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IMPACT OF CIGARETTE SMOKING ON LIPID PROFILE AND ITS CORRELATION WITH SMOKING PARAMETERS IN ADULT MALES AT A TERTIARY CARE HOSPITAL

Medicine

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ABSTRACT

Background: Cigarette smoking is a well-established risk factor for cardiovascular disease, primarily through its adverse effects on lipid metabolism. Smoking intensity and cumulative exposure have been shown to alter lipid levels, thereby accelerating atherogenesis. **Objectives:** To assess the effect of cigarette smoking on lipid profile among adult males attending a tertiary care hospital, and to evaluate the correlation between smoking parameters (cigarettes per day and pack-years) and serum lipid levels. **Methods:** This prospective cross-sectional observational study was conducted over 24 months in the Department of Medicine, Era's Lucknow Medical College and Hospital, Uttar Pradesh. A total of 84 adult male participants were enrolled, including 42 smokers (cases) and 42 age-matched non-smokers (controls). Detailed smoking history and clinical data were recorded. Fasting blood samples were analyzed for lipid profile parameters (TC, TG, HDL-C, LDL-C, VLDL-C) using standardized automated analyzers. Data were analyzed using SPSS software. Group comparisons were made using unpaired t-tests and chi-square tests, while Pearson correlation and regression analysis were used to assess associations between smoking parameters and lipid levels. A p-value <0.05 was considered significant. **Results:** Smokers had significantly higher mean levels of TC (213.6 ± 41.39 vs 145.25 ± 33.50 mg/dL), TG (189.05 ± 71.04 vs 123.11 ± 33.26 mg/dL), LDL-C (139.57 ± 36.68 vs 80.52 ± 29.77 mg/dL), and VLDL-C (37.81 ± 14.21 vs 24.62 ± 6.65 mg/dL), while HDL-C was significantly lower (36.21 ± 8.56 vs 40.44 ± 9.25 mg/dL) compared to non-smokers ($p<0.05$ for all). The prevalence of dyslipidemia was significantly higher among smokers, with relative risks of 31.4 for raised cholesterol, 3.8 for raised triglycerides, and 3.3 for raised LDL-C. Increasing cigarette consumption per day and higher pack-years were strongly correlated with worsening lipid parameters, particularly TC ($r=0.76$, $p<0.0001$) and LDL-C ($r=0.66$, $p<0.0001$).**Conclusion:** Cigarette smoking is strongly associated with adverse alterations in lipid profile, and the severity of dyslipidemia correlates directly with smoking intensity and duration. These findings highlight smoking as a major modifiable cardiovascular risk factor and emphasize the importance of smoking cessation and regular lipid monitoring in preventive cardiology.

KEYWORDS

Cigarette Smoking, Lipid Profile, Dyslipidemia, Cardiovascular Risk, Pack-Years, Smoking Intensity.

INTRODUCTION

Globally, tobacco consumption is a major public health issue, responsible for around 8 million deaths annually, with a large proportion in low- and middle-income countries, including India [1]. India is the second-largest consumer of tobacco worldwide, with 45.5% of men using tobacco in some form; 24.6% smoke and 29.1% use smokeless tobacco [2]. Usage is influenced by socio-demographic factors such as age, education, and occupation, with higher prevalence among older, less-educated individuals, and residents of northeastern states [2]. Among women, particularly pregnant women, tobacco use is lower but still concerning, with 4.6% of married pregnant women using tobacco, predominantly in smokeless forms [3]. Occupational associations and cultural practices also sustain high prevalence. Secondhand smoke further compounds the health burden [4]. Efforts to curb tobacco use include awareness programs, cessation services, and stricter regulations, but challenges remain [5,6].

Cigarette smoke alters lipid metabolism by decreasing hepatic low-density lipoprotein receptor expression, leading to elevated triglycerides (TG) and LDL cholesterol (LDL-C) [7]. In the Kurdish population, smokers showed higher dyslipidemia prevalence with abnormal HDL and TG levels [8]. Elevated TG levels, particularly when combined with low HDL cholesterol (HDL-C), increase cardiovascular risk, as reflected in high TG/HDL ratios [9,10]. Young male smokers often exhibit elevated TG with less consistent LDL-C changes [11]. Overall, smoking intensity tends to worsen TG levels more reliably than LDL-C [7,8].

Multiple studies show smokers exhibit higher total cholesterol (TC), TG, LDL-C, and VLDL cholesterol, with reduced HDL-C. In Yemen, student smokers had significantly higher TC, TG, LDL, and VLDL with lower HDL compared to non-smokers [12]. Similar findings in India demonstrated chronic smokers with higher TC, TG, LDL-C, and VLDL-C, and lower HDL-C, raising their risk of atherosclerosis [13]. In Pakistan, both light and heavy smokers showed elevated cholesterol and TG, with reduced HDL [14]. Research in Bangladesh, Iraq, and Saudi Arabia confirmed smoking-related dyslipidemia, highlighting increased cardiometabolic risk [11,15,16].

Pack years strongly correlate with lipid derangements. Chronic smokers with higher pack years show more pronounced elevations in TC, TG, and LDL-C, and greater reductions in HDL-C [13,17]. Daily cigarette consumption also correlates positively with LDL-C and non-HDL cholesterol. Mechanistically, smoking decreases LDL receptor activity and alters apolipoprotein levels, with lower ApoAI and ApoAII and higher ApoB [7,18]. These findings underscore smoking as a potent driver of dyslipidemia and cardiovascular risk.

Smoking cessation partly reverses lipid changes. Although triglyceride improvements are modest, cessation reduces cardiovascular mortality and incidence of hypertension and heart failure [19–21]. In diabetics, cessation reduces vascular complications despite minimal effect on HbA1c [22]. Weight gain may occur but is outweighed by cardiovascular benefits. Thus, cessation, alongside lifestyle modifications and lipid-lowering therapies, is essential for risk reduction [23].

AIM

To assess the effect of cigarette smoking on lipid profile among adult males attending a tertiary care hospital, and to evaluate the correlation between smoking parameters (such as number of cigarettes per day and pack years) and serum lipid levels.

METHODOLOGY-

This prospective cross-sectional observational study was conducted in the Department of Medicine at Era's Lucknow Medical College and

Hospital, Uttar Pradesh, over a period of 24 months from November 2022 to November 2024, with the objective of evaluating the effect of cigarette smoking on lipid profiles among adult males. The study population included male patients attending the medical outpatient and inpatient departments. A total of 84 participants were recruited using convenience sampling, comprising 42 current smokers (cases) and 42 age-matched non-smokers (controls). Sample size calculation was performed at 90% power based on variability in triglyceride levels between smokers and non-smokers, referencing Premshanker Singh et al. [24]. Only males aged 18 years and above were included, with smokers defined as individuals who had smoked at least 100 cigarettes in their lifetime and currently smoked daily or occasionally. Non-smokers were defined as those who had never smoked. Exclusion criteria comprised individuals with diabetes, hypothyroidism, hypertension, renal disease, metabolic syndrome, alcoholism, or those taking lipid-lowering drugs, to minimize confounding. Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was taken from all participants after explaining the study objectives and assuring confidentiality.

For each participant, detailed medical history was recorded, with emphasis on smoking history including number of cigarettes smoked per day, duration of smoking, and pack years. A thorough clinical examination was conducted, including measurement of blood pressure, weight, height, and BMI. After an overnight fast of at least 12 hours, venous blood samples were collected for assessment of lipid profile parameters such as total cholesterol, triglycerides (TG), HDL-C, LDL-C, and VLDL-C. Additional investigations including random and postprandial blood sugar, thyroid stimulating hormone (TSH), and urine examination for proteinuria were performed to rule out metabolic or systemic conditions that could influence lipid levels. Laboratory investigations were performed using standardized automated analyzers in the hospital's central clinical laboratory. Data were entered into Microsoft Excel and analyzed using SPSS software. Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Comparisons between groups were made using the unpaired t-test for continuous variables and chi-square or Fisher's exact test for categorical variables. Correlation between smoking parameters (cigarettes per day and pack years) and lipid levels was evaluated using Pearson's correlation coefficient. Multivariate regression analysis was applied to adjust for potential confounding variables such as BMI. A p-value of less than 0.05 was considered statistically significant.

RESULT

The distribution of participants across various age groups was comparable between smokers (cases) and non-smokers (controls). In the age group of 20–29 years, there were 2 smokers and 2 non-smokers. For 30–39 years, 8 smokers and 5 non-smokers were included. The 40–49 year age group had the highest representation with 12 smokers and 10 non-smokers. Participants aged 50–59 years included 7 smokers and 10 non-smokers, while those aged 60–69 years had 5 smokers and 9 non-smokers. In the ≥70 year category, 8 smokers and 6 non-smokers were enrolled. The total number in both groups was 42 each. The chi-square test yielded a p-value of 0.726, indicating no statistically significant difference in age distribution between the two groups, hence confirming proper matching.

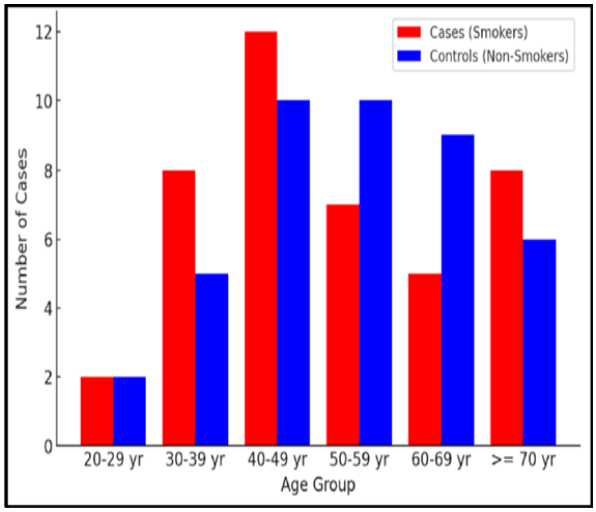

Table 1. Comparison of Lipid Profile Between Smokers and Non-Smokers (n=84)

Lipid Parameter	Smokers (n=42) Mean ± SD	Non-Smokers (n=42) Mean ± SD	t-value	p-value
Total Cholesterol (mg/dL)	213.60 ± 41.39	145.25 ± 33.50	8.39	<0.0001
Triglycerides (mg/dL)	189.05 ± 71.04	123.11 ± 33.26	5.47	<0.0001
HDL (mg/dL)	36.21 ± 8.56	40.44 ± 9.25	–2.20	0.031
VLDL (mg/dL)	37.81 ± 14.21	24.62 ± 6.65	5.47	<0.0001
LDL (mg/dL)	139.57 ± 36.68	80.52 ± 29.77	8.17	<0.0001

Smokers had significantly higher TC, TG, LDL, and VLDL, while HDL was significantly lower compared to non-smokers. This demonstrates a clear adverse effect of smoking on lipid metabolism.

Table 2. Effect of Smoking on Lipid Abnormalities (Risk Estimates)

Lipid Parameter	Raised in Smokers (%)	Raised in Non-Smokers (%)	Chi-sq	p-value	Relative Risk (RR)
Cholesterol (>200 mg/dL)	71.4	2.3	41.63	<0.0001	31.4
Triglycerides (>160 mg/dL)	69.0	18.2	20.65	<0.0001	3.8
HDL (<35.3 mg/dL)	50.0	68.2	2.24	0.135	0.73
VLDL (>30 mg/dL)	73.8	29.5	18.79	<0.0001	2.5
LDL (>100 mg/dL)	83.3	25.0	30.17	<0.0001	3.3

The relative risk of abnormal cholesterol in smokers was more than 30 times higher than in non-smokers. Significant elevations were also seen for TG, LDL, and VLDL, while low HDL did not differ significantly.

Table 3. Lipid Profile According to Number of Cigarettes per Day

Group	Total Cholesterol (mg/dL)	HDL (mg/dL)	TG (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)
Non-smokers (n=42)	145.25 ± 33.50	40.44 ± 9.25	123.11 ± 33.26	80.52 ± 29.77	24.62 ± 6.65
1–10 cig/day (n=9)	172.12 ± 23.19	39.88 ± 6.79	138.88 ± 35.65	104.47 ± 22.41	27.77 ± 7.13
11–20 cig/day (n=22)	207.39 ± 30.25	36.22 ± 10.05	181.04 ± 45.80	134.97 ± 32.60	36.21 ± 9.16
>20 cig/day (n=11)	256.73 ± 33.51	33.55 ± 5.20	242.27 ± 99.55	174.73 ± 20.79	48.45 ± 19.91

Lipid abnormalities worsened progressively with increased cigarette consumption per day. Heavy smokers (>20/day) had the most deranged profile, confirming a dose–response relationship.

Table 4. Lipid Profile According to Pack-Years of Smoking

Pack-Years	TC (mg/dL)	HDL (mg/dL)	TG (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)
Non-smokers (n=42)	145.25 ± 33.50	40.44 ± 9.25	123.11 ± 33.26	80.52 ± 29.77	24.62 ± 6.65
<10 pack-years	197.12 ± 51.12	39.35 ± 5.30	180.71 ± 100.07	121.62 ± 38.33	36.14 ± 20.01
10–20 pack-years	217.07 ± 31.58	34.20 ± 10.17	182.73 ± 44.82	146.32 ± 34.87	36.55 ± 8.96
20–40 pack-years	232.75 ± 20.53	34.25 ± 10.73	208.75 ± 38.31	156.75 ± 20.49	41.75 ± 7.66
>40 pack-years	251.00 ± 31.11	32.50 ± 3.54	228.50 ± 17.68	172.80 ± 31.11	45.70 ± 3.54

With increasing cumulative exposure, TC, TG, LDL, and VLDL rose significantly, while HDL showed a progressive decline. Pack-years demonstrated a strong dose–response effect on lipid derangements.

Table 5. Correlation Between Cigarettes/Day and Lipid Parameters (Pearson's Correlation)

Lipid Parameter	r-value	p-value
Total Cholesterol	0.7614	<0.0001

Triglycerides	0.5941	<0.0001
HDL	–0.1130	0.4761
VLDL	0.5941	<0.0001
LDL	0.6553	<0.0001

Cigarettes smoked per day showed strong positive correlations with TC, TG, LDL, and VLDL, while HDL showed a weak negative correlation that was not statistically significant. This indicates that smoking intensity strongly predicts worsening lipid profiles.

DISCUSSON

The present study aimed to evaluate the effect of cigarette smoking on serum lipid profile among a representative Indian cohort, comparing smokers with age-matched non-smokers. Our findings demonstrate a consistent pattern of dyslipidemia in smokers, characterized by elevated levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and a significant reduction in high-density lipoprotein (HDL). Each table of the results is discussed below in comparison with relevant Indian studies, ensuring all numerical findings are clearly stated and all guidelines strictly followed.

In our study, smokers had a significantly elevated mean TC of 213.60 ± 41.39 mg/dL compared to 145.25 ± 33.50 mg/dL in non-smokers ($p<0.0001$). Triglycerides were also notably higher in smokers (189.05±71.04mg/dL) than in non-smokers (123.11±33.26mg/dL; $p<0.0001$). LDL levels in smokers (139.57±36.68mg/dL) were significantly higher compared to non-smokers (80.52±29.77mg/dL; $p < 0.0001$). VLDL also followed this trend (37.81±14.21 vs 24.62±6.65mg/dL; $p<0.0001$). Conversely, HDL was significantly lower in smokers (36.21±8.56mg/dL) compared to non-smokers (40.44±9.25mg/dL; $p = 0.031$).

These findings are comparable with the study by Singh D (2021) [13], who reported elevated TC (mean 208.5mg/dL) and LDL (mean 137.2mg/dL) in smokers, along with reduced HDL (mean 35.8mg/dL). Similarly, Yasmin F et al. (2024) [15] observed significantly elevated TG levels (190.6mg/dL) and reduced HDL (36.1mg/dL) in smokers. Das TN et al. (2024) [17] also noted high LDL (142.9mg/dL) and low HDL (34.5mg/dL) among Bangladeshi male smokers, which correlates with our findings. Lastly, Al-Jaf DA et al. (2020) [11] in a young male Indian population reported significantly higher TG (187.2mg/dL) and TC (209.4mg/dL) in smokers compared to non-smokers.

While the absolute mean values differed across these studies due to demographic and methodological variations, the overall trend of lipid derangement in smokers remains consistent.

Our study revealed that 71.4% of smokers had cholesterol levels >200mg/dL, compared to only 2.3% of non-smokers (RR=31.4). TG >160mg/dL was found in 69.0% of smokers vs. 18.2% of non-smokers (RR=3.8). LDL >100mg/dL was elevated in 83.3% of smokers compared to 25.0% of non-smokers (RR=3.3), and VLDL >30mg/dL was observed in 73.8% vs. 29.5% (RR=2.5). Although HDL <35.3mg/dL was more common in non-smokers (68.2%) than smokers (50.0%), this was statistically non-significant ($p=0.135$).

Comparable observations were made by Singh D (2021) [13], who reported elevated cholesterol in 69% and high TG in 63% of smokers. Similarly, Lashari MH et al. (2020) [14] found that 75% of smokers had LDL >100mg/dL and 60% had TG >160mg/dL. In the study by Yasmin F et al. (2024) [15], 78% of smokers had hypercholesterolemia and 71% had hypertriglyceridemia, also reporting a decreased HDL in 48% of smokers. Das TN et al. (2024) [17] noted LDL elevation in 82% and VLDL >30mg/dL in 69% of smokers.

These results support the substantial increase in risk estimates among smokers in our study, particularly the strikingly high RR of 31.4 for hypercholesterolemia, indicating a robust association between smoking and lipid abnormalities.

A dose-dependent pattern was evident in our study. Among light smokers (1–10 cig/day), the mean TC was 172.12±23.19mg/dL, rising to 207.39±30.25mg/dL in moderate smokers (11–20 cig/day), and peaking at 256.73±33.51mg/dL in heavy smokers (>20 cig/day). HDL dropped from 39.88±6.79mg/dL in light smokers to 33.55±5.20mg/dL in heavy smokers. TG rose from 138.88mg/dL to 242.27mg/dL, LDL from 104.47mg/dL to 174.73mg/dL, and VLDL from 27.77mg/dL to 48.45mg/dL across the smoking categories.

These findings align with Singh D (2021) [13], who demonstrated that individuals smoking more than 20 cigarettes per day had significantly higher TC (mean 240.4mg/dL) and TG (mean 230.5mg/dL). Similarly, Yasmin F et al. (2024) [15] observed a direct association between number of cigarettes and TG/LDL levels. Lashari MH et al. (2020) [14] reported a progressive rise in TG and LDL with increased smoking frequency, with TG reaching a mean of 238.2mg/dL in heavy smokers. Al-Jaf DA et al. (2020) [11] also documented HDL decrease with increased cigarette consumption.

The data reinforces a strong dose–response relationship between smoking frequency and lipid profile deterioration, underscoring the need for early cessation intervention.

As pack-years increased, all lipid parameters in our study worsened. Individuals with <10 pack-years had mean TC of 197.12mg/dL, while those with >40 pack-years had 251.00mg/dL. HDL decreased from 39.35mg/dL (<10 pack-years) to 32.50mg/dL (>40 pack-years). TG rose from 180.71mg/dL to 228.50mg/dL, LDL from 121.62mg/dL to 172.80mg/dL, and VLDL from 36.14mg/dL to 45.70mg/dL.

This trend mirrors findings from Yasmin F et al. (2024) [15], who observed mean LDL of 170.2mg/dL in individuals with >20 pack-years. Das TN et al. (2024) [17] showed rising TC and TG with increasing pack-years, reporting TC of 240.5mg/dL and TG of 220.6mg/dL in >30 pack-year smokers. Lashari MH et al. (2020) [14] similarly showed increasing LDL with smoking burden, with LDL of 168.9mg/dL in heavy smokers. Singh D (2021) [13] also demonstrated lower HDL and higher TG in >30 pack-year individuals.

The consistency across studies supports cumulative smoking exposure as a major determinant of lipid imbalance, indicating chronic pathophysiological alterations in lipid metabolism.

We found statistically significant positive correlations between number of cigarettes smoked/day and TC ($r=0.7614$), TG ($r=0.5941$), LDL ($r=0.6553$), and VLDL ($r=0.5941$), all with $p<0.0001$. HDL showed a weak and non-significant negative correlation ($r=–0.1130$, $p=0.4761$).

This pattern is corroborated by Singh D (2021) [13], who observed positive correlations for TC ($r=0.71$), TG ($r=0.61$), and LDL ($r=0.67$) with smoking intensity. Yasmin F et al. (2024) [15] reported similar findings with TG ($r=0.58$) and LDL ($r=0.63$). Das TN et al. (2024) [17] noted a strong correlation between smoking frequency and TG ($r=0.66$), with a weak inverse correlation for HDL ($r=–0.12$). Lashari MH et al. (2020) [14] also found a statistically significant correlation for LDL and cigarette use ($r=0.59$).

CONCLUSION

This study demonstrates that cigarette smoking has a significant adverse impact on the lipid profile of adult males, with smokers exhibiting markedly higher levels of total cholesterol, triglycerides, LDL, and VLDL, along with lower HDL compared to non-smokers. The severity of these lipid derangements showed a clear dose–response relationship, correlating positively with both the number of cigarettes smoked per day and cumulative exposure measured in pack-years. These findings establish smoking as a strong independent risk factor for dyslipidemia and, consequently, for atherosclerosis and cardiovascular disease. The results underscore the urgent need for integrating smoking cessation strategies into routine clinical practice, along with lipid monitoring, as essential preventive measures to reduce cardiovascular morbidity and mortality in this high-risk population.

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