistent with those of other studies. ^[14] However, a large Japanese cohort found no difference in TC and LDL-C of smokers despite increasing TG and decreasing HDL. ^[15] This might be because of various conflicting variables related to lipid metabolism, dietary lipid intake, ethnicity or other factors such as the exercises.

The results of this study revealed that smokers' HDL-C levels are significantly low compared with non-smokers ($p < 0.001$), consistent with other studies. [16] Decreasing the HDL-C and increasing the LDL-C and VLDL-C is mostly associated with CVDs. One of the reasons for atheroma and coronary artery disease is an imbalance between good and bad cholesterol. Serum lipid levels in smokers might rise for a variety of reasons. During smoking, high concentrations of nicotine are taken into the circulation via the lungs. Consistent nicotine exposure induces the release of catecholamines from the body, which is caused by the activation of adenylyl cyclase in adipose tissue and increase in lipolysis, and the release of free fatty acids into the circulation. Increased amounts of free fatty acids in the liver boost TG and VLDL-C production, resulting in higher levels of TG and VLDL-C in the blood. [17] Increased LDL-C and VLDL-C levels in the blood contribute to a decrease in HDL-C levels. [18] In addition to nicotine-mediated catecholamine release, researchers have found another mechanism that contributes to smokers' lower HDL-C levels. Smokers have higher levels of homocysteine than non-smokers. Increased homocysteine is known to have a negative impact on HDL-C levels and reduces HDL-C levels in smokers. [19]

Conclusion

From the results of the present study, it may be concluded that, cigarette smoking in young adults induces dyslipidaemic 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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