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ASSESSMENT OF PLATELET COUNT AND PLATELET INDICES (MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH, PLATELETCRIT) AMONG SMOKERS COMPARED TO NON – SMOKERS IN ALGAZIRA STATE - SUDAN 2026

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ABSTRACT

Smoking contributes substantially to the global burden of cardiovascular disease through mechanisms including endothelial damage, oxidative stress, and increased platelet reactivity. Platelet indices, Platelet Count (PLT), Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), and Plateletcrit (PCT) are important indicators of platelet activation and thrombotic risk. However, data assessing these parameters in Sudanese smokers remain limited. This comparative cross-sectional study was carried out in Algazira State, Sudan from May to August 2026, and aimed to evaluate and compare platelet count and platelet indices (MPV, PDW, PCT) between adult male smokers and non-smokers. A total of 150 adult males were recruited, including 70 current cigarette smokers and 80 non-smokers as controls. Complete blood count parameters were measured using automated hematology analyzers. Data analyzed using SPSS version 26). Platelet Distribution Width (PDW) was significantly elevated in smokers compared to non-smokers. In contrast, no significant differences were found in Platelet Count, MPV or PCT. Moreover, within the smoker group, Spearman's analysis showed no significant correlation between smoking duration or smoking intensity and any of the studied platelet indices (all $p>0.05$). In conclusion, among adult Sudanese males, cigarette smoking is associated with increased platelet size heterogeneity, as reflected by higher PDW levels. This suggests morphological and functional platelet alterations without significant changes in platelet count. PDW may thus represent a simple and cost-effective hematological marker for early detection of smoking-related platelet activation and thrombotic risk.

Keywords: Platelet Count, Platelet Indices (MPV, PDW, PCT), Cigarette Smoking, Cardiovascular Risk, Sudan

1 INTRODUCTION

Smoking remains a major preventable cause of morbidity and premature mortality worldwide. Despite global control measures, its prevalence remains high across both developed and developing countries. The World Health Organization identifies tobacco use as a leading contributor to cardiovascular, respiratory and malignant diseases, representing a major global health burden (1). Platelets are key components of primary hemostasis and also participate in inflammatory and vascular processes (2,3). Platelet activation is closely linked to thrombotic and atherosclerotic disease development, making platelet alterations potential early indicators of cardiovascular risk (3). In Sudan, limited data are available regarding this association, indicating a need for local investigation.

Cigarette smoking is a major risk factor for cardiovascular diseases and may affect platelet production, activation and function. These effects may be reflected in changes in platelet count and platelet indices. Several studies have investigated the effect of cigarette and tobacco smoking on platelet count and platelet indices but still findings were inconsistent among different populations. The importance of studying the Sudanese population lies in the limited availability of population-specific evidence on the hematological effects of cigarette smoking in Sudan. This study aims to assess platelet count, MPV, PDW, and PCT among cigarette smokers in Sudan and compare them with non-smokers. The findings may help clarify the inconsistent results reported in previous studies and contribute to the local evidence base, providing a foundation for future research on the hematological effects of cigarette smoking in Sudan.

Objectives

To assess platelet count and platelet indices (MPV, PDW and PCT) among smokers and compare them with non-smokers in Algazira State compare them with non-smokers in Algazira State

2 METHODOLOGY

This was a comparative cross-sectional study, conducted in Algazira State, Sudan between May 2026 and August 2026. The study population were adult male smokers and apparently healthy adult male non-smokers as a control group, participants included if they were (Adult males aged 18 years or older, smokers for at least one year (smoker group). And participants were excluded if they (Had any chronic respiratory disease, history of hematological disorders, an acute infection or fever during the previous two weeks, were taking medications that could affect blood cell counts or platelet function (aspirin, anticoagulants, or antiplatelet drugs), donated blood or undergone surgery within previous three months and refused to participate in the study)

A total of 150 participants were enrolled in the study using a convenience sampling technique (70 smokers - 80 non-smokers).

Data were collected using a structured interviewer-administered questionnaire designed to obtain demographic characteristics, smoking history (duration of smoking and number of cigarettes smoked per day), lifestyle factors, and relevant medical history. Laboratory data were obtained from complete blood count (CBC) analysis. Blood samples were collected aseptically from each participant into EDTA anticoagulant tubes for complete blood count (CBC) analysis, using automated hematology analyzers according to the manufacturers' instructions and standard laboratory operating procedures.

The following platelet parameters were measured: Platelet Count (PLT) - Mean Platelet Volume (MPV) - Platelet Distribution Width (PDW) - Plateletcrit (PCT)

Participation in this study was entirely voluntary. Written informed consent was obtained from all participants before enrollment.

Data were entered, coded and analyzed using the Statistical Package for the Social Sciences (SPSS) version 26. Descriptive statistics were used to summarize the study variables. Continuous variables were expressed as mean ± standard deviation (SD), median, minimum, and maximum values. The normality of data distribution was assessed using the Shapiro–Wilk test. Comparisons between smokers and non-smokers were performed using the Independent Samples t-test for normally distributed data. When the data were not normally distributed, the Mann–Whitney U test was used. Spearman's rank correlation analysis was performed to assess the relationship between smoking duration, the number of cigarettes smoked per day, and platelet parameters. A P-value of less than 0.05 was considered statistically significant.

3 RESULTS

A total of 150 participants were included, comprising 80 non-smokers (control group) and 70 smokers. Smokers showed a higher mean platelet count (251.629 ± 105.853 $\times 10^3/\mu L$) compared to non-smokers (230.525 ± 93.139 $\times 10^3/\mu L$). The mean platelet distribution width (PDW) was notably higher in smokers (15.051 ± 2.841 fL) than in non-smokers (12.866 ± 2.684 fL). Similarly, plateletcrit (PCT) was slightly elevated in smokers (0.231 ± 0.101%) compared to controls (0.214 ± 0.082%). In contrast, mean platelet volume (MPV) was marginally lower in smokers (9.503 ± 1.070 fL) than in non-smokers (9.631 ± 1.505 fL).

Table 1: Descriptive statistics of Platelet count and Platelet indices

Max	Min	Median	Mean ± SD	n	Group	Variable
583.000	52.000	217.000	230.525 ± 93.139	80	Control	Plt Count $\times 10^3/\mu L$
655.000	90.000	220.000	251.629 ± 105.853	70	Smoker	Plt Count $\times 10^3/\mu L$
19.500	7.300	9.350	9.631 ± 1.505	80	Control	MPV (fL)
12.200	7.400	9.450	9.503 ± 1.070	70	Smoker	MPV (fL)
21.400	8.200	12.300	12.866 ± 2.684	80	Control	PDW (fL)
20.500	7.600	16.100	15.051 ± 2.841	70	Smoker	PDW (fL)
0.550	0.060	0.220	0.214 ± 0.082	80	Control	PCT (%)
0.700	0.080	0.210	0.231 ± 0.101	70	Smoker	PCT (%)

The Shapiro-Wilk test was used to evaluate the normality of data distribution (Table 2). Most variables deviated significantly from normal distribution ($p<0.05$). Platelet count, PDW, and PCT were non-normally distributed in both groups, while MPV was non-normally distributed in non-smokers ($p = 0.003$). Only MPV in the smoker group followed a normal distribution ($W = 0.9867$, $p = 0.669$). Therefore, non-parametric statistical tests were applied for all subsequent analyses.

Table 2: Shapiro–Wilk Test for Normality

Distribution	p-value	W statistic	Group	Variable
Not normal	<0.001	0.9335	Control	Platelet Count ($\times 10^3/\mu L$)
Not normal	<0.001	0.8389	Smoker	Platelet Count ($\times 10^3/\mu L$)
Not normal	<0.001	0.7292	Control	MPV (fL)
Normal	0.669	0.9867	Smoker	MPV (fL)
Not normal	<0.001	0.9369	Control	PDW (fL)
Not normal	<0.001	0.9121	Smoker	PDW (fL)
Not normal	0.001	0.9420	Control	PCT (%)
Not normal	<0.001	0.8331	Smoker	PCT (%)

Because at least one group deviated significantly from normality for every variable (Shapiro–Wilk $p<0.05$), the non-parametric Mann–Whitney U test was applied throughout instead of the independent-samples t-test, per the standard decision rule (t-test only when both groups are normally distributed).

Comparison between groups was performed using the Mann-Whitney U test to assess differences in platelet indices between smokers and non-smokers. The rank-biserial correlation coefficient (r) was calculated as a measure of effect size (Table 3).

The analysis revealed a statistically significant difference only for PDW ($U = 1428.00$, $p<0.001$). Smokers had a significantly higher median PDW (16.100 fL) compared to non-smokers (12.300 fL), with a large effect size ($r = 0.490$).

No statistically significant differences were observed in the comparison between groups for platelet count (U = 2509.50, p = 0.275, r = 0.104), MPV (U = 2839.00, p = 0.885, r = -0.014), and PCT (U = 2634.00, p = 0.532, r = 0.059), all showing small effect sizes.

Table 3: Comparison of Platelet count and Platelet indices between groups

p-value	Statistic	Test Applied	Smoker (Mean±SD / Median)	Control (Mean±SD / Median)	Variable
0.275	2509.50	Mann-Whitney U	251.629±105.853/ 220.000	230.525±93.139/ 217.000	Platelet Count ($\times 10^3/\mu L$)
0.885	2839.00	Mann-Whitney U	9.503±1.070 / 9.450	9.631±1.505 /9.350	MPV (fL)
<0.001 *	1428.00	Mann-Whitney U	15.051±2.841 / 16.100	12.866±2.684/12.300	PDW (fL)
0.532	2634.00	Mann-Whitney U	0.231±0.101 / 0.210	0.214±0.082 /0.220	PCT (%)

* Statistically significant at p<0.05

The relationship between smoking duration (years) and platelet indices among smokers was assessed using Spearman's rank correlation (Table 4).

No significant correlations were found between smoking duration and platelet count (ρ = 0.013, p = 0.916), MPV (ρ = -0.105, p = 0.385), PDW (ρ = -0.152, p = 0.208), or PCT (ρ = -0.066, p = 0.585). All correlations were weak.

Table 4: Correlation between smoking duration and platelet count and platelet indices

Significance	p-value	Spearman ρ	Platelet Index
Not significant	0.916	0.013	Platelet Count ($\times 10^3/\mu L$)
Not significant	0.385	-0.105	MPV (fL)
Not significant	0.208	-0.152	PDW (fL)
Not significant	0.585	-0.066	PCT (%)

Spearman's correlation was used to examine the association between smoking intensity (cigarettes per day) and platelet indices (Table 5).

No significant associations were identified. Platelet count (ρ = 0.065p = 0.591), MPV (ρ = 0.100, p = 0.409), PDW (ρ = -0.057, p = 0.640), and PCT (ρ = -0.003, p = 0.981) showed negligible and non-significant correlations with smoking intensity.

Table 5: Correlation between smoking intensity and platelet and platelet indices

Significance	p-value	Spearman ρ	Platelet Index
Not significant	0.591	0.065	Platelet Count ($\times 10^3/\mu L$)
Not significant	0.409	0.100	MPV (fL)
Not significant	0.640	-0.057	PDW (fL)
Not significant	0.981	-0.003	PCT (%)

4 DISCUSSION

This study evaluate platelet count and platelet indices (MPV, PDW and PCT) among apparently healthy male cigarette smokers and non-smokers in Algazira State Sudan. The findings showed that PDW was significantly higher among smokers compared with non-smokers, while platelet count, MPV and PCT showed no statistically significant differences

between the two groups. In addition, no significant correlations were observed between smoking duration or smoking intensity and any of the studied platelet parameters.

In this study, the mean platelet count was higher among smokers (251.629 ± 105.853 $\times 10^3/\mu L$) compared with non-smokers (230.525 ± 93.139 $\times 10^3/\mu L$); however, the difference was not statistically significant ($p = 0.275$).

This finding is consistent with the previous study conducted among healthy Sudanese males in Bahri Town, which reported no significant difference in platelet count between smokers and non-smokers. The similarity between the Sudanese studies suggests that smoking may not necessarily produce a significant change in the total circulating platelet count among apparently healthy individuals. However, the finding differs from Pujani et al. (2021), who reported a significant increase in platelet count among smokers ($p = 0.038$). Similarly, studies conducted in Rania City, Iraq, and Semnan, Iran, reported significant differences in platelet count between smokers and non-smokers. These differences may be related to variations in study populations, sample sizes, smoking exposure, smoking intensity, and laboratory methods (4).

Regarding MPV, this study showed a slightly lower mean MPV among smokers (9.503 ± 1.070 fL) than among non-smokers (9.631 ± 1.505 fL), but the difference was not statistically significant ($p = 0.885$). This result agrees with the study conducted in Rania City, Iraq, which found no significant difference in MPV between smokers and non-smokers (5). These differences indicate that the relationship between smoking and MPV may vary among populations. In addition, MPV can be affected by pre-analytical factors such as anticoagulant, sample storage time, and handling, which may contribute to differences between studies. In the present dataset, MPV also showed non-normal distribution in the control group.

The most important finding of the present study was the significant increase in PDW among smokers. The mean PDW was 15.051 ± 2.841 fL in smokers compared with 12.866 ± 2.684 fL in non-smokers, and the difference was highly statistically significant ($p < 0.001$), with a moderate-to-large effect size (rank-biserial $r = 0.490$). The present finding is consistent with Pujani et al. (2021), who reported significantly higher PDW among smokers ($p = 0.001$) (4) .In contrast, the present finding differs from the study conducted in Rania City, Iraq, where PDW did not differ significantly between smokers and non-smokers (5), as well as from the study conducted in Semnan, Iran, where no significant difference was reported in the platelet indices apart from the parameters that showed significant changes (6). The increased PDW observed among smokers may reflect greater variation in platelet size and changes in platelet morphology associated with smoking. Cigarette smoking may promote vascular and inflammatory stress, which can influence platelet activation and platelet production. Consequently, the significant increase in PDW in the absence of significant changes in platelet count, MPV, and PCT may indicate that smoking affects platelet morphology or heterogeneity more than the total number of circulating platelets. The present results showed that PDW was the only platelet parameter with a statistically significant difference between smokers and non-smokers, while the other parameters remained comparable between the two groups.

For PCT, the mean value was slightly higher among smokers (0.231 ± 0.101%) than among non-smokers (0.214 ± 0.082%), but the difference was not statistically significant ($p = 0.532$). This finding differs from Pujani et al. (2021), who reported a significant increase in PCT among smokers ($p = 0.041$) (4), and from studies conducted in Rania City, Iraq (5), and Semnan, Iran, which also reported significant differences in PCT (6). The lack of a significant difference in the present study may be related to the absence of significant differences in platelet count and MPV, as PCT is influenced by these platelet parameters. Therefore, the present findings suggest that smoking did not produce a sufficient change in overall platelet mass to result in a statistically significant alteration in PCT.

The present study also investigated the relationship between smoking duration and platelet parameters. No statistically significant correlations were observed between smoking duration and platelet count, MPV, PDW, or PCT. The correlation coefficients were 0.013, −0.105, −0.152, and −0.066, respectively, and all p-values were greater than 0.05.. Although PDW was significantly higher among smokers in the present study, the absence of a significant relationship with smoking duration may suggest that the presence of smoking exposure was more important than the number of years smoked within the studied population. Similarly, no statistically significant association was found between smoking intensity, measured as cigarettes smoked per day, and platelet count, MPV, PDW, or PCT. The p-values for platelet count, MPV, PDW, and PCT were 0.591, 0.409, 0.640, and 0.981, respectively. These findings differ from Pujani et al. (2021), who reported significant differences in platelet count, PCT, MPV, and PDW according to smoking intensity (4) . One possible explanation for this difference is the method used to measure smoking exposure. Some previous studies used pack-years or categorized smokers according to smoking intensity, whereas the present study assessed cigarettes per day and smoking duration separately. Pack-years may provide a better estimate of cumulative smoking exposure because they incorporate both the amount and duration of smoking. In the present study, smoking duration ranged from 1 to 43 years and smoking intensity ranged from 1 to 20 cigarettes per day, but no significant dose-response relationship was detected.

Overall, the findings of the present study demonstrate that the effect of cigarette smoking on platelet parameters was not uniform.

5 CONCLUSION

The present study concluded that cigarette smoking was significantly associated with an increase in platelet distribution width (PDW) among apparently healthy adult male smokers compared with non-smokers. However, no statistically significant differences were observed in platelet count, mean platelet volume (MPV), or plateletcrit (PCT) between the two groups. Furthermore, neither smoking duration nor the number of cigarettes smoked per day showed a significant association with any of the studied platelet parameters. These findings suggest that cigarette smoking may primarily affect platelet size variability and morphology rather than platelet count or overall platelet mass. The significant elevation of PDW may therefore indicate smoking-related platelet changes even in apparently healthy individuals. However, further studies with larger sample sizes and more detailed assessment of cumulative smoking exposure are recommended to confirm these findings.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

Statement of informed consent

Ethical approval was granted by the institutional ethical committee, and informed consent was obtained from all individual participants included in the study.

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