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Original Research Article

Evaluation of Platelet Indices and Their Clinical Significance in Thrombocytopenia

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Conflict of interest: Nil

Abstract

Background: Thrombocytopenia is a common hematological disorder with diverse etiologies and variable clinical manifestations. Platelet count alone provides limited information regarding the underlying mechanism and disease severity. Automated platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (P-LCR), may serve as useful adjunctive biomarkers for evaluating thrombocytopenia.

Aim: To evaluate platelet indices and determine their clinical significance in differentiating the underlying mechanism of thrombocytopenia, assessing disease severity, and predicting bleeding manifestations.

Materials & Methods: This hospital-based cross-sectional study was conducted in the Department of Pathology, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Bihar, India, from May 2025 to April 2026. A total of 140 participants were enrolled, comprising 70 patients with thrombocytopenia and 70 age- and sex-matched healthy controls. Platelet count, MPV, PDW, PCT, and P-LCR were measured using an automated hematologyanalyzer. Peripheral blood smear examination was performed for all thrombocytopenic cases. Statistical analysis was carried out using IBM SPSS Statistics version 27.0, with a p-value <0.05 considered statistically significant.

Results: Baseline demographic characteristics were comparable between the two groups ($p>0.05$). Thrombocytopenic patients had significantly lower platelet count (67.84 ± 21.36 vs. 258.42 ± 48.17 $\times 10^3/\mu L$) and PCT (0.082 ± 0.031 vs. 0.241 ± 0.054%), while MPV (11.42 ± 1.52 vs. 9.18 ± 0.84 fL), PDW (17.61 ± 2.31 vs. 11.92 ± 1.47%), and P-LCR (38.96 ± 8.42 vs. 24.38 ± 5.67%) were significantly higher than controls (all $p<0.001$). Patients with peripheral platelet destruction exhibited significantly higher MPV, PDW, and P-LCR than those with decreased platelet production (all $p<0.001$). Bleeding manifestations were associated with lower platelet count and PCT but higher MPV, PDW, and P-LCR (all $p\leq0.005$). Increasing severity of thrombocytopenia showed progressive elevation of MPV, PDW, and P-LCR with a corresponding decline in PCT ($p<0.001$). Dengue fever (25.7%) was the most common etiology, followed by immune thrombocytopenic purpura (20.0%).

Conclusion: Platelet indices, particularly MPV, PDW, and P-LCR, are inexpensive, readily available, and clinically valuable adjunctive biomarkers in thrombocytopenia. They facilitate differentiation between peripheral platelet destruction and decreased platelet production, correlate with disease severity and bleeding manifestations, and complement platelet count in routine clinical evaluation and risk stratification.

Keywords: Thrombocytopenia; Platelet indices; Mean platelet volume; Platelet distribution width; Plateletcrit.

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Introduction

Thrombocytopenia, defined as a platelet count of $<150 \times 10^9/L$, is a common hematological abnormality encountered in both outpatient and inpatient settings. It is associated with an increased risk of bleeding, prolonged hospitalization, and adverse clinical outcomes depending on its severity and underlying etiology. Thrombocytopenia may result from decreased platelet production, increased

peripheral destruction, splenic sequestration, dilutional mechanisms, or a combination of these processes. Since the management and prognosis vary considerably according to the underlying mechanism, prompt and accurate etiological evaluation is essential for appropriate clinical decision-making [1].

Platelet count remains the primary laboratory parameter for diagnosing thrombocytopenia; however, it provides limited information regarding platelet kinetics, bone marrow response, and the underlying pathophysiological mechanism. Patients with similar platelet counts may exhibit markedly different bleeding manifestations and clinical outcomes, suggesting that platelet count alone is insufficient for comprehensive assessment. Consequently, increasing attention has been directed toward platelet indices generated automatically by modern hematologyanalyzers as potential adjunctive biomarkers in the evaluation of thrombocytopenia [2].

Automated complete blood count analyzers routinely report platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (P-LCR). These indices reflect platelet size, size heterogeneity, circulating platelet mass, and the proportion of large platelets, thereby providing indirect information regarding platelet production, activation, and turnover. Because these parameters are inexpensive, rapidly available, reproducible, and obtained without additional laboratory testing, they have attracted considerable interest as practical tools for differentiating the mechanisms of thrombocytopenia and assisting routine clinical evaluation [2,3].

Several recent studies have demonstrated that platelet indices may help distinguish thrombocytopenia caused by increased peripheral platelet destruction from that resulting from decreased bone marrow production. Patients with hyperdestructive thrombocytopenia, such as immune thrombocytopenia, often exhibit higher MPV, PDW, and P-LCR values because of increased release of young, larger platelets from the bone marrow. Conversely, hypoproductive thrombocytopenia is generally associated with relatively lower platelet indices due to impaired megakaryopoiesis. Furthermore, platelet indices have been shown to correlate with disease severity, bleeding manifestations, and response to therapy in selected patient populations, suggesting their potential value as diagnostic and prognostic markers [3,4].

Despite these encouraging findings, the routine clinical application of platelet indices remains limited by inter-analyzer variability, lack of universally accepted reference ranges, and heterogeneity among published studies. Recent systematic reviews have emphasized the need for further validation in well-characterized patient populations before platelet indices can be incorporated into standardized diagnostic algorithms for thrombocytopenia [2,5].

Aim & Objectives

Aim: To evaluate platelet indices and determine their clinical significance in patients with thrombocytopenia, with emphasis on their utility in differentiating the underlying mechanism of thrombocytopenia, assessing disease severity, and predicting bleeding manifestations.

Objectives

- To compare platelet indices (platelet count, MPV, PDW, P-LCR, and PCT) between patients with thrombocytopenia and healthy controls.
- To assess differences in platelet indices between thrombocytopenia caused by peripheral platelet destruction and decreased platelet production.
- To evaluate the association between platelet indices and bleeding manifestations in patients with thrombocytopenia.
- To analyze the relationship between platelet indices and the severity of thrombocytopenia based on platelet count.
- To describe the etiological spectrum of thrombocytopenia among the study population.

Materials & Methods

Study Design: This observational, hospital-based, cross-sectional study was conducted to evaluate platelet indices and their clinical significance in patients with thrombocytopenia.

Study Place: The study was carried out in the Department of Pathology, Bhagwan Mahavir Institute of Medical Sciences (BMIMS), Pawapuri, Nalanda, Bihar, India.

Study Period: The study was conducted over a period of one year from May 2025 to April 2026.

Study Population: A total of 140 participants were enrolled, including 70 patients with thrombocytopenia (cases) and 70 age- and sex-comparable healthy individuals with normal hematological parameters (controls).

Consecutive eligible patients whose blood samples were received in the central clinical laboratory for complete blood count (CBC) analysis during the study period were included.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee of Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, prior to commencement of the study.

Written informed consent was obtained from all participants or from parents/legal guardians in the case of minors. Patient confidentiality was maintained throughout the study, and all procedures were performed in accordance with the ethical principles of the Declaration of Helsinki.

Inclusion Criteria: Participants fulfilling the following criteria were included:

- Patients aged >1 year.
- Platelet count <150 × 10^9/L (thrombocytopenia group).
- Blood samples suitable for automated hematological analysis.
- Healthy individuals with normal complete blood count parameters (control group).

Exclusion Criteria

The following participants were excluded:

- Infants aged <1 year.
- Patients with myelodysplastic syndrome (MDS).
- Patients with autoimmune disorders including systemic lupus erythematosus, type 1 diabetes mellitus, vitiligo, or juvenile rheumatoid arthritis.
- Patients receiving antiplatelet drugs or medications known to induce thrombocytopenia.
- Hemolyzed, clotted, or inadequately collected blood samples.

Methodology: Approximately 2 mL of peripheral venous blood was collected aseptically into K_3-EDTA anticoagulated vacutainer tubes. Samples were analyzed within 1 hour of collection to minimize pre-analytical variation. Complete blood count was performed using a 5-part automated hematologyanalyzer (Pentra ES 60, Horiba Medical, Montpellier, France**)**. The hematological parameters recorded included platelet count (PLT), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet large cell ratio (P-LCR). Peripheral blood smears were prepared for all thrombocytopenic samples, stained using Leishman stain, and examined microscopically to confirm platelet counts, assess platelet morphology, identify platelet clumping, and exclude pseudothrombocytopenia.

Investigations: Investigations included automated complete blood count (CBC), assessment of platelet indices (MPV, PDW, PCT, and P-LCR), peripheral blood smear examination using Leishman stain, and manual microscopic correlation of platelet count and platelet morphology.

Outcome Measures

Primary Outcome Measures

- Comparison of platelet indices (MPV, PDW, PCT, and P-LCR) between thrombocytopenic patients and healthy controls.
- Assessment of the relationship between platelet indices and platelet count.
- Evaluation of the diagnostic and clinical significance of platelet indices in thrombocytopenia.

Secondary Outcome Measures

- Correlation of automated platelet indices with peripheral smear findings.
- Assessment of platelet indices as adjunctive markers for differentiating thrombocytopenic disorders.

Statistical Analysis: Data were entered into Microsoft Excel 365 and analyzed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test. Normally distributed data were expressed as mean ± standard deviation (SD), whereas non-normally distributed data were presented as median (interquartile range [IQR]).

Categorical variables were summarized as frequency and percentage. Comparisons between two independent groups were performed using the independent-samples Student's t-test for normally distributed variables or the Mann–Whitney U test for non-parametric data. Comparisons among more than two groups were carried out using one-way analysis of variance (ANOVA) followed by Bonferroni post hoc analysis for pairwise comparisons.

Associations between categorical variables were evaluated using the Chi-square test or Fisher's exact test, as appropriate. Correlations between platelet indices and platelet count were assessed using Pearson's correlation coefficient for normally distributed variables or Spearman's rank correlation coefficient for non-parametric variables. A two-tailed P-value <0.05 was considered statistically significant throughout the analysis.

Results

Table 1: Baseline Demographic Characteristics of the Study Population (N = 140)

Characteristics	Variable	Thrombocytopenia (n = 70)	Controls (n = 70)	Total (N = 140)	p-value*
Age (years), Mean ± SD	-	41.82 ± 16.37	39.91 ± 15.84	40.87 ± 16.09	0.482
Age Group, n (%)	18–30 years	18 (25.7)	20 (28.6)	38 (27.1)	0.918
	31–45 years	20 (28.6)	19 (27.1)	39 (27.9)	
	46–60 years	17 (24.3)	16 (22.9)	33 (23.6)	
	>60 years	15 (21.4)	15 (21.4)	30 (21.4)	
Gender, n (%)	Male	40 (57.1)	38 (54.3)	78 (55.7)	0.734
	Female	30 (42.9)	32 (45.7)	62 (44.3)	
Residence, n (%)	Rural	43 (61.4)	40 (57.1)	83 (59.3)	0.589
	Urban	27 (38.6)	30 (42.9)	57 (40.7)	
BMI (kg/m²), Mean ± SD	-	23.41 ± 3.84	23.97 ± 3.61	23.69 ± 3.72	0.372
Smoking Status, n (%)	Smoker	14 (20.0)	12 (17.1)	26 (18.6)	0.661
	Non-smoker	56 (80.0)	58 (82.9)	114 (81.4)	
Alcohol Consumption, n (%)	Yes	12 (17.1)	10 (14.3)	22 (15.7)	0.512
	No	58 (82.9)	60 (85.7)	118 (84.3)	

BMI: Body Mass Index

Table 1 present the mean age was comparable between the thrombocytopenia group (41.82 ± 16.37 years) and controls (39.91 ± 15.84 years), with no statistically significant difference (p = 0.482).

The distribution of participants across age groups was similar in both groups (p = 0.918). The largest proportion belonged to the 31–45 years age group (28.6% vs. 27.1%), followed by 18–30 years (25.7% vs. 28.6%), 46–60 years (24.3% vs. 22.9%), and >60 years (21.4% in both groups). There was no significant difference in gender distribution between the groups (p = 0.734), with males constituting 57.1% of the thrombocytopenia group and 54.3% of the control group. Similarly, the proportion of participants from rural and urban areas was comparable (p = 0.589).

The mean BMI did not differ significantly between thrombocytopenia patients (23.41 ± 3.84 kg/m²) and controls (23.97 ± 3.61 kg/m²) (p = 0.372). Likewise, smoking status (20.0% vs. 17.1%; p = 0.661) and alcohol consumption (17.1% vs. 14.3%; p = 0.512) were comparable between the two groups.

Table 2: Comparison of Platelet Indices between Thrombocytopenia Cases and Controls (N = 140)

Platelet Parameter	Thrombocytopenia (n = 70), Mean ± SD	Controls (n = 70) Mean ± SD	t-value	p-value
Platelet Count (×10³/μL)	67.84 ± 21.36	258.42 ± 48.17	-31.96	<0.001*
Mean Platelet Volume (MPV, fL)	11.42 ± 1.52	9.18 ± 0.84	10.89	<0.001*
Platelet Distribution Width (PDW, %)	17.61 ± 2.31	11.92 ± 1.47	17.60	<0.001*
Platelet Large Cell Ratio (P-LCR, %)	38.96 ± 8.42	24.38 ± 5.67	12.05	<0.001*
Plateletcrit (PCT, %)	0.082 ± 0.031	0.241 ± 0.054	-21.51	<0.001*

*Statistically significant (p < 0.001).

Table 2 demonstrates the mean platelet count was markedly lower in the thrombocytopenia group (67.84 ± 21.36 ×10³/μL) compared with controls (258.42 ± 48.17 ×10³/μL), and this difference was highly significant (t = −31.96, p < 0.001).

Conversely, the mean platelet volume (MPV) was significantly higher among thrombocytopenia patients (11.42 ± 1.52 fL) than controls (9.18 ± 0.84 fL) (t = 10.89, p < 0.001). Similarly, the platelet distribution width (PDW) was significantly elevated in cases (17.61 ± 2.31%) compared with controls (11.92 ± 1.47%) (t = 17.60, p < 0.001). The platelet large cell ratio (P-LCR) was also significantly greater in the thrombocytopenia group (38.96 ± 8.42%) than in controls (24.38 ± 5.67%) (t = 12.05, p < 0.001).

In contrast, plateletcrit (PCT) was significantly reduced in thrombocytopenia patients (0.082 ± 0.031%) compared with healthy controls (0.241 ± 0.054%) (t = −21.51, p < 0.001).

Table 3: Comparison of Platelet Indices According to the Mechanism of Thrombocytopenia (n = 70)

Platelet Parameter	Peripheral Destruction (n = 40) Mean ± SD	Decreased Production (n = 30) Mean ± SD	t-value	p-value
Platelet Count (×10³/μL)	69.85 ± 19.82	65.17 ± 23.48	0.91	0.366
Mean Platelet Volume (MPV, fL)	12.24 ± 1.18	10.31 ± 1.12	6.89	<0.001*
Platelet Distribution Width (PDW, %)	18.63 ± 1.87	16.24 ± 1.69	5.51	<0.001*
Platelet Large Cell Ratio (P-LCR, %)	42.78 ± 6.94	33.87 ± 5.83	5.72	<0.001*
Plateletcrit (PCT, %)	0.085 ± 0.029	0.078 ± 0.034	0.93	0.355

*Statistically significant ($p < 0.001$).

Table 3 present the mean platelet count was slightly higher in the peripheral destruction group (69.85 ± 19.82 ×10³/μL) than in the decreased production group (65.17 ± 23.48 ×10³/μL); however, the difference was not statistically significant ($t = 0.91$, $p = 0.366$). The mean platelet volume (MPV) was significantly higher in patients with peripheral destruction (12.24 ± 1.18 fL) compared with those with decreased production (10.31 ± 1.12 fL) ($t = 6.89$, $p < 0.001$). Likewise, platelet distribution width (PDW) was significantly elevated in the peripheral destruction group (18.63 ± 1.87%) versus the decreased production group (16.24 ± 1.69%) ($t = 5.51$, $p < 0.001$). Similarly, the platelet large cell ratio (P-LCR) was significantly greater in patients with peripheral destruction (42.78 ± 6.94%) than in those with decreased production (33.87 ± 5.83%) ($t = 5.72$, $p < 0.001$). In contrast, plateletcrit (PCT) showed no significant difference between the two groups (0.085 ± 0.029% vs. 0.078 ± 0.034%; $t = 0.93$, $p = 0.355$).

Table 4: Association of Platelet Indices with Bleeding Manifestations among Thrombocytopenia Cases (n = 70)

Platelet Parameter	Bleeding Present (n = 32) Mean ± SD	Bleeding Absent (n = 38) Mean ± SD	t-value	p-value
Platelet Count (×10³/μL)	49.87 ± 15.63	82.98 ± 18.74	-8.06	<0.001*
Mean Platelet Volume (MPV, fL)	12.08 ± 1.34	10.86 ± 1.29	3.88	<0.001*
Platelet Distribution Width (PDW, %)	18.42 ± 1.98	16.93 ± 2.11	3.04	0.003*
Platelet Large Cell Ratio (P-LCR, %)	41.82 ± 7.23	36.57 ± 7.86	2.88	0.005*
Plateletcrit (PCT, %)	0.061 ± 0.022	0.099 ± 0.027	-6.40	<0.001*

*Statistically significant ($p < 0.01$).

Table 4 show that Patients with bleeding had a significantly lower mean platelet count (49.87 ± 15.63 ×10³/μL) than those without bleeding (82.98 ± 18.74 ×10³/μL) ($t = −8.06$, $p < 0.001$).

The mean platelet volume (MPV) was significantly higher in patients with bleeding (12.08 ± 1.34 fL) compared with those without bleeding (10.86 ± 1.29 fL) ($t = 3.88$, $p < 0.001$). Similarly, platelet distribution width (PDW) was significantly increased in the bleeding group (18.42 ± 1.98% vs. 16.93 ± 2.11%) ($t = 3.04$, $p = 0.003$), while the platelet large cell ratio (P-LCR) was also significantly higher (41.82 ± 7.23% vs. 36.57 ± 7.86%) ($t = 2.88$, $p = 0.005$).

Conversely, plateletcrit (PCT) was significantly lower among patients with bleeding (0.061 ± 0.022%) than those without bleeding (0.099 ± 0.027%) ($t = −6.40$, $p < 0.001$).

Table 5: Etiological Distribution of Thrombocytopenia among Cases (n = 70)

Etiology	Frequency (n)	Percentage (%)
Dengue fever	18	25.7
Immune thrombocytopenic purpura (ITP)	14	20.0
Sepsis	8	11.4
Malaria	5	7.1
Chronic liver disease / Hypersplenism	4	5.7
Megaloblastic anemia	7	10.0
Aplastic anemia	5	7.1
Acute leukemia	5	7.1
Chemotherapy-induced marrow suppression	2	2.9
Myelodysplastic syndrome	2	2.9
Total	70	100.0

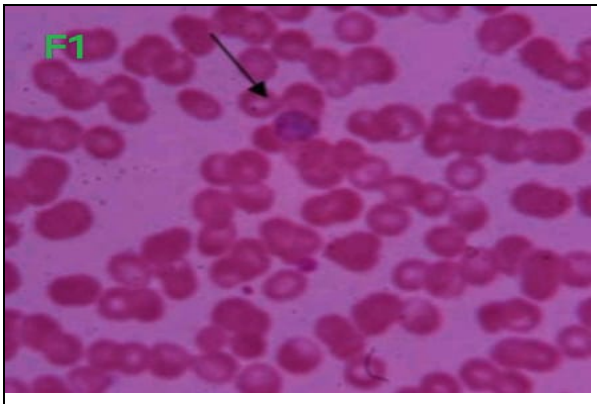
Table 5 show that Dengue fever was the most common cause, accounting for 18 cases (25.7%), followed by immune thrombocytopenic purpura (ITP) in 14 patients (20.0%). Sepsis was identified in 8 cases (11.4%), while megaloblastic anemia accounted for 7 cases (10.0%). Malaria, aplastic anemia, and acute leukemia each contributed 5 cases (7.1%), whereas chronic liver disease/hypersplenism was observed in 4 patients (5.7%). Less frequent etiologies included chemotherapy-induced marrow suppression and myelodys plastic syndrome, each accounting for 2 cases (2.9%).

Table 6: Association of Platelet Indices According to Different Levels of Platelet Count among Thrombocytopenic Patients (n = 70)

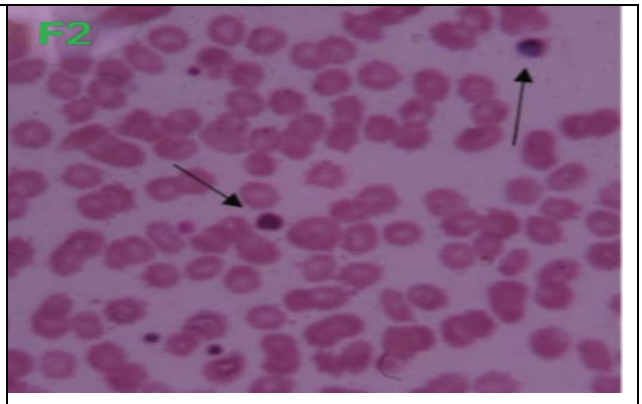
Platelet Parameter	Severe Thrombocytopenia (<20 ×10³/µL) (n = 12) Mean ± SD	Moderate Thrombocytopenia (20–50 ×10³/µL) (n = 24) Mean ± SD	Mild Thrombocytopenia (51–100 ×10³/µL) (n = 34) Mean ± SD	p-value
Platelet Count (×10³/µL)	14.83 ± 3.92	37.54 ± 8.17	76.91 ± 13.48	<0.001*
Mean Platelet Volume (MPV, fL)	12.82 ± 1.11	11.71 ± 1.19	10.78 ± 1.18	<0.001*
Platelet Distribution Width (PDW, %)	19.18 ± 1.52	17.93 ± 1.74	16.71 ± 1.86	<0.001*
Platelet Large Cell Ratio (P-LCR, %)	45.87 ± 6.32	40.21 ± 6.81	35.92 ± 6.49	<0.001*
Plateletcrit (PCT, %)	0.031 ± 0.011	0.061 ± 0.017	0.108 ± 0.023	<0.001*

*Statistically significant ($p < 0.001$).

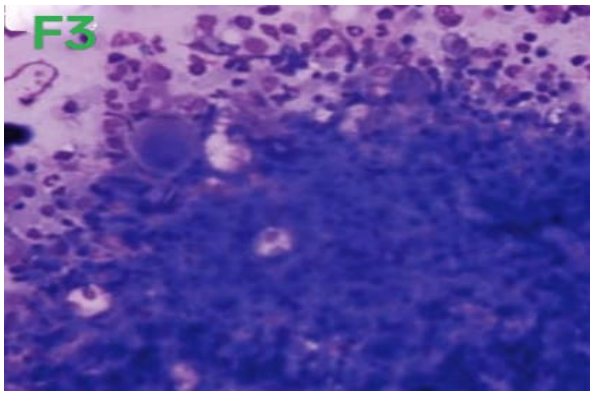
Table 6 show the mean platelet count increased significantly from the severe (14.83 ± 3.92 ×10³/µL) to the moderate (37.54 ± 8.17 ×10³/µL) and mild thrombocytopenia groups (76.91 ± 13.48 ×10³/µL) ($p < 0.001$). In contrast, the mean platelet volume (MPV) showed a significant inverse relationship with platelet count, with the highest values observed in severe thrombocytopenia (12.82 ± 1.11 fL), followed by moderate (11.71 ± 1.19 fL) and mild thrombocytopenia (10.78 ± 1.18 fL) ($p < 0.001$). Similarly, platelet distribution width (PDW) was highest in severe thrombocytopenia (19.18 ± 1.52%) and progressively decreased in the moderate (17.93 ± 1.74%) and mild groups (16.71 ± 1.86%) ($p < 0.001$). The platelet large cell ratio (P-LCR) also demonstrated a significant declining trend from severe (45.87 ± 6.32%) to moderate (40.21 ± 6.81%) and mild thrombocytopenia (35.92 ± 6.49%) ($p < 0.001$). Conversely, plateletcrit (PCT) increased significantly with increasing platelet count, rising from 0.031 ± 0.011% in severe thrombocytopenia to 0.061 ± 0.017% in moderate and 0.108 ± 0.023% in mild thrombocytopenia ($p < 0.001$).



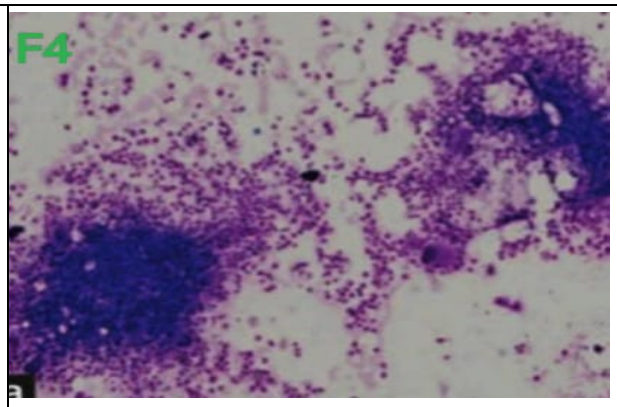
F1: Peripheral smear showing reduced platelet count with *Plasmodium vivax* schizont form (Leishman stain).



F2: Peripheral smear showing giant platelets (arrow) in dengue fever (Leishman stain).



F3: Bone marrow aspirate showing increased megakaryocytes in immune thrombocytopenic purpura (ITP) (1000×, Giemsa stain).



F4: Bone marrow aspirate showing increased megakaryocytes in immune thrombocytopenic purpura (ITP) (40×, Giemsa stain).

Discussion

In present study, no statistically significant differences were observed with respect to age, age-group distribution, sex, body mass index, smoking status, alcohol consumption, or place of residence (all $p>0.05$). These findings indicate that both groups were well matched, thereby minimizing the influence of demographic confounding factors on platelet indices. Similar observations have been reported by Reddy et al. (2025), who demonstrated that demographic variables had no significant influence on platelet indices when comparing thrombocytopenic patients with healthy individuals and concluded that alterations in platelet indices primarily reflected the underlying disease process rather than baseline patient characteristics [6]. Likewise, Kumar et al. (2024) observed no significant differences in age or sex distribution between patients with hypoproductive and hyperdestructive thrombocytopenia, supporting the validity of comparing platelet indices independent of demographic characteristics [7]. These findings strengthen the internal validity of the present study and suggest that the observed alterations in platelet indices are attributable to thrombocytopenia rather than demographic variation.

The present study demonstrated markedly reduced platelet count and plateletcrit (PCT), accompanied by significantly increased MPV, PDW, and P-LCR among thrombocytopenia patients compared with healthy controls (all $p<0.001$). These findings indicate increased platelet turnover and enhanced release of larger, immature platelets into the peripheral circulation, particularly in conditions associated with accelerated platelet destruction. Comparable findings were reported by Sharma et al. (2024), who demonstrated significantly higher MPV, PDW, and P-LCR values among thrombocytopenic patients than healthy controls and suggested that these indices represent useful surrogate markers of platelet regeneration and activation [8]. Similarly, Rao et al. (2024) observed that MPV and PDW were consistently elevated in thrombocytopenic disorders, whereas PCT showed a significant reduction because of decreased circulating platelet mass [9].

A major objective of the present study was to evaluate the utility of platelet indices in differentiating peripheral platelet destruction from decreased platelet production. Although platelet count and PCT did not differ significantly between the two groups, MPV, PDW, and P-LCR were significantly higher in patients with peripheral destruction (all $p<0.001$). These findings are consistent with those reported by Singh et al. (2024), who demonstrated significantly higher MPV and PDW values in hyperdestructive thrombocytopenia compared with hypoproductive thrombocytopenia and concluded that these parameters could reduce the need for invasive bone marrow examination [10]. Similarly, Patel et al. (2025) found that P-LCR showed excellent discriminatory ability for identifying peripheral platelet destruction because increased platelet turnover stimulates release of larger, immature platelets from the bone marrow [11].

The present study further demonstrated that thrombocytopenic patients with bleeding manifestations had significantly lower platelet count and PCT but significantly higher MPV, PDW, and P-LCR than patients without bleeding. These findings indicate that bleeding severity depends not only on platelet number but also on platelet morphology and turnover. Similar findings were reported by Ahmed et al. (2025), who demonstrated that elevated MPV and PDW were independently associated with clinically significant bleeding among thrombocytopenic patients and suggested that these indices improve bleeding risk stratification [12]. Likewise, Perera et al. (2025) observed that patients with higher MPV and P-LCR experienced more frequent mucocutaneous bleeding despite comparable platelet counts, emphasizing that platelet indices reflect platelet

functional activity in addition to platelet quantity [13].

More recently, Fernandez et al. (2025) reported that combining platelet count with MPV, PDW, and P-LCR significantly improved prediction of bleeding risk compared with platelet count alone, supporting the concept that platelet indices provide clinically relevant prognostic information [14]. The findings of the present study are therefore in agreement with current evidence that platelet indices should be interpreted as complementary biomarkers rather than isolated laboratory parameters.

The present study identified dengue fever (25.7%) as the leading cause of thrombocytopenia, followed by immune thrombocytopenic purpura (20.0%), sepsis, megaloblastic anemia, malaria, aplastic anemia, acute leukemia, chronic liver disease with hypersplenism, chemotherapy-induced marrow suppression, and myelodysplastic syndrome. This distribution reflects the broad spectrum of infectious, immune-mediated, and bone marrow disorders encountered in tertiary care hospitals. The predominance of dengue fever is consistent with the findings of Rashid et al. (2024), who reported that dengue-associated thrombocytopenia results from transient bone marrow suppression, immune-mediated platelet destruction, and increased peripheral platelet consumption, leading to significant alterations in platelet indices [15]. Similarly, Lee et al. (2025) demonstrated that platelet indices are useful in identifying distinct pathogenic mechanisms and predicting bleeding risk in dengue patients [16]. Guo et al. (2025) further reported that serial assessment of platelet count together with platelet indices provides valuable information regarding disease progression and recovery in dengue infection [17].

Immune thrombocytopenic purpura was the second most common etiology in the present study. This finding agrees with Nathan et al. (2024), who demonstrated that immune-mediated peripheral platelet destruction is associated with increased MPV and P-LCR due to enhanced release of large immature platelets from the bone marrow, highlighting the diagnostic utility of platelet indices in differentiating hyperdestructive thrombocytopenia from hypoproductive disorders [3]. The present study demonstrated that increasing severity of thrombocytopenia was associated with significantly higher MPV, PDW, and P-LCR, whereas plateletcrit (PCT) progressively decreased. These findings indicate enhanced platelet turnover and compensatory thrombopoiesis in patients with severe thrombocytopenia. Comparable findings were reported by Sontakke et al. (2024), who observed significant increases in MPV and PDW with worsening thrombocytopenia among dengue patients and concluded that platelet indices are useful markers for assessing disease severity [18]. Likewise, Guo et al. (2025) demonstrated an inverse relationship between platelet count and MPV/P-LCR, while PCT showed a positive correlation with platelet count, suggesting that combined interpretation of platelet count and platelet indices improves assessment of disease progression [17]. Furthermore, Lee et al. (2025) reported that elevated platelet indices were associated with increased bleeding risk and reflected enhanced platelet activation and regeneration in severe thrombocytopenia [16].

Limitations of the Study

- The study was conducted at a single tertiary care centre, which may limit the generalizability of the findings.
- The relatively small sample size may have reduced the statistical power for subgroup analyses.
- The cross-sectional design precluded assessment of temporal changes in platelet indices and clinical outcomes.
- Platelet indices were measured at a single time point without serial follow-up.
- Bone marrow examination was not performed in all patients; therefore, etiological classification was based on available clinical and laboratory findings.
- The prognostic value of platelet indices for long-term outcomes could not be evaluated.

Conclusion

Platelet indices differed significantly between thrombocytopenia patients and healthy controls, demonstrating their potential clinical utility as adjunctive hematological markers. Patients with thrombocytopenia exhibited significantly higher MPV, PDW, and P-LCR and significantly lower PCT compared with controls. Among thrombocytopenic patients, MPV, PDW, and P-LCR were significantly higher in peripheral platelet destruction than in decreased platelet production, indicating their usefulness in differentiating the underlying mechanism of thrombocytopenia. Patients with bleeding manifestations had significantly lower platelet counts and PCT, together with higher MPV, PDW, and P-LCR, suggesting that these indices are associated with an increased risk of bleeding. Furthermore, worsening thrombocytopenia was associated with progressively higher MPV, PDW, and P-LCR and lower PCT, reflecting increasing disease severity. Dengue fever was the most common etiology, followed by immune thrombocytopenic purpura. Overall, platelet indices are inexpensive, readily available, and clinically valuable parameters that complement platelet count in the evaluation of thrombocytopenia, facilitate etiological differentiation, and aid in risk stratification and clinical decision-making.

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