Section: Physiology

Original Research Article

COMPARATIVE ANALYSIS OF PLASMA FIBRINOGEN AND PLATELET COUNTS IN SMOKERS AND NON-SMOKERS

Anil Das R[1], Deepa V K[2], Geetha N[3], Lakshmi G[4]

[1]Assistant Professor, Department of Physiology, Government Medical College, Konni, Pathanamthitta, Kerala, India.
[2]Assistant Professor, Department of Physiology, Government Medical College, Kollam, Kerala, India.
[3]Professor, Department of Physiology, SUT Academy of Medical Sciences, Vattappara, Thiruvananthapuram, Kerala, India.
[4]Professor, Department of Physiology, Government Medical College, Thiruvananthapuram, Kerala, India.

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Corresponding Author:
Dr. Anil Das R,
Email: dranildasr@gmail.com

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ABSTRACT

Background: Smoking, the prime cause of preventable deaths worldwide can alter several haematological parameters. The objectives of this study were to compare the plasma fibrinogen level and study its association between smokers and non-smokers and to compare the platelet counts between them. **Materials and Methods** A cross-sectional study was done in healthy male smokers and non-smokers. Data was collected from each participant using a proforma and their blood investigated for plasma fibrinogen, platelet count and other selected haematological parameters. **Result:** In smokers, statistically significant increase in plasma fibrinogen level and platelet count was found. Serum total Cholesterol, LDL and Triglycerides were decreased while serum HDL was found increased. Haemoglobin levels and haematocrit value did not show any significant difference. **Conclusion:** Elevation of plasma fibrinogen, platelet count and dyslipidaemia were important cardiovascular risk factors in otherwise healthy smokers. A regular monitoring of these parameters can aid in early detection of cardio-vascular disease in them so that remedial measures can be instituted early.

INTRODUCTION

Worldwide, smoking is a major cause of preventable death, contributing to respiratory,[1,2] cardiovascular,[3,4] and malignant diseases; including lung, oral, and bladder cancers, as well as stroke and diabetes.[5] Globally, over 1.1 billion people smoke, with 250 million in India.[6] Despite declining smoking rates, tobacco-related diseases continue to cause over four million deaths annually, including about 800,000 in India.[7]

In Kerala, smoking prevalence among men aged 15–64 years was reported at 35%, with around 24,000 annual tobacco-related deaths.[8] Smoking generates free radicals and oxidative stress, promoting atherosclerosis and reducing antioxidant levels, thereby increasing the risk of coronary artery disease and sudden cardiac death through arrhythmias and coronary spasm.[9,10]

Furthermore, smoking elevates plasma fibrinogen levels- an indicator of cardiovascular risk, and enhances platelet activity, contributing to thrombosis and vascular damage.[11-13] Regular monitoring of plasma fibrinogen, platelet count, and other hematological parameters may aid in early detection and prevention of smoking-induced cardiovascular diseases, reducing long-term morbidity and mortality.[11,12]

MATERIALS AND METHODS

This study was conducted at an urban health center of medical college Thiruvananthapuram, Kerala over one year. Sixty apparently healthy male participants aged 30-40 years were enrolled consecutively from attendees of Non- Communicable Disease (NCD) clinic into two groups- 30 smokers and 30 non-smokers. Men aged 30-40 years without chronic illnesses were included. Ex- smokers, passive smokers, individuals with diabetes, hypertension, thyroid or liver disease, obesity and with alcohol addiction were excluded.

Ethical clearance was obtained from the institutional ethics committee. After taking informed consent; demographic details, smoking history and clinical parameters were recorded using a structured proforma. Height, weight, pulse rate and blood pressure were measured using standard procedures,

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and body mass index was calculated as weight(kg)/height(m)^2.

Five milliliters of venous blood was collected under aseptic precautions and distributed into plain, EDTA, and trisodium citrate vials for hematological, biochemical and coagulation analysis respectively. Citrated samples were centrifuged at 1700g for 30 minutes to obtain platelet poor plasma.

Plasma fibrinogen was estimated by the Clauss method,[14] using an STA compact Max 2 coagulation analyzer (Diagnostica Stago, France). Hematological parameters, including platelet count, hemoglobin, and packed cell volume, were analyzed on a Sysmex KX-21N semi- automated hematology analyzer (Sysmex Corporation, Japan) based on the Coulter principle. Lipid profile (Total cholesterol, HDL, Triglycerides) was determined by standard enzymatic colorimetric methods, and LDL cholesterol was calculated using Friedewald's formula.

Data were entered into Microsoft excel and analyzed using SPSS version 20 (IBM Corp, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Differences between smokers and non-smokers were assessed using the independent samples t-test. A p-value <0.05 was considered statistically significant.

RESULTS

The present cross sectional analytical study was conducted among 30 healthy male smokers and 30 male non- smokers attending the outpatient department of an urban health center of Government Medical College, Thiruvananthapuram.

The mean age of the study population was 35.07 and 35.47 years respectively in smokers and non-smokers. Mean BMI of the non- smoker group was less when compared to that of the smoker group and the difference was statistically significant.

The average number of cigarettes smoked by the smoker group was 14.53 with a range of 10 to 20 cigarettes smoked per day. The mean duration of smoking was found to be 11.2 ± 4 years. 43.3 % of smokers were at 8 pack years or below while 56.7% recorded above 8 pack years in cigarette consumption.

The pulse rates of smokers were found to be more in comparison to non-smokers and this difference was statistically significant. Smokers also showed an increase in mean systolic and diastolic blood pressure- again statistically significant. The fibrinogen level and platelet count among smokers was more compared to the non- smoker group and this difference also showed statistical significance.

The mean haematocrit of smokers was 45.7 ± 2.4 and of non- smokers 45.7 ± 2.7 percent. Independent samples t test did not show any significant difference here. The mean cholesterol, mean LDL cholesterol, mean HDL cholesterol levels were all found to be increased among smokers and showed statistical significance. Though the mean triglyceride level of the smoker group was raised in comparison to the non- smoker group, no statistical significance was found.

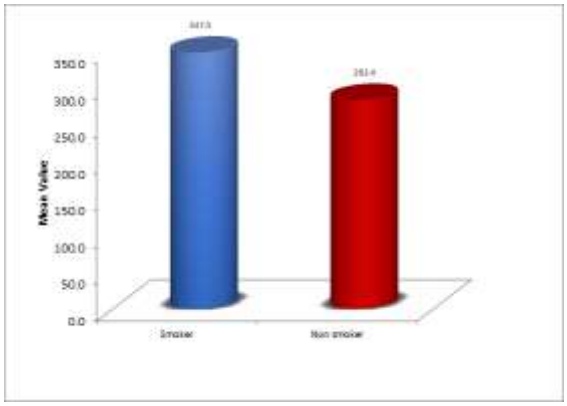

Figure 1: Mean Plasma Fibrinogen Level Between Smokers and Non-Smokers

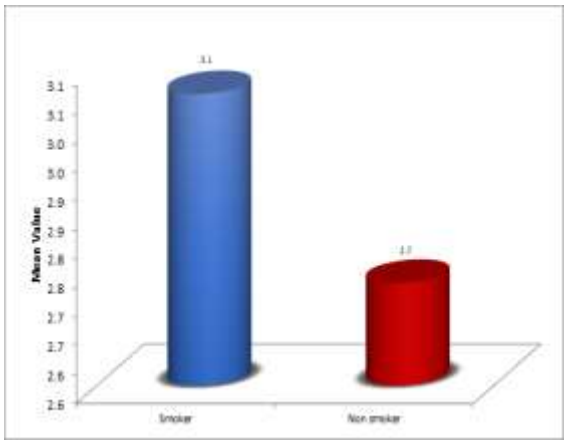

Figure 2: Mean Platelet Counts of Smokers and Non-Smokers

Table 1: Comparison of Study Parameters between Smokers and Non-Smokers

	Smoker			Non- Smoker			t	p
	Mean	SD	N	Mean	SD	N		
Pulse Rate	80.5	4.2	30	74.2	6.3	30	4.58	**<0.01**
Systolic BP	132.6	7.3	30	126.4	9.1	30	2.92	**0.005**
Diastolic BP	85.9	6.1	30	76.1	6.3	30	6.13	**<0.01**
Body Mass Index	25.6	1.6	30	24.8	1.4	30	2.04	**0.046**
Fibrinogen	347.5	80.9	30	283.4	79.4	30	3.1	**0.003**
Platelet count	3.1	0.6	30	2.7	0.6	30	2.16	**0.035**
Haemoglobin	13.7	0.7	30	13.7	0.8	30	0	1

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Packed Cell Volume	45.7	2.4	30	45.7	2.7	30	0.1	0.923
Total Cholesterol	203.6	17.3	30	183.7	20.4	30	4.07	**<0.01**
LDL Cholesterol	128.1	22.9	30	110.9	24.5	30	2.8	**0.007**
HDL Cholesterol	40.2	5.3	30	45.5	5	30	3.95	**<0.01**
Triglycerides	159	27.4	30	136.3	25.4	30	3.33	**0.002**

Values highlighted in bold shows statistical significance ($p<0.05$)

DISCUSSION

Cigarette smoking exerts profound effects on cardiovascular health and hematological parameters. Nicotine and other toxic components of cigarette smoke accelerate atherosclerotic plaque formation, leading to coronary artery disease and myocardial infarction through hypoxia and reduced myocardial oxygen supply. Oxidative stress and inflammation induced by smoking contribute significantly to these pathophysiological changes.

In this study, smokers showed a statistically significant elevation in plasma fibrinogen levels compared to non-smokers. Elevated fibrinogen is a known independent risk factor for ischemic heart disease and stroke, reflecting a chronic inflammatory and hypercoagulable state.[15] The increase is likely mediated through smoking-induced cytokine release, especially interleukin-6,[16] which stimulates hepatic fibrinogen synthesis. Although no clear dose-response relationship was observed with pack years, this may be attributed to increased fibrinogen consumption in long-term smokers due to ongoing endothelial injury.

The mean platelet count was also significantly higher among smokers, suggesting enhanced thrombopoiesis and platelet activation secondary to endothelial dysfunction and chemical stimulation by nicotine and carbon monoxide. However, prolonged smoking appeared to decrease platelet counts slightly, possibly reflecting platelet aggregation and consumption in atherogenic processes.[17] These findings align with previous studies that demonstrate smoking- related endothelial damage and altered platelet kinetics.

Lipid profile alterations among smokers were evident, with significantly higher total cholesterol, LDL, and triglyceride levels while HDL concentration was lower. Such dyslipidemia promotes atherogenesis and further elevates cardiovascular risk. The mechanisms may involve nicotine-induced catecholamine release, increased free fatty acid mobilization, reduced lipoprotein lipase activity, and decreased synthesis of apolipoprotein A1, a major component of HDL.[18] Although the dose-response trend was not statistically significant, the pattern of lipid alteration was consistent with the duration and intensity of smoking.

In contrast, hemoglobin and hematocrit levels did not show significant differences between smokers and non-smokers in this study, despite other reports indicating an increase as a compensatory response to hypoxia from carbon monoxide exposure. Variability in results may be influenced by sample size, duration of smoking, or individual physiological differences.

Overall, these findings support that cigarette smoking induces a complex interplay of inflammatory, oxidative, and hematologic changes that collectively increase the risk of cardiovascular disease. Even short-term cessation has been shown to reduce fibrinogen synthesis and improve vascular function, underscoring the reversibility of some smoking-induced alterations and the critical importance of smoking cessation for cardiovascular health.

CONCLUSION

On analysis it was found that in smokers, plasma fibrinogen concentration, platelet count and lipid profile were increased and the differences were statistically significant.

Present study therefore concludes that:

- In smokers, plasma fibrinogen concentration, platelet count and lipid profile increased significantly.
- Smoking may increase inflammation and dyslipidaemia which contributes to atherosclerosis in smokers.
- Fibrinogen could be used as a bio-marker of atherogenic risk prediction among healthy male smokers.
- Routine monitoring of Plasma Fibrinogen and platelets in smokers is advisable as changes can be detected at an early stage and preventive measures adopted.
- Prevention through health education and behavioural modification may bring down the mortality and morbidity due to cardiovascular disease to a great extent.
- High commitment to smoking cessation can be ensured when tangible evidence of developing abnormalities in blood can be presented to smokers early.
- The impediment to plasma fibrinogen testing in India is the high cost involved which could be addressed by appropriate market or governmental interventions to bring down the cost.

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