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Original Research Article

STUDY OF LIPID PROFILE IN YOUNG SMOKERS

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ABSTRACT

Background: Cigarette smoking is a major modifiable risk factor for cardiovascular diseases and is associated with significant alterations in lipid metabolism. It leads to increased levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides, along with decreased levels of anti-atherogenic high-density lipoprotein (HDL). The present study was undertaken to evaluate the lipoprotein profile in healthy young smokers and to assess the effect of smoking severity and duration.

Materials and Methods: This cross-sectional study included 50 apparently healthy young male smokers and 25 age- and weight-matched non-smokers as controls. All participants underwent routine clinical evaluation and biochemical investigations, including lipid profile estimation. Smoking severity was assessed based on the number of cigarettes smoked per day, and duration was categorized in years.

Results: The mean duration of smoking was 5.9 years. The mean body mass index (BMI) was comparable between smokers (23.4 kg/m²) and non-smokers (22.8 kg/m²). Smokers exhibited significantly higher levels of total cholesterol, LDL cholesterol, and triglycerides, while HDL cholesterol levels were significantly reduced compared to non-smokers ($p < 0.001$). A dose-dependent relationship was observed, with lipid levels increasing in proportion to the number of cigarettes smoked per day and duration of smoking, particularly beyond 6 years. HDL cholesterol showed a consistent decline with increasing smoking exposure.

Conclusion: Smoking is associated with significant dyslipidemia characterized by elevated atherogenic lipids and reduced HDL cholesterol. Both severity and duration of smoking have a direct impact on lipid alterations, emphasizing the importance of early smoking cessation to reduce cardiovascular risk.

Keywords: Lipid Profile, Dyslipidemia, Total Cholesterol, LDL Cholesterol, HDL Cholesterol and Triglycerides.

INTRODUCTION

Smoking of tobacco is a vice that has been practiced by people all over the world cutting across national and social barriers. The increasing habit of smoking has resulted in a high-incidence of tobacco related diseases all over the developing world. Though smoking of tobacco started centuries ago, the health and environmental hazards posed by it was recognized only in the 20th century. Illness caused by both passive and active smoking is a major cause of morbidity and mortality in developing as well as developed nations. Besides it is also a cause of concern for the economy due to loss of man-hours and increased expenditure. The psychological reinforcing effects of nicotine on the CNS encourage the progression from early experimentation of tobacco to chronic exposure, then addiction. Once addicted, the smoker faces an unacceptable risk for a frightening array of neoplastic and cardiovascular disorders. Even without pulmonary symptoms, the smoker has a chronic inflammatory disease of lower airways. The effects of tobacco on the teeth and mouth can range from unsightly to disfiguring to life threatening. Adjacent structures such as pharynx, larynx and esophagus are also at risk for cancer. And few realize how the ability to heal surgical or other wounds is jeopardised.[1,2]

The greatest responsibility of physicians is to combat smoking, both through their advice to patients and through their influence- in the community through health education. The addictive effects of nicotine account for most of this persistent personal and public health dilemma. Recognition of tobacco use as an addiction and of nicotine as the addictive drug is essential for effective patient management. The essential criteria for defining drug addiction are compulsive use, psychoactive effects and drug reinforced behavior. Nicotine use fulfils these criteria because it causes a compelling urge to smoke, gives a pleasurable alteration in mood and motivates chronic tobacco seeking. Tolerance and physical dependence, manifested by abstinence, mediated withdrawal syndrome, contribute to the strong control exerted by nicotine on smoking behavior.

Smokers regulate their nicotine dose to obtain desired effects: these include both intrinsic positive effects, such as pleasure and enhanced performance and avoidance of the withdrawal syndrome. This syndrome is characterized by anger, anxiety, difficulty in concentration, impatience, restlessness and craving for tobacco products. Most of these symptoms peak in 1-2 days and, return to base line within 3-4weeks of quitting. The use of tobacco products is a complex; learned behavior that is woven into the fiber of daily living and. is linked to how the smoker deals with the world. Numerous daily activities, thoughts and emotions serve as powerful cues to smoke, such as conditioned tics become paired with positive neuro regulatory effects on nicotine to reinforce the addictive process. Personal characteristics such as educational level, belief in one's ability to change and coping skills are determinants of tobacco use. Similarly environmental factors such as level of acceptance of smoking in the home, peer group, workplace and commonly norms influence smoking behavior. Cigarette smoking leads to increased serum level of total cholesterol, LDL choles-terol, Triglyceride levels and decreased level of anti atherogenic HDL cholesterol. Many studies have shown a dose-dependent relationship between smoking and lipoprotein profile.[3] Hence this study was-taken up to know the lipoprotein pattern in healthy young smokers.

MATERIALS AND METHODS

The present study was carried out in fifty apparently healthy male smokers and twenty-five healthy non-smokers. All the subjects hailed from mid-socio economic strata. The age group was between 25-35 yrs. Persons abusing alcohol, ex-smokers and having risk factors like diabetes mellitus, hypertension, and obesity were excluded from the study. Patients on drugs like beta-blockers and thiazide diuretics which were found to interfere with lipid profile were also excluded from the study.

All the smokers were divided into two groups according to their severity of smoking.

A. Those who regularly smoke 1-10 cigarettes per day
B. Those who regularly smoke 11-20 cigarettes per day

There were no subjects who were smoking more than 21 cigarettes per day in the present study. Depending on the duration of smoking they were divided into two groups.

A. Smoking for the duration of 1 to 5 years
B. Smoking for the duration of 6 to 10 years.

After a detailed history followed by clinical examination, routine investigations like Hb, TC, DC, ESR, FBS, Serum creatinine, urine analysis and electrocardiogram were done in all the cases.

Instructions were given to the smokers as well as controls to take a normal diet for 3 days before the lipid profile estimation was done, since food intake few hours before study would have impaired the results. Venous blood was drawn after overnight fasting. Lipid was estimated which included total serum cholesterol, HDL-cholesterol, LDL-cholesterol and serum triglycerides.

Lipid profile was estimated by CHOD-PAP method for total cholesterol, PT-PAP method for HDL-cholesterol, GPD=PAP, method for triglycerides. LDL-cholesterol was calculated from the formula: **LDL – C = Total cholesterol – (TGL / 5) – HDL-C**.

RESULTS

A total of 75 subjects were included in the study, comprising 50 smokers and 25 non-smokers. Both groups were comparable in terms of age, BMI, weight, and dietary habits.

Table 1: Distribution of Smokers According to Duration and Quantity of Smoking

Duration (Years)	1–10 Cig/day (n, %)	11–20 Cig/day (n, %)	Total
1–5	15 (30%)	10 (20%)	25
6–10	15 (30%)	10 (20%)	25
Total	30 (60%)	20 (40%)	50

Majority of smokers (60%) consumed 1–10 cigarettes/day, while 40% consumed 11–20 cigarettes/day. Equal distribution was observed in both duration groups.

Table 2: Lipid Profile in Smokers and Non-Smokers

Parameter (mg/dl)	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	t-value	p-value
Total Cholesterol	204.7 ± 27.2	169.2 ± 18.5	5.8	<0.001
HDL Cholesterol	36.02 ± 5.9	40.84 ± 3.4	15.4	<0.001
LDL Cholesterol	159.6 ± 31.1	140.84 ± 16.53	11.5	<0.001

Triglycerides	163.28 ± 38.7	116.72 ± 23.3	22.6	<0.001

Smokers showed significantly higher total cholesterol, LDL, and triglycerides, along with significantly lower HDL levels compared to non-smokers ($p < 0.001$).

Table 3: Lipid Profile According to Severity of Smoking

Parameter (mg/dl)	1–10 Cig/day (Mean ± SD)	11–20 Cig/day (Mean ± SD)	t-value	p-value
Total Cholesterol	197.13 ± 22.9	216.25 ± 33.2	2.6	<0.01
HDL Cholesterol	37.08 ± 6.1	35 ± 5.9	1.2	<0.01
LDL Cholesterol	157.35 ± 20.7	163 ± 42.8	0.92	NS
Triglycerides	158.13 ± 35.1	171 ± 44.4	1.1	NS

With increasing smoking severity, total cholesterol increased significantly, while HDL decreased significantly. LDL and triglycerides showed an increasing trend but were not statistically significant.

Table 4: Lipid Profile According to Duration of Smoking

Parameter (mg/dl)	1–5 Years (Mean ± SD)	6–10 Years (Mean ± SD)	t-value	p-value
Total Cholesterol	195.24 ± 26.9	214.32 ± 22.7	2.9	<0.001
HDL Cholesterol	39.4 ± 6.3	33.08 ± 3.4	4.1	<0.001
LDL Cholesterol	145.86 ± 33.4	173.36 ± 21.3	3.3	<0.01
Triglycerides	148.32 ± 41.7	178.24 ± 30.1	3.1	<0.01

Longer duration of smoking was associated with significant increase in total cholesterol, LDL, and triglycerides, along with significant reduction in HDL levels.

Table 5: Lipid Profile in Smokers (1–5 Years) According to Severity

Parameter (mg/dl)	1–10 Cig/day (Mean ± SD)	11–20 Cig/day (Mean ± SD)	t-value	p-value
Total Cholesterol	190.7 ± 22.3	202 ± 31.8	1.5	<0.1
HDL Cholesterol	40.5 ± 6.2	37.81 ± 6.4	1.1	<0.2
LDL Cholesterol	148.7 ± 22.2	141.61 ± 46.5	—	NS
Triglycerides	138.96 ± 32.6	162.5 ± 50.7	1.4	<0.1

Among individuals smoking for 1–5 years, changes in lipid profile with increasing severity were mild and not statistically significant.

Table 6: Lipid Profile in Smokers (6–10 Years) According to Severity

Parameter (mg/dl)	1–10 Cig/day (Mean ± SD)	11–20 Cig/day (Mean ± SD)	t-value	p-value
Total Cholesterol	203.5 ± 16.8	230.51 ± 21.2	3.6	<0.001
HDL Cholesterol	33.66 ± 3.3	32.21 ± 3.4	1.1	<0.2
LDL Cholesterol	166 ± 15	184.41 ± 25.1	0.9	<0.2
Triglycerides	177 ± 25.3	179.51 ± 36.1	0.2	NS

In smokers with longer duration (6–10 years), total cholesterol increased significantly with smoking severity, while other lipid parameters showed non-significant trends.

DISCUSSION

Cigarette smoking is a well-established modifiable risk factor for cardiovascular disease, exerting its deleterious effects through multiple mechanisms, including dyslipidemia, oxidative stress, endothelial dysfunction, and inflammation. In the present study, smoking was found to significantly alter lipid profile parameters, with increased total cholesterol, LDL cholesterol, and triglycerides, along with decreased HDL cholesterol. These findings are consistent with both classical and recent literature.[3,4]

Our results demonstrate a clear association between smoking and dyslipidemia, which is in agreement with recent studies by Momayyezi et al,[5] who reported significantly higher LDL and total cholesterol levels and reduced HDL levels among smokers compared to non-smokers . Similarly, a recent comparative study on young smokers (2025) also confirmed that smokers exhibit significantly elevated triglycerides, LDL, and total cholesterol, along with reduced HDL, indicating a pro-atherogenic lipid profile.[6]

The present study also demonstrated a dose-dependent relationship between smoking severity and lipid alterations. This finding aligns with the observations of Girish & Harish,[7] and more recent evidence from SAGE, which showed that increasing cigarette consumption is associated with progressively higher LDL levels and significantly lower HDL levels. Furthermore, higher smoking intensity has been shown to increase the odds of dyslipidemia by more than twofold, emphasizing the cumulative metabolic impact of smoking.

The effect of duration of smoking on lipid profile was also evident in this study. Longer duration (6–10 years) was associated with significantly higher total cholesterol, LDL, and triglycerides, along with lower HDL levels. These findings are supported by studies such as Ashraf et al,[8] and Patil et al,[9] which reported that lipid abnormalities worsen with increasing duration of smoking exposure . This

cumulative effect may be attributed to prolonged exposure to nicotine and other toxic substances, leading to persistent alterations in lipid metabolism.

The decrease in HDL cholesterol observed in smokers is particularly significant, as HDL plays a protective role against atherosclerosis. Smoking-induced oxidative stress is known to impair HDL function and reduce its concentration. Studies have shown that smoking increases lipid peroxidation and decreases lipoprotein lipase activity, thereby elevating triglycerides and lowering HDL levels. Additionally, smoking promotes hepatic overproduction of VLDL, contributing to increased circulating triglycerides.

Despite strong overall agreement with most studies, some earlier studies such as those by Brischetto et al,[10] reported non-significant changes in certain lipid parameters, particularly in light smokers. This discrepancy may be due to differences in sample size, age group, ethnicity, and smoking patterns.

The findings of the present study are clinically significant, as dyslipidemia is a major contributor to atherosclerosis and coronary artery disease. The observed linear relationship between smoking severity, duration, and lipid abnormalities highlights the importance of early intervention. Notably, several studies have demonstrated that lipid levels, particularly HDL cholesterol, improve following smoking cessation, further reinforcing the reversibility of these changes.[11,12]

CONCLUSION

The present study confirms that smoking induces significant dyslipidemia characterized by increased atherogenic lipids and decreased HDL cholesterol. Both the severity and duration of smoking have a direct and cumulative impact on lipid profile alterations. These findings are consistent with recent global evidence and emphasize the urgent need for smoking cessation strategies, especially among young adults, to reduce long-term cardiovascular risk.

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