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ASSESSMENT OF LIPID PROFILE AND FIBRINOGEN LEVEL IN SMOKERS

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ABSTRACT **Background:** Cigarette smoking is a proven risk factor for cardiovascular disease and has potential effects on blood clotting factors and lipid metabolism. This study aimed to evaluate plasma fibrinogen levels and lipid parameters in smokers compared to non-smokers. **Methods:** In this cross-sectional study, a total of 50 smokers and 50 age- and sex-matched non-smokers were included for analysis. The levels of plasma fibrinogen were determined using the clotting method. Additionally, the lipid profile parameters such as total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were assessed through standard laboratory techniques. Statistical tests were employed to analyze the differences in fibrinogen and lipid levels between the smokers and non-smokers. **Results:** Smokers had much higher levels of plasma fibrinogen than non-smokers, with levels of 350 ± 75 mg/dL for smokers and 280 ± 60 mg/dL for non-smokers, which is a significant difference ($p < 0.001$). Smokers had higher amounts of total cholesterol (210 ± 40 mg/dL compared to 180 ± 30 mg/dL, $p < 0.001$), triglycerides (160 ± 60 mg/dL versus 120 ± 50 mg/dL, $p < 0.001$), and LDL-C (130 ± 35 mg/dL against 110 ± 25 mg/dL, $p < 0.001$). At the same time, their HDL-C levels were lower (38 ± 8 mg/dL compared to 45 ± 10 mg/dL, $p < 0.001$) when looking at non-smokers. **Conclusions:** Smokers exhibited elevated plasma fibrinogen levels and a lipid profile predisposing them to atherosclerosis, characterized by higher levels of total cholesterol, triglycerides, and LDL-C (bad cholesterol), and lower levels of HDL-C (good cholesterol) compared to non-smokers. These findings suggest that smoking may contribute to an increased risk of cardiovascular disease by affecting blood clotting and lipid metabolism.

KEYWORDS : Smoking, Plasma Fibrinogen, Lipid Profile, Cardiovascular Risk.

INTRODUCTION

Smoking is still a major public health issue, leading to many negative health effects around the world. The World Health Organization estimates that tobacco use causes over 8 million deaths each year.[1] While the harmful effects of smoking on the lungs and heart are well-known, researchers are still exploring how it affects blood health and fat metabolism. Fibrinogen, which is a protein that helps with blood clotting, has become an important marker for assessing heart health risks.[2] Higher levels of fibrinogen in the blood have been linked to a greater chance of developing atherosclerosis, coronary heart disease, and blood clots. Recent studies indicate that smoking might affect fibrinogen levels, which could increase the risk of heart problems seen in smokers.[3] At the same time, researchers have shown a strong interest in how smoking impacts the way fats are processed in the body.[4] Cigarette smoking is connected to changes in fat levels in the body. It can lead to higher amounts of total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides, while also causing lower levels of high-density lipoprotein cholesterol (HDL-C).[5] These changes in fat levels can help cause atherosclerosis and raise the chances of heart disease.[6] The ways that smoking impacts fibrinogen levels and how fats are processed in the body are complicated and involve many factors. Long-term exposure to cigarette smoke has been found to cause a mild form of inflammation.[7] which can lead to the liver producing more fibrinogen. Moreover, the stress caused by smoking and problems with blood vessel function can affect how fats are processed in the body and how lipoproteins work.[8] It is important to understand how smoking relates to plasma fibrinogen levels and lipid profiles for several reasons. First of all, it could give us a better understanding of the biological reasons behind the higher risk of heart problems in people who smoke. Secondly, it might help find possible treatment options and guide strategies for quitting smoking. Finally, it could help create better tools for evaluating risks for smokers and those who use to smoke.[9] The current study seeks to examine the levels of plasma fibrinogen and lipid profiles in smokers in comparison to non-smokers. By looking at these factors, we aim to explain how smoking might affect blood clotting and fat processing in the body. We also want to look into how smoking more or for a longer time might be related to the changes we see in fibrinogen and lipid levels. This study adds to the increasing evidence about how smoking affects the body as a whole and could impact how doctors practice, especially in evaluating and managing heart-related risks in smokers.

MATERIALS & METHOD

This study compared two groups of people at SCPM Hospital Gonda, taking place from January 2026 to May 2026. The study plan was approved by the Institutional Ethics Committee, and all participants gave their informed consent before they joined the study. The study included two groups of people: one group had 50 smokers, and the other group had 50 non-smokers. The people in the case group were current smokers who had been smoking for at least one year. The control group included people who had never smoked. Both groups were similar in age and gender to reduce any possible confusing factors. The requirements for being in the case group were: being 18 years old or older, currently smoking, and having a smoking history of at least one year. Both groups had certain reasons for not being included: a past of heart disease, diabetes, high blood pressure, liver problems, kidney issues, and taking medicines that are known to change fibrinogen levels or affect how fats are processed in the body (like Statins and fibrates). When they signed up, we collected a thorough medical history from each person, which included their smoking habits, such as how long they had been smoking and how many cigarettes they smoked each day, specifically for the group that was being studied. We recorded measurements related to the body, such as height, weight, and waist size. Body mass index (BMI) is found by taking a person's weight in kilograms and dividing it by their height in meters squared. Blood samples were taken from all participants after they fasted for at least 8 hours overnight. Plasma was separated using a centrifuge and checked for fibrinogen levels with the Clauss method. The lipid profile was checked by measuring total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) using regular enzyme methods. Blood pressure was taken while sitting down after resting for 5 minutes, using a regular mercury sphygmomanometer. Two measurements were taken 5 minutes apart, and the average of those readings was noted.

Statistical Analysis

The statistical analysis was carried out using SPSS version 25.0.

Continuous variables were represented as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. To compare continuous variables between the case and control groups, the Student's t-test was utilized, while the chi-square test was employed for categorical variables. Pearson's correlation coefficient was computed to evaluate the relationship between smoking intensity (cigarettes per day) and duration with plasma fibrinogen levels and lipid profile parameters. Multiple linear regression analyses were performed to identify independent predictors of plasma fibrinogen levels and lipid profile alterations. A p-value less than 0.05 was considered statistically significant.

RESULTS

The demographic and clinical features of the study participants [Table No- 1] indicated that smokers and non-smokers had similar age and gender distributions. However, smokers showed a bit higher blood pressure levels, and this difference was statistically important. The group of smokers had an average smoking habit that lasted for 15.6 years and they smoked around 18 cigarettes each day.

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	-

Table No. 1: Demographic and Clinical Characteristics of Study Participants.

The comparison of plasma fibrinogen and lipid profile measurements [Table No. 2] showed important differences between smokers and non-smokers. Smokers showed much higher levels of plasma fibrinogen (350 mg/dL compared to 280 mg/dL), which suggests they may be in a state that promotes blood clotting. Smokers had a lipid profile that showed higher levels of total cholesterol, triglycerides, and LDL-C, while their HDL-C levels were lower. The total cholesterol to HDL-C ratio was higher in smokers, indicating they have a lipid profile that is more likely to cause artery problems.

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

Table No. 2: Comparison of Plasma Fibrinogen and Lipid Profile Parameters between Smokers and Non-smokers.

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. Freeman DJ et al. [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. Kannel WB et al. [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

The findings of our study serve to emphasize the harmful impact of smoking on cardiovascular risk factors, particularly plasma fibrinogen and lipid profiles. These results underscore the crucial role of smoking cessation in preventing cardiovascular disease and emphasize the necessity for tailored interventions to address these modifiable risk factors among smokers.

REFERENCES

1. World Health Organization. Tobacco [Internet]. 2021 [cited 2024 Jun 29]. Available from:
2. National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2014.Available from
3. Fibrinogen Studies Collaboration; Danesh J, Lewington S, Thompson SG, Lowe GD, Collins R, Kostis JB, et.al. Plasma fibrinogen level and the risk of major cardiovascular diseases and nonvascular mortality: an individual participant meta-analysis. JAMA. 2005 Oct 12; 294(14):1799-809.
4. Bazzano LA, He J, Muntner P, Vupputuri S, Whelton PK. Relationship between cigarette smoking and novel risk factors for cardiovascular disease in the United States.Ann Intern Med. 2003; 138(11):891-897
5. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. BMJ. 1989 Mar 25; 298(6676):784-8
6. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. Am Heart J. 2011 Jan; 161(1):145-51.
7. Yanbaeva DG, Dentener MA, Creutzberg EC, Wesseling G, Wouters EF. Systemic effects of smoking. Chest. 2007; 131(5):1557-1566.
8. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update.J Am Coll Cardiol. 2004;43(10):1731-1737.
9. Wannamethee SG, Lowe GD, Shaper AG, Rumley A, Lennon L, Whincup PH. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and hemostatic and inflammatory markers for cardiovascular disease.Eur Heart J. 2005; 26(17):1765-1773.
10. Kung CM, Wang HL, Tseng ZL. Cigarette smoking exacerbates health problems in young men.Clin Invest Med. 2008; 31(3):E138-E149.
11. Forey BA, Fry JS, Lee PN, Thornton AJ, Coombs KJ. The effect of quitting smoking on HDL cholesterol - review based on within-subject changes. Biomark Res. 2013 Sep 13; 1(1):26.
12. Freeman DJ, Caslake MJ, Griffin BA, et al. The effect of smoking on post-heparin lipoprotein and hepatic lipase, cholesteryl ester transfer protein, and lecithin: cholesterol acyl transferees activities in human plasma.Eur J Clin Invest. 1998; 28(7):584-591. 13. Kannel WB, Wolf PA, Castelli WP, D'Agostino RB. Fibrinogen and risk of cardiovascular disease. The Framingham Study.JAMA. 1987; 258(9):1183-1186.
14. Mammas IN, Bertsias GK, Linardakis M, Tzanakis NE, Labadarios DN, Kafatos AG. Cigarette smoking, alcohol consumption, and serum lipid profile among medical students in Greece.Eur J Public Health. 2003; 13(3):278-282.
15. Emerging Risk Factors Collaboration, Di Angelantonio E, Salwar N, et al. Major lipids, apolipoproteins, and risk of vascular disease.JAMA. 2009;302(18):1993-2000.