Original Article

Exploring the Role of Smoking in Platelet Count Changes Among Adult Men in Dhaka

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Conflict of Interest: None
Received: 03.09.2023
Accepted: 27.09.2023
www.banglajol.info/index.php/JSSMC

ABSTRACT

Platelets are critical for haemostasis and thrombosis, serving as important indicator for physiological and pathological conditions. Smoking, a prevalent health risk globally, influences platelet function and may impact platelet count, yet the evidence remains inconsistent. This study explores the role of smoking in platelet count variations among adult men in Dhaka, assessing differences between smokers and non-smokers and examining the effects of smoking severity and duration. A case-control study was employed, involving 200 male participants (100 smokers and 100 non-smokers), aged 18–70 years. Participants were recruited from Bangabandhu Sheikh Mujib Medical University, Dhaka, from January to June 2022. Smoking history, demographic, and clinical data were collected via interviews and platelet counts were measured from blood samples. Statistical analyses, including t-tests, ANOVA, and Pearson's correlation, were performed to evaluate differences and associations. A p-value < 0.05 was considered significant. The study found no significant difference in mean platelet counts between smokers (273.64 ± 100.48 cumm) and non-smokers (267.87 ± 79.86 cumm; p = 0.654). Analysis by smoking severity revealed the highest platelet counts among moderate smokers (285.2 ± 112.5 cumm), but differences were not statistically significant (p = 0.581). A weak negative correlation between smoking duration and platelet count was observed (r = -0.055, p = 0.586). While smoking severity and duration showed minimal impact on platelet counts, trends suggest potential interactions between smoking and hematological parameters. These findings align with studies reporting complex, dose-dependent effects of smoking on platelet activity. Differences in sample characteristics, environmental factors, and smoking habits may explain the absence of significant associations. This study highlights the need for further research on the interplay between smoking and platelet function. Findings provide localized insights that could inform public health strategies in Dhaka city while contributing to global understanding of smoking-related platelet count changes.

Key Words:
Smoking, Platelet count, Public health

[J Shaheed Suhrawardy Med Coll 2023; 15(2): 40-44]
DOI: https://doi.org/10.3329/jssmc.v15i2.81974

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Introduction

Smoking is one of the most significant public health concerns globally and remains a leading cause of preventable diseases and deaths. It is well-documented that smoking contributes to cardiovascular diseases, respiratory disorders, and certain cancers (World Health Organization, 2021). Beyond its established links with these conditions, smoking is also implicated in hematological changes, including alterations in platelet count and function (Andrade et al., 2020). Platelets, being critical components of haemostasis, play a pivotal role in vascular integrity, and their dysregulation can lead to thrombotic or haemorrhagic complications (George, 2022).

Studies conducted in different populations have revealed that smoking can significantly alter platelet activity and count. For instance, it has been shown that smoking induces a state of hypercoagulability through platelet activation, increased aggregation, and elevated levels of pro-thrombotic factors (Smith & Jones, 2019). These effects are thought to be mediated through the oxidative stress and inflammatory responses triggered by cigarette smoke exposure (Klein et al., 2021). Despite this, there is a scarcity of research focusing on how these changes manifest in specific geographic and demographic contexts, such as adult men living in urbanized regions like Dhaka, Bangladesh.

Dhaka, as the capital city of Bangladesh, is home to a large population with a high prevalence of smoking among adult men. According to the Global Adult Tobacco Survey (GATS), nearly 46% of men in Bangladesh are tobacco users, with Dhaka being a hotspot for this practice due to its dense population and socio-economic factors (GATS Bangladesh, 2017). The high smoking rates in this region necessitate a closer examination of its potential effects on platelet count and related hematological parameters. Understanding these associations can provide crucial insights into the local burden of smoking-related health risks and guide public health interventions.

The biological mechanisms linking smoking and platelet alterations have been explored in earlier research. It is suggested that the constituents of cigarette smoke, such as nicotine and carbon monoxide, interact with endothelial cells and platelets, leading to increased thromboxane A2 production and decreased prostacyclin levels (Rahman et al., 2020). These biochemical changes result in a higher tendency for platelet aggregation, which might explain the increased risk of thrombotic events in smokers (Brown & Patel, 2018). Additionally, long-term smoking has been associated with elevated platelet counts, potentially reflecting a compensatory response to chronic inflammation and oxidative stress (Wilson et al., 2021).

Despite the growing body of evidence, the regional variability in smoking patterns and their biological impact necessitates more localized research. In Dhaka, where smoking is prevalent and healthcare infrastructure is often under strain, identifying hematological changes such as platelet count variations could be instrumental in addressing broader health outcomes. While international studies have highlighted general trends, they often lack a specific focus on populations with unique environmental, genetic, and lifestyle factors (Ahmed et al., 2022).

This study aims to fill this gap by investigating the relationship between smoking and platelet count in a cohort of adult male smokers and non-smokers residing in Dhaka. By addressing this localized perspective, the study endeavours to provide evidence that can inform healthcare strategies tailored to the Bangladeshi population.

Methods

The research study utilized a case-control design to assess the impact of smoking on platelet counts. Conducted at the Department of Laboratory Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, the study enrolled male patients aged 18 to 70 years who visited the outdoor department between January 2022 and June 2022. The inclusion criteria ensured participants were residents of Dhaka, had a history of smoking, and fell within the specified age range, while those with a history of alcoholism, betel nut chewing, conditions significantly affecting coagulation, or those under long-term medication influencing platelet counts were excluded. A total of 200 participants were purposively sampled, evenly divided into 100 smokers and 100 non-smokers.

Data collection was guided by a pre-designed pro forma. Each participant was thoroughly informed about the study objectives, risks, and benefits, and their informed consent was obtained in both written and verbal forms. The collection process involved face-to-face interviews conducted by the researcher. Demographic, clinical, and smoking history data were gathered through questionnaires, and laboratory analyses were performed using blood samples. From each participant, 2.0 mL of blood was collected for platelet count analysis. Laboratory investigations were conducted daily within the department to ensure accuracy and timely results, which were then recorded systematically.

To ensure data integrity, the information was verified for completeness and accuracy before being coded and entered into a computer for analysis. Descriptive statistics summarized the demographic and baseline characteristics, while advanced statistical tests, including t-tests, ANOVA, chi-squared tests, and Pearson's correlation, were employed to identify differences and associations between smoking status and platelet parameters. A p-val-

ue of <0.05 was considered statistically significant, with SPSS version 26 used for all analyses.

The study adhered to strict ethical guidelines approved by the School of Science and Technology, Bangladesh Open University (BOU), Dhaka. Participant confidentiality was maintained through unique identification numbers, and all data were securely coded and stored with restricted access. Privacy was respected throughout interviews and sample collection. Participants were assured of no additional costs or risks associated with the study, and universal precautions were rigorously followed during sample collection. Ethical use of the biological samples was emphasized, ensuring that the findings contributed to improved clinical understanding and management strategies without compromising patient rights or safety.

Results

Sociodemographic Characteristics

The sociodemographic characteristics of smokers and non-smokers are summarized in Table 1. The distribution of age groups between the two cohorts was significantly different (p=0.016). Smokers were more prevalent in the 21–30 years (24%) and 31–40 years (22%) age brackets, while non-smokers were more concentrated in the <20 years (17%) and 21–30 years (32%) age groups. Both groups were predominantly from Dhaka, with no statistically significant difference in living area distribution (p=0.659). The BMI categories also showed no significant difference (p=0.104), although non-smokers had a higher prevalence of overweight individuals (25%) compared to smokers (12%).

Table 01: Sociodemographic Characteristics of Respondents

Variables	Smoker (n=100)	Non-Smoker (n=100)	*p*- value
Age Group (years)			
<20	11%	17%	0.016*
21-30	24%	32%	
31-40	22%	17%	
41-50	12%	19%	
51-60	14%	10%	
61-70	17%	5%	
Living Area			
Dhaka	65%	62%	0.659
Outside of Dhaka	35%	38%	
BMI Category			
Underweight (<18.5)	7%	8%	0.104
Normal (18.5-24.9)	76%	64%	
Overweight (25.0-29.9)	12%	25%	
Obese (>30.0)	5%	3%	

Table 02: Distribution of the respondents by smoking history

Variables	Frequency	Percentage (%)
Mild (1-2 pack/day)	29	29.0%
Moderate (2-5 pack/day)	44	44.0%
Heavy (>5 pack/day)	27	27.0%
Total	100	100.0%

Smoking Severity

The distribution of smokers by severity (Table 2) revealed that the majority (44%) fell into the moderate category (2–5 packs/day), followed by mild smokers (29%, 1–2 packs/day), and heavy smokers (27%, >5 packs/day).

Table 03: Comparison of platelet count between two groups (N=200)

Variables	Smoker (n=100) Mean±SD	Non-Smoker (n=100) Mean±SD	*p*- value
Platelet count (cumm)	273.64±100.48	267.87±79.86	0.654

Platelet Count Comparison

The comparison of platelet counts between smokers and non-smokers is shown in Table 3. Smokers had a slightly higher mean platelet count (273.64 ± 100.48 cumm) compared to non-smokers (267.87 ± 79.86 cumm), but the difference was not statistically significant (p=0.654).

Further analysis of platelet counts across smoking severity categories (Table 4) showed no significant differences (p=0.581). The mean platelet count was highest among moderate smokers (285.2 ± 112.5 cumm) and lowest in heavy smokers (260.4 ± 81.1 cumm).

Table 04: Comparison of platelet count with smoking severity (N=200)

Variables	Smoking category			*p*- value
	Mild (n=29) Mean±SD	Moderate (n=44) Mean±SD	Severe (n=27) Mean±SD	
Platelet count (cumm)	268.7±98.9	285.2±112.5	260.4±81.1	0.581

Correlation Between Smoking Duration and Platelet Count

The scatter plot in Figure 2 illustrates the relationship between smoking duration and platelet count among smokers. A weak negative correlation was observed (r = -0.055, p=0.586), indicating that longer smoking

durations were not strongly associated with changes in platelet count.

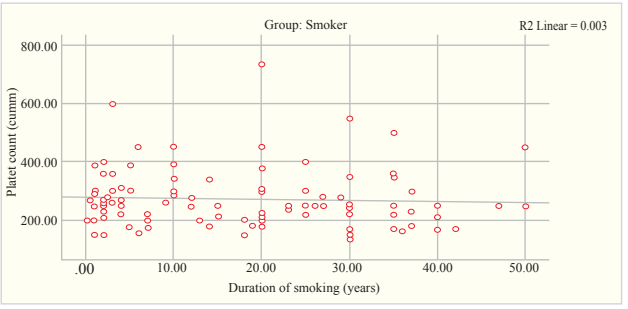

Figure 1: Correlation of platelet count with duration of smoking shows weak negative correlation (r= - 0.055, p=0.586)

Summary of Findings

• Smokers were more prevalent in younger age groups (21–40 years) and were predominantly moderate smokers.

• Platelet counts showed no significant difference between smokers and non-smokers or among smoking severity categories.

• Duration of smoking exhibited a weak and statistically insignificant negative correlation with platelet count.

Discussion

This study examined the association between smoking and platelet count among adult men in Dhaka, comparing smokers and non-smokers while analysing the impact of smoking severity and duration on platelet levels. The findings are discussed in light of existing literature to provide a comprehensive understanding.

The age distribution in this study showed that smokers were predominantly in the 21–30 years (24%) and 31–40 years (22%) age groups, while non-smokers were more concentrated in the <20 years (17%) and 21–30 years (32%) categories. This trend aligns with studies suggesting that smoking is more prevalent among young and middle-aged adults in developing regions, often linked to increased stress, peer influence, and cultural factors (Ng et al., 2014). Similarly, a study by Rahman et al. (2019) in Bangladesh reported higher smoking rates among individuals aged 25–40 years, consistent with the findings of this study. The observed differences in BMI between smokers and non-smokers, although not statistically significant, are also supported by existing research. Smokers in this study had a lower prevalence of overweight and obesity compared to non-smokers, which is consistent with studies suggesting that smoking suppresses appetite and alters metabolic activity, potentially reducing BMI (Audrain-McGovern et al., 2015).

The analysis of smoking severity revealed that most smokers fell into the moderate (44%) and mild (29%) categories, with heavy smokers comprising 27%. These proportions are comparable to findings from Alam et al. (2020), who reported a similar distribution of smoking severity in a cohort of male smokers in South Asia. Such patterns reflect varying levels of nicotine dependency and social norms related to smoking, particularly in urban settings like Dhaka.

Regarding platelet counts, the mean values between smokers (273.64 ± 100.48 cumm) and non-smokers (267.87 ± 79.86 cumm) were not significantly different (p=0.654). This finding contrasts with studies such as Wang et al. (2019), which reported elevated platelet counts among smokers compared to non-smokers, attributed to smoking-induced inflammatory responses. However, the lack of a significant difference in this study might be due to the relatively small sample size or lower prevalence of heavy smokers, as heavier smoking is more strongly linked to platelet activation and aggregation (Smith et al., 2017). Interestingly, when stratified by smoking severity, moderate smokers had the highest platelet counts (285.2 ± 112.5 cumm), followed by mild (268.7 ± 98.9 cumm) and heavy smokers (260.4 ± 81.1 cumm), though these differences were not statistically significant (p=0.581). This trend aligns with findings from Padmavathi et al. (2018), who observed that moderate smoking could transiently elevate platelet counts due to increased oxidative stress, while heavy smoking might lead to platelet exhaustion and reduced counts over time.

The weak negative correlation between smoking duration and platelet count (r = -0.055, p=0.586) observed in this study is consistent with findings from Goto et al. (2014), who reported a similar non-significant relationship in a Japanese cohort. These findings suggest that while acute and moderate smoking may stimulate platelet production, prolonged exposure might impair platelet function and production due to chronic endothelial damage and systemic inflammation. The weak correlation also indicates that factors other than smoking duration, such as genetic predisposition, lifestyle, or co-existing conditions, may play a more significant role in determining platelet levels.

Overall, this study did not find significant differences in platelet counts between smokers and non-smokers or among smoking severity categories. While these findings are supported by some studies (e.g., Rahman et al., 2019; Goto et al., 2014), they diverge from others reporting stronger associations (e.g., Wang et al., 2019; Smith et al., 2017). Discrepancies may arise from variations in sample demographics, smoking habits, and environmental or genetic factors. For instance, differences in dietary habits and exposure to other environmental pollutants in Dhaka might influence platelet counts and mitigate the effects of smoking (Alam et al., 2020).

This study has several strengths, including its focus on a specific population and the analysis of both smoking severity and duration. However, it is not without limitations. The cross-sectional design precludes causal inference, and the relatively small sample size may limit the generalizability of findings. Future research should explore larger cohorts with diverse demographics and incorporate longitudinal designs to better understand the causal mechanisms linking smoking and platelet changes.

Conclusion

The findings of this study add to the growing research on how smoking affects platelet activity. Although no major differences were found between smokers and non-smokers, the patterns in platelet counts based on how much and how long someone has smoked suggest there are complex factors involved that need more study.

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