RESEARCH ARTICLE

"Changes in platelet parameters in leucocytosis"

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ABSTRACT

Abstract

Introduction: Leukocytosis is a common hematological finding in infections, inflammation and hematological disorders. To evaluate changes in platelet parameters among patients with leukocytosis compared with healthy individuals and to assess their diagnostic and clinical significance.

Materials & Methods: This cross-sectional analytical study was conducted in the Department of Pathology, SGT University, from 1st August 2025 to 1st September 2025. It included 800 participants divided into two groups: 400 patients with leukocytosis (total leukocyte count > 11,000/µL) and 400 controls with normal leukocyte counts. Venous blood samples were analyzed using an automated hematology analyzer. Platelet indices assessed included platelet count (PLT), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), platelet large cell count (PLCC) and platelet large cell ratio (PLCR).Data were compiled and analyzed using SPSS statistical software version 26.0.

Results: The mean platelet count was significantly higher in leukocytosis compared with controls (245.49 ± 173.92 vs. 147.84 ± 107.81 $\times 10^3/\mu L$, $p < 0.001$). PCT and PLCC were also significantly elevated ($p < 0.001$), whereas MPV was significantly reduced in leukocytosis (12.19 ± 3.16 vs. 13.97 ± 8.38 fL, $p < 0.001$). PDW and PLCR showed no significant differences between groups. Correlation analysis demonstrated a moderate positive correlation of leukocyte count with PLT ($r = +0.42$), PCT ($r = +0.39$), and PLCC ($r = +0.36$), and a weak negative correlation with MPV ($r = -0.28$). Thrombocytosis (>400 $\times 10^3/\mu L$) was more frequent in leukocytosis (10.8%) compared with controls (3.0%, $p < 0.001$).

Discussion: Leukocytosis is associated with significant increases in platelet count, PCT and PLCC, and a reduction in MPV, indicating enhanced bone marrow activity and altered platelet morphology. Platelet indices, being inexpensive and readily available, may serve as useful adjuncts in the evaluation of leukocytosis, supporting differentiation between reactive and clonal causes. .

Keywords: Leukocytosis, Platelet parameters, Mean platelet volume, Plateletcrit, Platelet indices.

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INTRODUCTION

Leukocytosis, defined as a peripheral white blood cell (WBC) count exceeding the normal upper limit, is a frequent laboratory finding encountered across diverse clinical conditions. Its causes could include benign physiological conditions (exercise, pregnancy, or even acute stress), and pathological ones (bacterial infections, chronic inflammation, trauma and hematological malignancies) [1]. Historically, the assessment of leukocytosis has focused on the type, degree and morphology of leukocytes because these variables are directly related to diagnosis and treatment [2]. This has however changed as more focus is put on simultaneous changes in platelet parameters as hematopoietic cell lines advance and more studies are done to find out more diagnostic and prognostic information. Platelets consist of small, anucleated, cytoplasmic fragments formed of megakaryocytes [3]. In addition to a classical role in hemostasis and thrombus formation, platelets actively engage in inflammation, immunity, angiogenesis and tissue repair [4]. The modern automated analyzers are able to produce not only the counts of platelets but also indices including the mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT) and platelet-large cell ratio (P-LCR) [5]. These indices indicate the heterogeneity of platelet size, platelet activation state, and total platelet biomass

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providing an understanding of both the quantitative and qualitative features of platelet physiology [6]. Notably, a change in these indices is commonly witnessed during systemic inflammatory diseases, infections, autoimmune diseases, and malignancy. Bone marrow activity in leukocytosis is frequently increased leading to the growth of the leukocytic lineage and megakaryocytic lineage [7]. This common activation may be in the form of changed platelet parameters. As an illustration, higher MPV is often linked with a higher platelet production and platelet reactivity as bigger platelets are more active in metabolism and have more granules and thromboxane A2, which add to pro-inflammatory and pro-thrombotic effects [8]. On the same note, high PDW reflects heterogeneity in platelet morphology, which usually due to a rapid platelet turnover or more immature platelets are released into circulation. Plateletcrit is the total mass of platelets and this can vary based on whether the underlying cause of leukocytosis is stimulating or inhibitory of thrombopoiesis [9]. The similarity in the regulatory cytokine environment in the bone marrow can be attributed to the pathophysiological association between the leukocytosis and platelet dynamics. Interleukin-6 (IL-6), granulocyte colony-stimulating factor (G-CSF), and thrombopoietin (TPO) are growth factors that simultaneously affect platelet production and leukocyte production [10]. These mediators increase megakaryocyte proliferation and platelet activation in response to inflammatory conditions that are a frequent cause of leucocytosis [11]. In addition, both activated leukocytes and activated platelets release reactive oxygen species and cytokines that further regulate platelet activity, and platelet adhesion and transmigration, respectively. This reciprocal relationship intensifies the systemic inflammatory processes and it could be the reason why platelet indices change considerably in the case of leucocytosis [12].

The basic aim of the study is to evaluate changes in platelet parameters among patients with leukocytosis compared with healthy individuals and to assess their diagnostic and clinical significance.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted at in the Department of Pathology, SGT University, from 1st August 2025 to 1st September 2025. A total of 800 participants were included in the study. The sample was divided into two equal groups:

- **Group A (Control group):** 400 healthy individuals with normal complete blood counts and no evidence of infection, inflammation, or hematological disorder.
- **Group B (Leukocytosis group):** 400 patients presenting with leukocytosis, defined as a total leukocyte count (TLC) above 11,000 cells/μL, irrespective of underlying etiology.

Inclusion Criteria

- Age 18–70 years.
- Both male and female participants.
- For the leukocytosis group: documented leukocyte count > 11,000 cells/μL on complete blood count.
- For the control group: healthy volunteers or hospital visitors undergoing routine checkups with normal hematological profiles and no acute or chronic illness.

Exclusion Criteria

- Patients with known hematological malignancies such as leukemia or myeloproliferative neoplasms.
- Patients with chronic liver disease, renal failure, or autoimmune disorders known to influence platelet indices.
- Patients on medications affecting platelet production or function, including antiplatelet drugs, chemotherapy, or corticosteroids.
- Recent history of blood transfusion within the last three months.

Data collection

Demographic details, including age and gender, were documented for each participant. Venous blood samples, ranging between 3 and 5 milliliters, were collected aseptically into EDTA-containing tubes to prevent clotting. To minimize pre-analytical errors, samples were analyzed within two hours of collection. The leukocyte counts were confirmed to classify individuals into the leukocytosis or control groups, and platelet indices were measured concurrently. Strict standard operating procedures were followed for specimen handling and processing.All blood samples were analyzed using an automated hematology analyzer (e.g., Sysmex, Beckman Coulter, or equivalent), which provided comprehensive complete blood count results, including platelet parameters. The primary indices assessed were platelet count, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT). These indices were chosen as they reflect both quantitative and qualitative aspects of platelet physiology and are known to vary in systemic inflammatory and hematological conditions.

Statistical Analysis

Data were compiled and analyzed using SPSS statistical software version 26.0. Continuous variables, such as platelet count, MPV, PDW, and PCT, were expressed as mean ± standard deviation, while categorical variables, such as gender, were presented as frequencies and percentages. Independent sample t-tests were applied to compare

the means of continuous variables between the leukocytosis and control groups. A p-value of less than 0.05 was considered statistically significant.

Formal written informed consent was not required with a waiver by the appropriate IRB and/or national research ethics committee.

RESULTS

Data were collected from 800 participants, the mean age of the normal TLC group was 34.6 ± 18.2 years, while that of the raised TLC group was 36.8 ± 17.5 years, showing no significant difference between groups. Males accounted for 53.1% of the normal TLC group and 56.4% of the raised TLC group, whereas females comprised 46.9% and 43.6%, respectively. In terms of platelet parameters, notable differences were observed between the groups. The mean platelet count (PLT) was significantly higher in the raised TLC group (245.49 ± 173.92 $\times 10^3/\mu L$) compared to the normal TLC group (147.84 ± 107.81 $\times 10^3/\mu L$). Plateletcrit (PCT) and platelet large cell count (PLCC) were also increased in leukocytosis (0.28 ± 0.17 vs. 0.18 ± 0.12; and 86.41 ± 47.74 vs. 60.49 ± 38.12, respectively). Conversely, mean platelet volume (MPV) was lower in the raised TLC group (12.19 ± 3.16 fL) compared to the normal TLC group (13.97 ± 8.38 fL). Platelet distribution width (PDW) showed no meaningful difference between groups (16.33 ± 0.61 vs. 16.36 ± 1.27). Platelet large cell ratio (PLCR) also demonstrated a lower mean in raised TLC (41.15 ± 15.40) compared to normal TLC (63.97 ± 266.64), but this difference was not statistically significant.

(Table 1)Independent t-test results confirmed that platelet count, MPV, PCT, and PLCC were significantly different between groups ($p < 0.001$). Patients with leukocytosis had higher platelet counts, PCT, and PLCC, while MPV was significantly reduced. PDW did not differ significantly ($p = 0.686$), and PLCR showed only a trend towards reduction, which was not statistically significant ($p = 0.089$).

Table 1. Baseline Demographic Characteristics of Study Population

Characteristic	Normal TLC (n = 400)	Raised TLC (n = 400)
Age (years), Mean ± SD	34.6 ± 18.2	36.8 ± 17.5
Sex		
Male	212 (53.1%)	225 (56.4%)
Female	187 (46.9%)	174 (43.6%)
Biochemical parameters		
PLT ($\times 10^3/\mu L$)	147.84 ± 107.81	245.49 ± 173.92
MPV (fL)	13.97 ± 8.38	12.19 ± 3.16
PDW (%)	16.36 ± 1.27	16.33 ± 0.61
PCT (%)	0.18 ± 0.12	0.28 ± 0.17
PLCC	60.49 ± 38.12	86.41 ± 47.74
PLCR (%)	63.97 ± 266.64	41.15 ± 15.40

(Table 2)Thrombocytopenia (<150 $\times 10^3/\mu L$) was more common in the normal TLC group (53.9%) compared to the raised TLC group (24.1%). Conversely, normal platelet counts (150–400 $\times 10^3/\mu L$) were more prevalent in leukocytosis (65.2%) compared to controls (43.1%). Thrombocytosis (>400 $\times 10^3/\mu L$) was also more frequent in the raised TLC group (10.8%) compared to the normal TLC group (3.0). These differences were statistically significant ($p < 0.001$).

Table 2. Comparative Analysis of Platelet Parameters Between Groups (Independent t-test)

Parameter	Normal TLC (Mean ± SD)	Raised TLC (Mean ± SD)	t-statistic	p-value
PLT	147.84 ± 107.81	245.49 ± 173.92	-9.53	<0.001
MPV	13.97 ± 8.38	12.19 ± 3.16	3.94	<0.001
PDW	16.36 ± 1.27	16.33 ± 0.61	0.40	0.686
PCT	0.18 ± 0.12	0.28 ± 0.17	-9.42	<0.001
PLCC	60.49 ± 38.12	86.41 ± 47.74	-8.47	<0.001
PLCR	63.97 ± 266.64	41.15 ± 15.40	1.70	0.089

Table 3. Platelet Count Categories in Normal vs. Raised TLC Groups

PLT Category	Normal TLC (n = 399)	Raised TLC (n = 399)	p-value
<150 $\times 10^3/\mu L$ (Thrombocytopenia)	215 (53.9%)	96 (24.1%)	<0.001
150–400 $\times 10^3/\mu L$ (Normal)	172 (43.1%)	260 (65.2%)	
>400 $\times 10^3/\mu L$ (Thrombocytosis)	12 (3.0%)	43 (10.8%)	

(Figure 1)

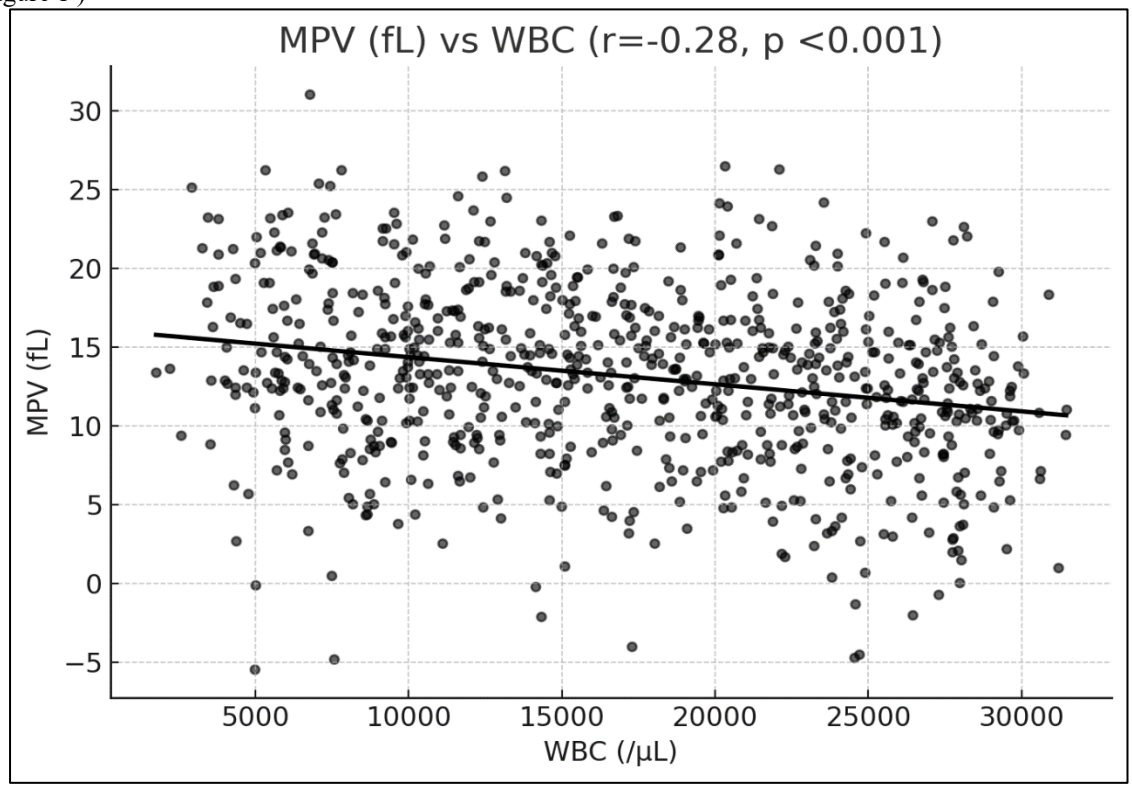


Figure 1: A weak negative correlation **(r = –0.28, p < 0.001) demonstrates that MPV decreases as leukocyte counts rise.**

(Figure 2)

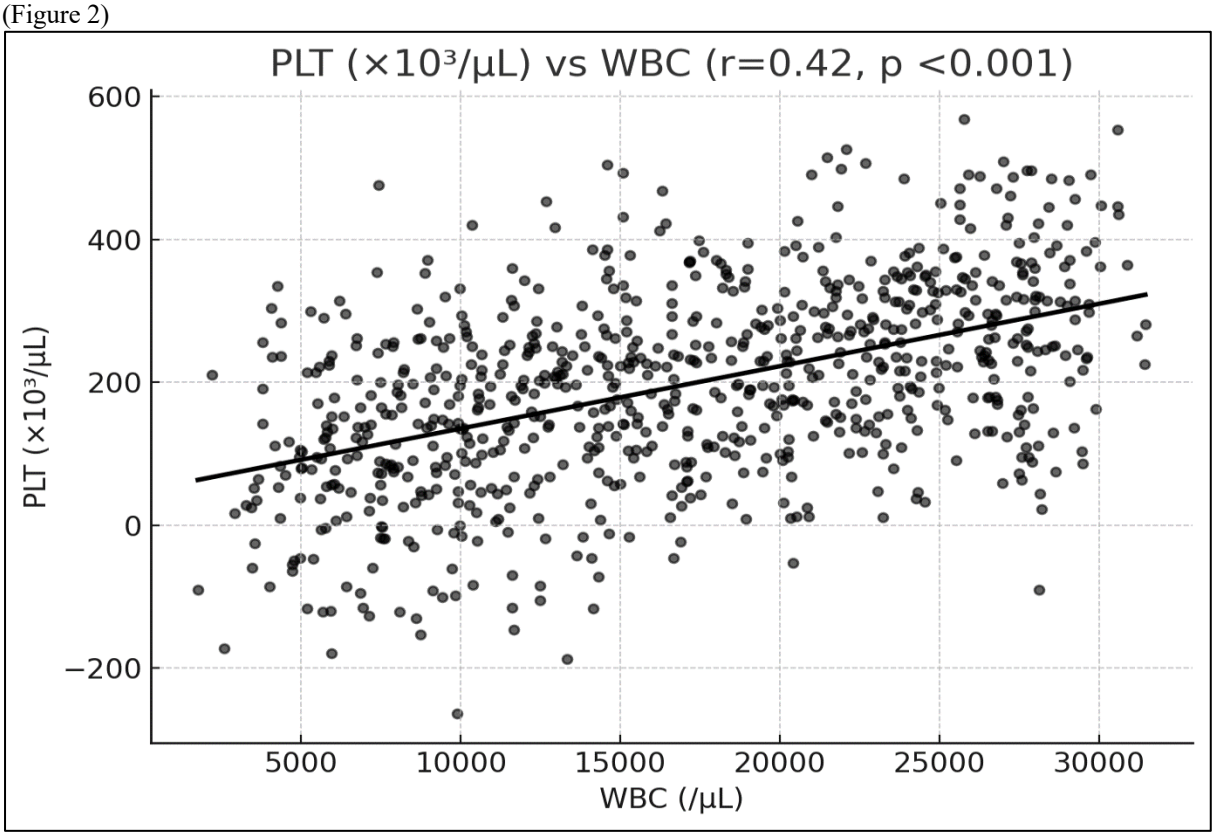


Figure 2: Scatter plot showing platelet count (PLT) vs. WBC count with a fitted trend line. A moderate positive correlation (r = 0.42, p < 0.001) indicates that platelet counts increase with leukocytosis

DISCUSSION

The present study evaluated changes in platelet parameters among patients with leukocytosis compared with individuals with normal total leukocyte counts. The results reveal that there are dramatic changes in platelet indices in relation to the increased leukocyte counts indicating the close interaction between leukopoiesis and thrombopoiesis. In particular, the number of platelets, plateletcrit (PCT), and the platelet large cell count (PLCC) were greatly increased in leukocytosis, and the mean platelet volume (MPV) was decreased. There was no significant difference in platelet large cell ratio (PLCR) and platelet distribution width (PDW). These findings indicate that platelet quantity and morphology alterations accompany leukocytosis and this may give indicators of the underlying hematopoietic processes.Besides the increased number of platelets, the current study also noted that PCT and PLCC were considerably elevated in the leukocytosis group. The two parameters indicate the total amount of platelets and turnover, which supports the hypothesis that bone marrow activity increases during the process of leucocytosis [13]. These indices are comparatively not used in normal practice but can be used as indicators of the hematopoietic activity in inflammatory and reactive states. The positive moderate correlations between PCT and PLCC and the counting of leukocytes in this study put emphasis on their possible use as secondary indicators in assessing bone marrow response. Interestingly, mean platelet volume (MPV) was greatly diminished in patients with leukocytosis. This observation is contrary to the generally accepted belief that larger platelets that are relatively more active in their metabolism are dominant during inflammatory conditions. The next conceivable reason is the premature discharge of platelets in leukocytosis, when the marrow selectively produces and discharges smaller platelets when in high demand [14]. Alternatively, decreased MPV can be an indication of the consumption of bigger, more reactive platelets at inflammatory or infection sites and had left smaller platelets in peripheral blood. Past studies have demonstrated the variability of MPV behavior in disease states in which increases have been reported in cardiovascular and thrombotic states, whereas decreases have been reported in acute infections, anemia, and sepsis. This study did not find a significant difference between groups in terms of Platelet distribution width (PDW) that indicates heterogeneity in platelet size. The same occurred with PLC, which did not indicate statistically significant change, although this change was in the downward direction in the leukocytosis group. These findings indicate that the number of platelets and the total mass of platelets is sensitive, but the size variations in circulating platelets might occur relatively stable during leukocytosis. This interpretation is also supported by the fact that there is no correlation between PDW and the count of leukocytes [15]. Past researches have given conflicting reports on PDW in inflammatory conditions with some indicating that high PDW is associated with platelet activation and others report no interaction [16]. Moreover, the correlations found in this study indicate that platelet indices may be used as surrogate endpoints of bone marrow activity in leukocytosis patients to complement prognosis and monitoring of the disease. Nevertheless, the use of platelet indices should be used with caution because they are sensitive to a variety of factors [17]. Pre-analytical variables can greatly influence the result including sample handling, the type of anticoagulant and the time taken to analysis. The platelet index variability among analyzers also restricts the comparability between various labs as well. Besides, the current research failed to randomize the patients according to the etiology of leukocytosis, which could have caused heterogeneity. Future research must also consider the effect of individual etiologies (e.g. bacterial infections, viral infections, or hematological malignancies) on platelet parameters in a different manner. Longitudinal studies can also help to understand whether the changes in platelet indices have prognostic values regarding the disease progression or response to treatment.

CONCLUSION

It is concluded that leukocytosis is associated with significant alterations in platelet parameters, reflecting the close interplay between leukopoiesis and thrombopoiesis. In this study, patients with raised total leukocyte counts exhibited higher platelet counts, plateletcrit, and platelet large cell counts, accompanied by a reduction in mean platelet volume. These findings suggest that leukocytosis is not only a marker of heightened immune activity but also a state of increased platelet production and altered platelet morphology. Platelet distribution width and platelet large cell ratio, however, showed no significant association, indicating that not all indices are equally affected.

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