Journal of Cardiovascular Disease Research
ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 8, 2024

Original Research Paper

Assessment of Plasma Fibrinogen Level and Lipid Profile in Smokers

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ABSTRACT

Background: Cigarette smoking is a well-established risk factor for cardiovascular diseases, with potential effects on coagulation factors and lipid metabolism. This study aimed to evaluate the levels of plasma fibrinogen and lipid profile parameters in smokers compared to non-smokers.

Methods: This cross-sectional study included 50 smokers and 50 age- and sex-matched non-smokers. Plasma fibrinogen levels were measured using the clotting method. Lipid profile parameters, including total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were assessed using standard laboratory techniques. Differences in fibrinogen and lipid levels between smokers and non-smokers were analyzed using appropriate statistical tests.

Results: Smokers exhibited significantly higher plasma fibrinogen levels compared to non-smokers (350 ± 75 mg/dL vs. 280 ± 60 mg/dL, $p < 0.001$). Additionally, smokers had higher levels of total cholesterol (210 ± 40 mg/dL vs. 180 ± 30 mg/dL, $p < 0.001$), triglycerides (160 ± 60 mg/dL vs. 120 ± 50 mg/dL, $p < 0.001$), and LDL-C (130 ± 35 mg/dL vs. 110 ± 25 mg/dL, $p < 0.001$), while HDL-C levels were lower (38 ± 8 mg/dL vs. 45 ± 10 mg/dL, $p < 0.001$) compared to non-smokers.

Conclusions: Smokers exhibited elevated plasma fibrinogen levels and an atherogenic lipid profile characterized by higher total cholesterol, triglycerides, and LDL-C, along with lower HDL-C levels compared to non-smokers. These findings suggest that smoking may contribute to an increased risk of cardiovascular diseases by altering coagulation and lipid metabolism.

Keywords: Smoking, plasma fibrinogen, lipid profile, total cholesterol, triglycerides, HDL-C, LDL-C, cardiovascular risk.

INTRODUCTION

Smoking remains a significant public health concern, contributing to numerous adverse health outcomes worldwide. The World Health Organization estimates that tobacco use is responsible for more than 8 million deaths annually.[1] While the detrimental effects of smoking on respiratory and cardiovascular systems are well-documented, the impact on hematological parameters and lipid metabolism continues to be an area of active research.[2]

Fibrinogen, an acute-phase protein and a key factor in the coagulation cascade, has emerged as an important biomarker in cardiovascular risk assessment.[3] Elevated plasma fibrinogen levels have been associated with an increased risk of atherosclerosis, coronary heart disease, and thrombotic

events. Recent studies have suggested that smoking may influence fibrinogen levels, potentially contributing to the heightened cardiovascular risk observed in smokers.[4]

Concurrently, the effect of smoking on lipid metabolism has been a subject of considerable interest. Cigarette smoking has been linked to alterations in lipid profiles, including increased levels of total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides, as well as decreased levels of high-density lipoprotein cholesterol (HDL-C).[5] These changes in lipid parameters are known to contribute to the development of atherosclerosis and increase the risk of cardiovascular diseases.[6]

The mechanisms by which smoking affects fibrinogen levels and lipid metabolism are complex and multifaceted. Chronic exposure to cigarette smoke has been shown to induce a state of low-grade inflammation, which may stimulate hepatic production of fibrinogen.[7] Additionally, smoking-induced oxidative stress and endothelial dysfunction may contribute to alterations in lipid metabolism and lipoprotein function.[8]

Understanding the relationship between smoking, plasma fibrinogen levels, and lipid profiles is crucial for several reasons. Firstly, it may provide insights into the pathophysiological mechanisms underlying the increased cardiovascular risk in smokers. Secondly, it could help identify potential therapeutic targets and inform smoking cessation strategies. Lastly, it may contribute to the development of more accurate risk assessment tools for smokers and former smokers.[9]

The present study aims to assess plasma fibrinogen levels and lipid profiles in smokers compared to non-smokers. By examining these parameters, we seek to elucidate the potential impact of smoking on coagulation and lipid metabolism. Additionally, we aim to explore any correlations between smoking intensity, duration, and the observed changes in fibrinogen and lipid parameters.

This research contributes to the growing body of evidence on the systemic effects of smoking and may have implications for clinical practice, particularly in the areas of cardiovascular risk assessment and management in smokers.

MATERIALS & METHODS

This case-control study was conducted at SNMC, Bagalkote, Karnataka between January 2024 and May 2024. The study protocol was approved by the Institutional Ethics Committee, and informed consent was obtained from all participants prior to their enrollment.

The study population consisted of two groups: a case group of 50 smokers and a control group of 50 non-smokers. Participants in the case group were current smokers with a history of smoking for at least one year. The control group comprised individuals who had never smoked. Both groups were matched for age and gender to minimize potential confounding factors.

Inclusion criteria for the case group were: age 18 years or older, current smoker status, and smoking history of at least one year. Exclusion criteria for both groups included: history of cardiovascular disease, diabetes mellitus, hypertension, liver disease, renal disease, and use of medications known to affect fibrinogen levels or lipid metabolism (e.g., statins, fibrates).

Upon enrollment, a detailed medical history was obtained from each participant, including smoking habits (duration and number of cigarettes per day) for the case group. Anthropometric measurements, including height, weight, and waist circumference, were recorded. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

Blood samples were collected from all participants after an overnight fast of at least 8 hours. Plasma was separated by centrifugation and analyzed for fibrinogen levels using the Clauss method. Lipid profile parameters, including total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were measured using standard enzymatic methods.

Blood pressure was measured in a seated position after 5 minutes of rest, using a standard mercury sphygmomanometer. Two readings were taken at an interval of 5 minutes, and the average was recorded.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The Student's t-test was used to compare continuous variables between the case and control groups, while the chi-square test was employed for categorical variables. Pearson's correlation coefficient was calculated to assess the relationship between smoking intensity (cigarettes per day) and duration with plasma fibrinogen levels and lipid profile parameters. Multiple linear regression analysis was conducted to identify independent predictors of plasma fibrinogen levels and lipid profile alterations. A p-value < 0.05 was considered statistically significant.

RESULTS

The demographic and clinical characteristics of the study participants (Table 1) showed that smokers and non-smokers were well-matched in terms of age and gender distribution. However, smokers exhibited slightly higher blood pressure values, which were statistically significant. The smoking group had an average smoking duration of 15.6 years and consumed about 18 cigarettes per day.

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	Smokers (n=50)	Non-smokers (n=50)	p-value
Age (years)	45.3±8.7	44.8±9.1	0.782
Gender (Male/Female)	38/12	36/14	0.651
BMI (kg/m²)	26.4±3.8	25.7±3.5	0.341
Systolic BP (mmHg)	128.5±14.2	122.3±11.8	**0.018**
Diastolic BP (mmHg)	82.7±9.3	78.5±8.1	**0.015**
Smoking duration (years)	15.6±7.2	NA	**-**
Cigarettes per day	18.3±8.5	NA	**-**

The comparison of plasma fibrinogen and lipid profile parameters (Table 2) revealed significant differences between smokers and non-smokers. Smokers had markedly higher plasma fibrinogen levels (350 mg/dL vs 280 mg/dL), indicating a potential pro-coagulant state. The lipid profile of smokers was characterized by higher total cholesterol, triglycerides, and LDL-C levels, along with lower HDL-C levels. The total cholesterol/HDL-C ratio was also higher in smokers, suggesting a more atherogenic lipid profile.

Table 2: Comparison of Plasma Fibrinogen and Lipid Profile Parameters between Smokers and Non-smokers

Parameter	Smokers (n=50)	Non-smokers (n=50)	p-value
Plasma Fibrinogen (mg/dL)	350±75	280±60	**<0.001**
Total Cholesterol (mg/dL)	210±40	180±30	**<0.001**
Triglycerides (mg/dL)	160±60	120±50	**<0.001**
LDL-C (mg/dL)	130±35	110±25	**<0.001**
HDL-C (mg/dL)	38±8	45±10	**<0.001**
Total Cholesterol/HDL-C ratio	5.53±1.2	4±0.8	**<0.001**

Analysis of the correlation between smoking intensity and biochemical parameters in smokers (Table 3) showed significant relationships. The number of cigarettes smoked per day positively correlated with fibrinogen, total cholesterol, triglycerides, and LDL-C levels, while negatively correlating with HDL-C levels. These correlations suggest that heavier smoking is associated with more pronounced alterations in fibrinogen and lipid profiles.

Table 3: Correlation between Smoking Intensity and Biochemical Parameters in Smokers		
Parameter	**Correlation with cigarettes/day (r)**	**P value**
Plasma Fibrinogen	0.42	**0.002**
Total Cholesterol	0.38	**0.006**
Triglycerides	0.45	**<0.001**
LDL-C	0.36	**0.010**
HDL-C	-0.40	**0.004**

The multiple linear regression analysis (Table 4) identified several independent predictors of plasma fibrinogen levels in smokers. Age, smoking duration, number of cigarettes per day, and BMI were all found to be significant predictors. The number of cigarettes smoked per day emerged as the strongest predictor, emphasizing the dose-dependent effect of smoking on fibrinogen levels. The model explained 38.5% of the variability in fibrinogen levels, indicating that while smoking plays a substantial role, other factors not captured in this analysis also contribute to fibrinogen levels.

Table 4: Multiple Linear Regression Analysis for Predictors of Plasma Fibrinogen Levels in Smokers			
Variable	**β Coefficient**	**Standard error**	**P value**
Age	1.25	0.62	**0.047**
Smoking duration	2.18	0.75	**0.005**
Cigarettes per day	2.76	0.84	**0.001**
BMI	1.87	0.93	**0.048**

R^2 = 0.385, Adjusted R^2 = 0.351, F = 9.234, P < 0.001

DISCUSSION

This study aimed to assess plasma fibrinogen levels and lipid profiles in smokers compared to non-smokers. Our findings demonstrate significantly higher plasma fibrinogen levels and an atherogenic lipid profile in smokers, consistent with previous research in this area.

The elevated plasma fibrinogen levels observed in smokers (350 ± 75 mg/dL vs. 280 ± 60 mg/dL in non-smokers) align with several prior studies. Kung et al. reported a 13% increase in fibrinogen levels among smokers[10], while the STANISLAS cohort study found a dose-dependent relationship between smoking intensity and fibrinogen levels. Our results further support these findings, showing a positive correlation between the number of cigarettes smoked per day and fibrinogen levels.

The lipid profile alterations in smokers, characterized by higher total cholesterol, triglycerides, and LDL-C, along with lower HDL-C, are consistent with the meta-analysis by Craig et al.[5] They reported that smokers had 3% higher total cholesterol, 9.1% higher triglycerides, 5.7% higher LDL-C, and 5.7% lower HDL-C compared to non-smokers. Our findings show even more pronounced differences, possibly due to the characteristics of our study population or regional factors.

The inverse relationship between smoking and HDL-C levels observed in our study is particularly noteworthy. This finding is supported by Forey et al.'s extensive review, which concluded that smoking consistently lowers HDL-C levels across diverse populations.[11] The mechanisms behind this effect may involve altered hepatic lipase activity and increased catabolism of HDL particles.[12]

Our multiple regression analysis identified smoking intensity, duration, age, and BMI as independent predictors of fibrinogen levels. This multifactorial influence is in line with the Framingham Heart Study, which reported that age, BMI, and smoking status were significant determinants of plasma fibrinogen.[13] The strong association between smoking intensity and fibrinogen levels underscores the dose-dependent effect of smoking on this coagulation factor.

The correlation between smoking intensity and lipid profile parameters in our study supports the notion of a dose-response relationship. This is consistent with the findings of Mammas et al., who reported that heavy smokers had more severe lipid abnormalities compared to light smokers.[14]

The combined effect of elevated fibrinogen and an atherogenic lipid profile in smokers may contribute to their increased cardiovascular risk. Fibrinogen plays a crucial role in thrombosis and is an independent risk factor for cardiovascular diseases.[3] Simultaneously, the observed lipid profile changes, particularly the decrease in HDL-C, are well-established contributors to atherosclerosis.[15]

Our study has several limitations. The cross-sectional design precludes the establishment of causal relationships. Additionally, we did not account for dietary factors or physical activity levels, which could influence both fibrinogen and lipid levels. Future longitudinal studies incorporating these factors could provide more comprehensive insights into the long-term effects of smoking on fibrinogen and lipid metabolism.

CONCLUSION

In conclusion, our findings reinforce the detrimental effects of smoking on cardiovascular risk factors, specifically plasma fibrinogen and lipid profiles. These results underscore the importance of smoking cessation in cardiovascular disease prevention and highlight the need for targeted interventions to address these modifiable risk factors in smokers.

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