

Diagnostic Performance of Platelet Volume Indices in Thrombocytopenia: A Single-Center Observational Study in Central India

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Abstract

Background: Platelet indices obtained from automated hematology analyzers may complement the evaluation of thrombocytopenia.

Aim and objectives: The objective of this study is to assess mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) in relation to thrombocytopenia severity, clinical etiology, and underlying mechanism.

Materials and methods: This observational cross-sectional study with prospective data collection was conducted over 18 months in the Department of Pathology at Amaltas Institute of Medical Sciences, Dewas, India. Convenience sampling yielded 324 patients with platelet counts below $150 \times 10^9/L$. EDTA-anticoagulated blood samples were analyzed within two hours using an Erba Elite 580 analyzer (Erba Diagnostics Mannheim GmbH, Mannheim, Germany), with peripheral smear assessment. Platelet indices were compared across severity and mechanism categories and evaluated using correlation and receiver operating characteristic analyses.

Results: The mean age was 44.23 ± 12.92 years; 227 (70.1%) patients were male. Mild, moderate, and severe thrombocytopenia occurred in 211 (65.1%), 96 (29.6%), and 17 (5.2%) patients, respectively. PDW increased, and PCT decreased across severity categories (both $p < 0.001$), whereas MPV showed no significant difference ($p = 0.94$). Platelet count correlated positively with PCT ($r = 0.91$) and inversely with PDW ($r = -0.31$). Reported areas under the curve were 0.82 for PDW, 0.71 for PCT, and 0.58 for MPV.

Conclusions: PDW showed the greatest reported discrimination between mechanism categories, while PCT closely reflected platelet count. The findings warrant further evaluation of platelet indices alongside clinical and peripheral smear assessment; their effect on diagnostic decisions and clinical outcomes was not assessed. Verification of mechanism classification and diagnostic analyses, followed by external validation, is required before clinical application.

Categories: Medical Education, Pathology, Public Health

Keywords: complete blood count, hyperdestructive thrombocytopenia, hypoproduative thrombocytopenia, immune thrombocytopenia, mean platelet volume, plateletcrit, platelet distribution width, platelet volume indices, thrombocytopenia

Introduction

Thrombocytopenia, defined as a peripheral blood platelet count below $150 \times 10^9/L$, is a common hematological finding with diverse underlying causes. It may result from reduced platelet production, increased peripheral destruction or consumption, splenic sequestration, or hemodilution. Apparent thrombocytopenia may also arise from platelet clumping and should be excluded during the initial evaluation. Identifying the underlying cause is essential because the clinical implications and treatment differ substantially between these conditions [1].

The evaluation of thrombocytopenia relies on clinical history, physical examination, complete blood count, and peripheral blood smear assessment, followed by investigations guided by the suspected diagnosis [1]. Bone marrow examination can help identify impaired thrombopoiesis and marrow disorders but is not required in every patient. For example, international consensus recommendations indicate that bone marrow examination is unnecessary at initial presentation when isolated thrombocytopenia is accompanied by otherwise typical clinical and peripheral smear findings of immune thrombocytopenia (ITP) [2]. Accessible laboratory parameters that complement this evaluation may therefore support the selection of further investigations.

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Automated hematology analyzers provide platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT), alongside the platelet count. MPV describes average platelet size, PDW reflects variation in platelet size, and PCT estimates the proportion of blood volume occupied by platelets [3]. These parameters may provide additional information about platelet production and turnover. Increased peripheral destruction can stimulate the release of larger platelets, whereas impaired marrow production may produce a different platelet size distribution. Studies have demonstrated the potential of MPV and PDW to help distinguish ITP from hypoproliferative thrombocytopenia, although their diagnostic performance varies between populations [4,5].

The available evidence supports cautious interpretation of these indices. A systematic review and meta-analysis reported pooled sensitivity and specificity of 76% and 79%, respectively, for MPV in differentiating ITP from hypoproliferative thrombocytopenia [6]. These findings support its use as an adjunct rather than a standalone diagnostic test. Furthermore, anticoagulant exposure, the interval between collection and analysis, and differences in analyzer technology can influence MPV measurements [7]. These estimates provide background evidence that MPV may help distinguish selected causes of thrombocytopenia and support further investigation of platelet indices. However, they were derived from comparisons of ITP and hypoproliferative thrombocytopenia, whereas the present study included a broader, more heterogeneous clinical population. Differences in disease spectrum, reference classification, analyzer methods, and diagnostic thresholds limit direct comparison. The published estimates therefore provide a rationale for this study rather than a benchmark for its expected diagnostic performance.

The present study aimed to evaluate MPV, PDW, and PCT in adult patients with thrombocytopenia at a tertiary care hospital in Central India. Specifically, it examined their associations with platelet count, thrombocytopenia severity, and clinical etiology, and assessed their ability to distinguish clinically categorized hypoproliferative and hyperdestructive mechanisms. The study sought to clarify their potential contribution to routine diagnostic assessment.

Materials And Methods

Study design and setting

This hospital-based cross-sectional observational study used prospective data collection and was conducted in the Department of Pathology at Amaltas Institute of Medical Sciences, Dewas, Madhya Pradesh, India, over an 18-month period from February 2024 to August 2025. The department received samples for routine hematological investigations from inpatient and outpatient services, including Medicine and Surgery.

Participants were recruited, and data were collected prospectively during the study period. The study was cross-sectional because platelet indices and their associations with clinical characteristics were assessed at the index evaluation, without serial monitoring of platelet indices. Thus, prospective refers to the method of recruitment and data collection rather than a longitudinal study design.

Study population and sampling

Convenience sampling was used to recruit eligible patients whose blood samples were submitted to the Department of Pathology for routine hematological examination. Thrombocytopenia was defined as a platelet count below $150 \times 10^9/L$ [1]. Adults aged 18 years or older with a platelet count below $150 \times 10^9/L$ were eligible for inclusion.

Patients with known leukemia, aplastic anemia, or bone marrow failure syndromes were excluded. Other exclusion criteria were treatment with antiplatelet or cytotoxic drugs, disseminated intravascular coagulation or other systemic consumptive disorders, and hemolyzed, clotted, or inadequate samples unsuitable for analysis. The exclusions were intended to limit heterogeneity related to established marrow disorders, treatment exposure, and systemic consumptive conditions that could complicate interpretation of platelet indices. Hemolyzed, clotted, or inadequate samples were excluded to ensure analytical suitability. These restrictions defined a selected clinical population rather than the full spectrum of patients with thrombocytopenia.

Participant screening, exclusions with reasons, enrollment, and inclusion in the analysis are summarized in Figure 1, following STROBE reporting guidance [8].

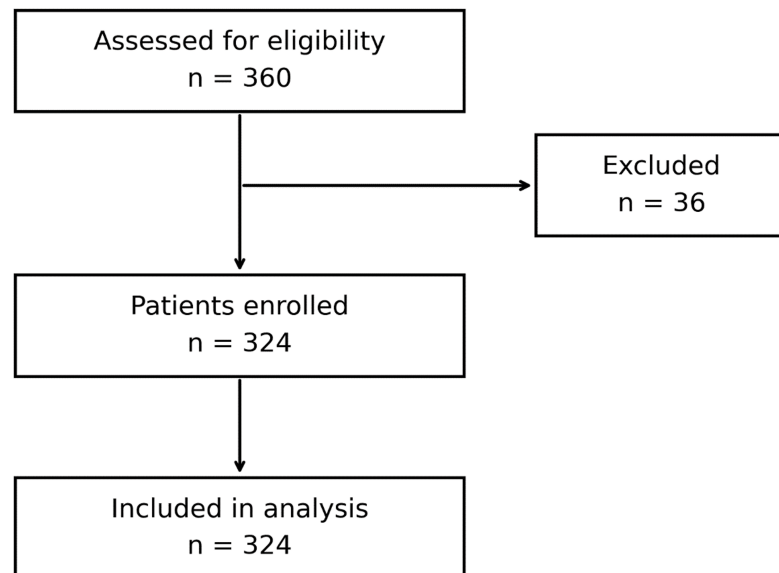


FIGURE 1: Participant flow diagram showing eligibility assessment, enrollment, and inclusion in the analysis

Sample size estimation

A sample-size assessment was performed using the single-proportion formula,

$$n = \frac{Z^2 p (1 - p)}{d^2}$$

Here, n represents the required sample size, Z is the standard normal deviate of 1.96 for a 95% confidence level, p is the anticipated proportion of platelet abnormalities based on previous literature, and d is the absolute precision of 0.05. Using an anticipated proportion of 29.8%, based on 149 new-onset thrombocytopenia cases among 500 ICU patients reported by Mehta et al. [9], a 95% confidence level ($Z = 1.96$), and an absolute precision of 5% ($d = 0.05$), the calculated sample size was 321.46, rounded up to a minimum required sample of 322 patients. The actual number of patients enrolled and included in the analysis was 324.

Clinical data collection

Demographic and clinical information was obtained from laboratory requisition forms and hospital records using a structured study proforma. Recorded information included age, sex, presenting complaints, duration of illness, relevant medical history, medication use, transfusion history, clinical examination findings, and available laboratory investigations. Platelet indices were examined in relation to thrombocytopenia severity, clinical etiology, and the reported mechanistic categories of hypoproliferative and hyperdestructive thrombocytopenia.

Blood collection and automated analysis

Approximately 2 mL of venous blood was collected aseptically into ethylenediaminetetraacetic acid (EDTA) tubes. Samples were analyzed within two hours of collection using an Erba Elite 580 five-part differential hematology analyzer (Erba Diagnostics Mannheim GmbH, Mannheim, Germany). The recorded parameters were platelet count, MPV, PDW, and PCT. Platelet count was expressed as $\times 10^9/L$, MPV in femtoliters, and PCT as a percentage.

MPV describes average platelet size, PDW reflects variability in platelet size, and PCT estimates the proportion of blood volume occupied by platelets [3]. Processing within two hours limited delays that could influence platelet-volume measurements; EDTA exposure and the interval before analysis can affect MPV [7].

Peripheral blood smear examination and quality control

A Leishman-stained peripheral blood smear was examined for each patient to assess platelet number and

morphology, identify associated red and white blood cell abnormalities, and distinguish true thrombocytopenia from pseudothrombocytopenia. Smears were independently evaluated by two observers using an Olympus CX23 binocular microscope (Olympus Corporation, Tokyo, Japan), with $\times 10$, $\times 40$, and $\times 100$ oil-immersion objectives. Slides with doubtful morphology were reviewed by senior pathologists.

Daily analyzer checks were performed using manufacturer-recommended controls. Quality-control procedures included routine instrument maintenance, monitoring of reagent expiry dates, and verification of automated findings by peripheral blood smear examination. Duplicate testing was performed in approximately 10% of cases as an internal quality-control procedure. No formal statistical assessment of repeatability or agreement was performed.

Classification of thrombocytopenia

For the study analysis, thrombocytopenia was categorized as mild (platelet count, $100\text{--}149 \times 10^9/\text{L}$), moderate ($50\text{--}99 \times 10^9/\text{L}$), or severe ($<50 \times 10^9/\text{L}$). MPV, PDW, and PCT were compared across these severity categories and clinical diagnoses. The underlying mechanisms of thrombocytopenia were considered within four broad categories: decreased platelet production, increased platelet destruction, platelet sequestration, and dilution. Decreased production may result from bone marrow failure or suppression, nutritional deficiencies, infections, alcohol use, or inherited disorders. Increased destruction may involve immune or non-immune processes. Sequestration occurs through increased platelet pooling in an enlarged spleen, whereas dilution may follow massive transfusion or fluid replacement. Clinical etiological categories and mechanism categories were treated as distinct descriptive concepts. Sepsis and postoperative status identify clinical contexts and do not, by themselves, establish hypoproducer or hyperdestructive thrombocytopenia. The operational criteria used for individual mechanism assignments were insufficiently documented to establish a reproducible reference classification.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics for Windows, Version 20 (Released 2011; IBM Corp., Armonk, New York, United States). Continuous variables were summarized as means and standard deviations, and categorical variables as frequencies and percentages. Independent-samples Student's *t*-tests were used for comparisons between two groups. The reported analyses also included chi-square tests for categorical associations, one-way analysis of variance for comparisons across severity groups, and Pearson's correlation coefficients for relationships between platelet count and individual platelet indices. A *p*-value below 0.05 was considered statistically significant.

Receiver operating characteristic (ROC) analysis was used to assess the discrimination of MPV, PDW, and PCT between the assigned hypoproducer and hyperdestructive thrombocytopenia categories. AUC, sensitivity, and specificity estimates were reported for each index.

Ethical considerations

The study protocol was approved by the Institutional Ethical Committee of Amaltas Institute of Medical Sciences, Dewas, on 15 February 2024. The committee's registration numbers are ECR/1145/Inst/MP/2018/RR-22 (CDSCO) and ECNEW/INST/2022/2692 (NECRBHR, Department of Health Research). Scientific approval was granted by the Scientific Research Committee on 15 February 2024.

The study used samples obtained for routine diagnostic investigations, and no additional invasive procedure was performed solely for research. Written informed consent was obtained from all participants before enrollment. Patient information was anonymized before analysis, and confidentiality was maintained. The study followed the ethical principles of the Declaration of Helsinki, 2013 revision [10].

Results

A total of 324 patients with thrombocytopenia were evaluated. The enrolled patients ranged in age from 18 to 68 years. The cohort comprised predominantly male patients, and the largest age group was 41–50 years. Baseline demographic and hematological characteristics are presented in Table 1.

Characteristic	Value
Age, years, mean $\pm$ SD	44.23 $\pm$ 12.92
Age group, years	n (%)
18–30	62 (19.1)
31–40	78 (24.1)
41–50	86 (26.5)
51–60	63 (19.4)
61–68	35 (10.8)
Sex	n (%)
Male	227 (70.1)
Female	97 (29.9)
Hematological parameter	Mean $\pm$ SD (range)
Platelet count, $\times 10^9/L$	107.6 $\pm$ 32.8 (16–149)
MPV, fL	10.81 $\pm$ 1.96 (7.2–16.8)
PDW-SD, fL	14.05 $\pm$ 3.94 (6.3–23.8)
PCT, %	0.112 $\pm$ 0.035 (0.017–0.203)

TABLE 1: Baseline characteristics of the study population

Values are presented as n (%) or mean $\pm$ standard deviation, as applicable. MPV: mean platelet volume; PDW: platelet distribution width; PCT: plateletcrit; SD: standard deviation.

Mild thrombocytopenia was the most frequent severity category. PDW increased and PCT decreased across progressively more severe categories, while MPV showed no consistent trend (Table 2).

Severity and platelet count, $\times 10^9/L$	Patients, n (%)	MPV, fL	PDW	PCT, %
Mild, 100–149	211 (65.1)	10.72 $\pm$ 1.84	13.11 $\pm$ 2.98	0.141 $\pm$ 0.031
Moderate, 50–99	96 (29.6)	11.02 $\pm$ 2.11	15.64 $\pm$ 3.26	0.087 $\pm$ 0.019
Severe, <50	17 (5.2)	10.95 $\pm$ 1.92	18.21 $\pm$ 3.94	0.041 $\pm$ 0.011
F statistic	—	0.058	10.31	22.48
p-value	—	0.94	<0.001	<0.001

TABLE 2: Platelet indices according to thrombocytopenia severity

Values are presented as mean $\pm$ standard deviation. Mild thrombocytopenia: platelet count 100–149 $\times 10^9/L$; moderate: 50–99 $\times 10^9/L$; severe: <50 $\times 10^9/L$. MPV: mean platelet volume; PDW: platelet distribution width; PCT: plateletcrit; SD: standard deviation; F: one-way analysis of variance test statistic.

Postoperative thrombocytopenia was reported in 79 (24.4%) patients, followed by dengue fever in 75 (23.1%) patients, undifferentiated acute febrile illness in 51 (15.7%) patients, sepsis in 43 (13.3%) patients, and other viral infections in 25 (7.7%) patients. Less frequent diagnoses were ITP in 21 (6.5%) patients, malaria in 10 (3.1%) patients, hypersplenism in six (1.9%) patients, chronic kidney disease in six (1.9%) patients, and chronic liver disease in four (1.2%) patients. An etiology was assigned in 320 of 324 patients (98.8%); the remaining four patients (1.2%) had an unclassified etiology.

Etiology-specific platelet measurements are presented in Table 3. ITP had the highest mean MPV and PDW,

followed by dengue fever. Hypersplenism had the lowest mean values for these indices. These comparisons are descriptive because no statistical test across etiological categories was reported.

Clinical etiology	MPV, fL	PDW	PCT, %
Postoperative	10.65 ± 1.78	13.72 ± 3.01	0.126 ± 0.028
Dengue fever	11.42 ± 1.95	16.48 ± 3.62	0.093 ± 0.021
Undifferentiated acute febrile illness	10.82 ± 1.81	14.12 ± 3.12	0.109 ± 0.025
Sepsis	11.18 ± 2.04	15.87 ± 3.45	0.102 ± 0.026
Other viral infections	10.97 ± 1.88	14.96 ± 3.08	0.108 ± 0.024
Immune thrombocytopenia	12.06 ± 2.11	17.95 ± 3.81	0.082 ± 0.018
Malaria	10.54 ± 1.72	14.22 ± 2.97	0.114 ± 0.027
Hypersplenism	9.78 ± 1.54	12.81 ± 2.66	0.098 ± 0.019
Chronic kidney disease	10.12 ± 1.63	13.20 ± 2.84	0.105 ± 0.022
Chronic liver disease	9.85 ± 1.49	12.94 ± 2.71	0.095 ± 0.018

TABLE 3: Platelet indices across clinical etiologies

Values are presented as mean ± standard deviation. MPV: mean platelet volume; PDW: platelet distribution width; PCT: plateletcrit; SD: standard deviation; ITP: immune thrombocytopenia; CKD: chronic kidney disease; CLD: chronic liver disease.

Pearson correlation analysis showed a strong positive association between platelet count and PCT ($r = 0.91$, $p < 0.001$) and an inverse association between platelet count and PDW ($r = -0.31$, $p < 0.001$). No significant association was reported between platelet count and MPV ($r = 0.02$, $p = 0.72$).

In the reported mechanism-based comparison, mean PDW was higher in the hyperdestructive group than in the hypoproductive group (16.78 ± 3.41 versus 12.41 ± 2.76 ; $p < 0.001$). PCT was also higher in the hyperdestructive group ($0.121 \pm 0.032\%$ versus $0.102 \pm 0.028\%$; $p = 0.04$). MPV was numerically higher in the hyperdestructive group, although the reported comparison was not statistically significant (11.68 ± 1.94 versus 9.84 ± 1.62 fL; $p = 0.07$).

The reported AUC point estimates were 0.82 for PDW, 0.71 for PCT, and 0.58 for MPV. The corresponding reported sensitivities were 84%, 72%, and 61%, and specificities were 76%, 63%, and 55%, respectively. PDW had the numerically highest AUC. The ROC curves and diagnostic performance estimates are illustrated in Figure 2.

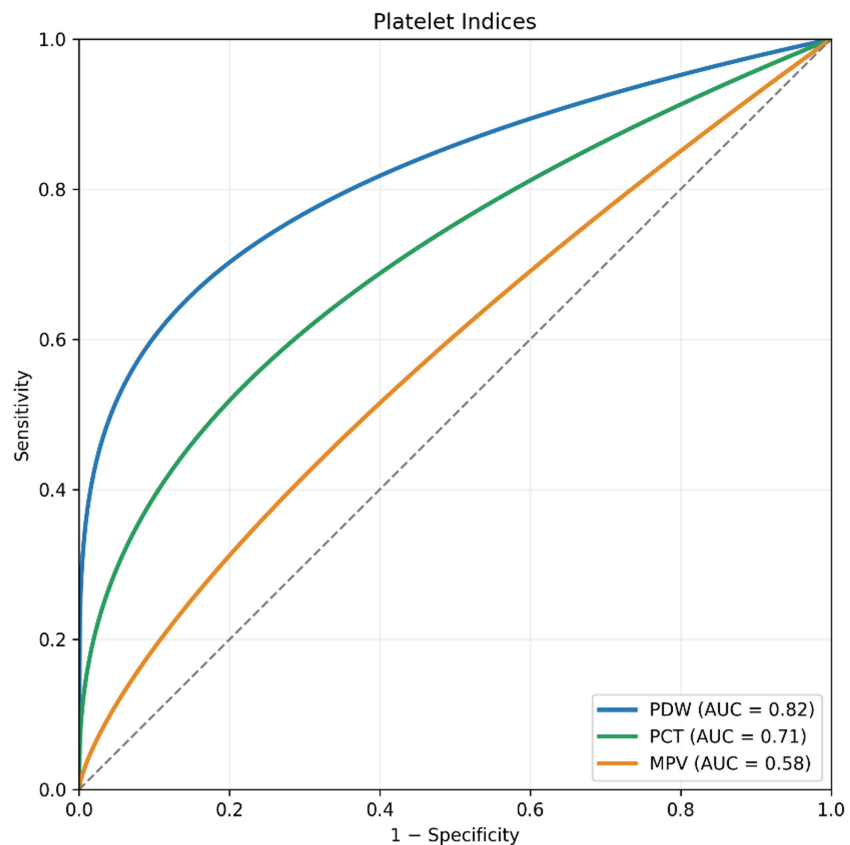


FIGURE 2: ROC curves for platelet indices

The reported ROC analysis yielded an AUC of 0.82 for PDW, with 84% sensitivity and 76% specificity. PCT had an AUC of 0.71, with 72% sensitivity and 63% specificity, whereas MPV had an AUC of 0.58, with 61% sensitivity and 55% specificity. ROC: receiver operating characteristic; PDW: platelet distribution width; PCT: plateletcrit; MPV: mean platelet volume.

Discussion

Thrombocytopenia requires a structured assessment because a reduced platelet count may result from impaired production, increased destruction or consumption, sequestration, or dilution [1]. Clinical history, examination, and peripheral blood smear findings remain central to identifying the cause, while platelet indices may provide complementary information [2-5]. In the present study, PDW had the numerically highest reported AUC among the evaluated platelet indices. However, uncertainties regarding the reference classification and incomplete reporting of the ROC methods limit interpretation of this finding. The result should be considered exploratory and does not establish PDW as a reliable diagnostic discriminator. These findings suggest that routinely available platelet indices warrant consideration as adjuncts to clinical assessment, although their diagnostic usefulness requires careful interpretation. Platelet indices are indirect measures and are not specific to a single mechanism of thrombocytopenia. Their interpretation depends on the clinical context, analyzer characteristics, preanalytical conditions, and the reference classification used to assign underlying mechanisms. Therefore, differences between groups should not be interpreted as establishing a mechanism in an individual patient.

The predominance of middle-aged patients and men describes the hospital population evaluated rather than establishing demographic susceptibility to thrombocytopenia. Convenience sampling and the distribution of patients across clinical departments may have influenced these characteristics. Postoperative conditions and acute infections accounted for most diagnoses, indicating a heterogeneous clinical population. Skeith et al. explain that early postoperative thrombocytopenia commonly involves hemodilution and perioperative platelet consumption, with the timing and magnitude of the decline helping distinguish expected changes from pathological causes [11]. Consequently, postoperative status alone does not establish a single mechanism, and platelet indices should be interpreted alongside the surgical course and associated clinical findings.

PDW increased across progressively more severe thrombocytopenia categories, whereas MPV remained relatively similar. This pattern suggests that variation in platelet size may capture changes that are less

apparent in average platelet volume. However, the association does not establish whether the observed heterogeneity arose from compensatory platelet production, peripheral destruction, or other processes. Kaito et al. demonstrated the potential diagnostic value of platelet size indices in ITP [4]. Ntaios et al. also reported higher MPV and PDW in ITP compared with chemotherapy-associated hypoproliferative thrombocytopenia [12]. Their comparison involved selected diagnostic groups, so its findings cannot be directly extended to the broader etiological spectrum of the present study.

The higher mean MPV and PDW observed in ITP are broadly consistent with previous investigations of platelet size and turnover. Bowles et al. evaluated platelet size as a diagnostic aid in thrombocytopenic patients [13], while Negash et al. found higher platelet indices in ITP than in hypoproliferative thrombocytopenia [5]. Nevertheless, increased platelet size is not specific to immune destruction. ITP itself can involve both accelerated clearance and impaired production [2]. Furthermore, the present comparisons across clinical etiologies were descriptive and should not be interpreted as statistically established differences.

MPV showed substantially lower reported diagnostic discrimination in this study than in several previous investigations. Numbenjapon et al. reported sensitivity of 82.3% and specificity of 92.5% for an MPV threshold above 7.9 fL in identifying hyperdestructive thrombocytopenia against bone marrow assessment [14]. However, less favorable findings have also been published. Chandra et al. reported sensitivity of 67.7% and specificity of 65% for identifying marrow disease using an MPV threshold below 8.15 fL, concluding that MPV could provide an initial indication but had limited diagnostic accuracy [15]. These contrasting results show that platelet-volume measurements do not have uniform performance across settings.

The systematic review and meta-analysis by Walle et al. found pooled sensitivity and specificity of 76% and 79%, respectively, for MPV in differentiating ITP from hypoproliferative thrombocytopenia [6]. In contrast, Xu et al. concluded that MPV and PDW lacked sufficient diagnostic efficiency for identifying bone marrow failure in their thrombocytopenic population [16]. Together, these studies support a contextual interpretation: platelet indices can contribute useful information, but neither a favorable result in one cohort nor a difference between group means establishes a universally reliable diagnostic test. Differences in disease spectrum, reference classification, thresholds, and laboratory methods may contribute to variation between studies.

PCT decreased with increasing thrombocytopenia severity and correlated strongly with platelet count. This finding is consistent with its role as an estimate of circulating platelet volume [3]. However, PCT is derived from platelet count and MPV, making its association with platelet count partly inherent in its calculation. The observed correlation therefore does not establish independent prognostic value or demonstrate platelet functional competence. Similarly, the inverse relationship between PDW and platelet count describes an association with size heterogeneity rather than proving increased peripheral destruction. The study did not assess whether these indices predicted bleeding, transfusion requirements, or recovery.

Analytical conditions also affect the interpretation of platelet indices. Lancé et al. described the influence of anticoagulant exposure, processing time, and measurement technique on MPV [7]. Although samples in the present study were analyzed within two hours, standardized processing does not eliminate all variability. Analyzer differences also limit direct application of published thresholds to the Erba Elite 580. Local validation, consistent units, and documented thresholds are necessary before platelet indices can be incorporated into diagnostic decision-making.

The reported ROC findings identify PDW as the most promising index within this cohort, but they do not establish that it can replace clinical assessment or bone marrow examination. Diagnostic interpretation requires clearly documented mechanism-assignment criteria, group sizes, thresholds, and confidence intervals. The exclusion of several major marrow disorders further restricts conclusions about hypoproliferative thrombocytopenia. International consensus recommendations already indicate that bone marrow examination is unnecessary at initial presentation in otherwise typical isolated ITP [2]. The present study did not measure investigations avoided or changes in management and therefore cannot demonstrate a reduction in invasive testing.

Future studies should evaluate platelet indices against a reproducible reference classification and validate their performance in independent populations. Serial measurements could help determine whether changes accompany clinical recovery. Comparison with the immature platelet fraction may also provide information beyond platelet size alone. Briggs et al. evaluated automated immature platelet fraction measurement in peripheral thrombocytopenia, supporting its investigation as an additional marker of platelet production [17]. Such comparisons could clarify whether combining indices improves diagnostic assessment beyond platelet count, smear findings, and clinical information.

Limitations

This single-center study used convenience sampling and excluded major marrow disorders, systemic consumptive conditions, and selected treatment exposures, limiting representativeness and generalizability.

Mechanism classification lacked a standardized, independently adjudicated reference standard. Incomplete documentation of screening for sepsis-associated coagulopathy and alternative causes of postoperative thrombocytopenia introduced potential overlap and misclassification; therefore, the ROC findings should be considered exploratory. Small subgroups limited precision, while the cross-sectional design precluded assessment of temporal changes, causality, and clinical outcomes.

Conclusions

Platelet indices provided complementary information in the assessment of thrombocytopenia in this hospital-based population. PDW showed a potential discriminatory signal between the assigned mechanism categories, but its diagnostic reliability remains unestablished pending verification of the reference classification, complete reporting of the analytical methods, and independent validation. PCT closely reflected platelet count, whereas MPV demonstrated limited diagnostic discrimination. These findings support interpreting platelet indices alongside clinical findings, platelet counts, and peripheral blood smear examination. The study did not evaluate whether platelet indices improved diagnostic decisions, changed patient management, reduced invasive investigations, or improved clinical outcomes. Therefore, no conclusions regarding these benefits can be drawn from the present findings. Clear mechanism-classification criteria, verification of the reported analyses, and external validation of diagnostic thresholds are required before their routine use for mechanism-based decision-making.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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ECNEW/INST/2022/2692. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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References

1. Stasi R: How to approach thrombocytopenia. *Hematology Am Soc Hematol Educ Program*. 2012, 2012:191-7.
2. Provan D, Arnold DM, Bussell JB, et al.: Updated international consensus report on the investigation and management of primary immune thrombocytopenia. *Blood Adv*. 2019, 3:3780-817. [10.1182/bloodadvances.2019000812](https://doi.org/10.1182/bloodadvances.2019000812)
3. Senthil Nathan S, Varadaraj P, Nallusamy G, Reddy KS: The significance of platelet indices in the evaluation

- of thrombocytopenia. *Cureus*. 2024, 16:e65756. [10.7759/cureus.65756](https://doi.org/10.7759/cureus.65756)
4. Kaito K, Otsubo H, Usui N, et al.: Platelet size deviation width, platelet large cell ratio, and mean platelet volume have sufficient sensitivity and specificity in the diagnosis of immune thrombocytopenia. *Br J Haematol*. 2005, 128:698-702. [10.1111/j.1365-2141.2004.05357.x](https://doi.org/10.1111/j.1365-2141.2004.05357.x)
5. Negash M, Tsegaye A, G/Medhin A: Diagnostic predictive value of platelet indices for discriminating hypo productive versus immune thrombocytopenia purpura in patients attending a tertiary care teaching hospital in Addis Ababa, Ethiopia. *BMC Hematol*. 2016, 16:18. [10.1186/s12878-016-0057-5](https://doi.org/10.1186/s12878-016-0057-5)
6. Walle M, Arkew M, Asmerom H, Tesfaye A, Getu F: The diagnostic accuracy of mean platelet volume in differentiating immune thrombocytopenic purpura from hypo-productive thrombocytopenia: a systematic review and meta-analysis. *PLoS One*. 2023, 18:e0295011. [10.1371/journal.pone.0295011](https://doi.org/10.1371/journal.pone.0295011)
7. Lancé MD, Sloep M, Henskens YM, Marcus MA: Mean platelet volume as a diagnostic marker for cardiovascular disease: drawbacks of preanalytical conditions and measuring techniques. *Clin Appl Thromb Hemost*. 2012, 18:561-8. [10.1177/1076029612458147](https://doi.org/10.1177/1076029612458147)
8. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP: The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLoS Med*. 2007, 4:e296. [10.1371/journal.pmed.0040296](https://doi.org/10.1371/journal.pmed.0040296)
9. Mehta C, George JV, Mehta Y, et al.: Incidence and risk factors for thrombocytopenia in the intensive care units of a tertiary hospital in northern India. *South Afr J Crit Care*. 2016, 32:28-31. [10.7196/SAJCC.2016.v32i1.234](https://doi.org/10.7196/SAJCC.2016.v32i1.234)
10. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA*. 2013, 310:2191-4. [10.1001/jama.2013.281053](https://doi.org/10.1001/jama.2013.281053)
11. Skeith L, Baumann Kreuziger L, Crowther MA, Warkentin TE: A practical approach to evaluating postoperative thrombocytopenia. *Blood Adv*. 2020, 4:776-83. [10.1182/bloodadvances.2019001414](https://doi.org/10.1182/bloodadvances.2019001414)
12. Ntaios G, Papadopoulos A, Chatzinikolaou A, et al.: Increased values of mean platelet volume and platelet size deviation width may provide a safe positive diagnosis of idiopathic thrombocytopenic purpura. *Acta Haematol*. 2008, 119:173-7. [10.1159/000135658](https://doi.org/10.1159/000135658)
13. Bowles KM, Cooke LJ, Richards EM, Baglin TP: Platelet size has diagnostic predictive value in patients with thrombocytopenia. *Clin Lab Haematol*. 2005, 27:370-3. [10.1111/j.1365-2257.2005.00726.x](https://doi.org/10.1111/j.1365-2257.2005.00726.x)
14. Numbenjapon T, Mahapo N, Pornvipavee R, Sriswasdi C, Mongkonsritragoon W, Leelasiri A, Prayoonwiwat W: A prospective evaluation of normal mean platelet volume in discriminating hyperdestructive thrombocytopenia from hypoproductive thrombocytopenia. *Int J Lab Hematol*. 2008, 30:408-14. [10.1111/j.1751-553X.2007.00969.x](https://doi.org/10.1111/j.1751-553X.2007.00969.x)
15. Chandra H, Chandra S, Rawat A, Verma SK: Role of mean platelet volume as discriminating guide for bone marrow disease in patients with thrombocytopenia. *Int J Lab Hematol*. 2010, 32:498-505. [10.1111/j.1751-553X.2009.01212.x](https://doi.org/10.1111/j.1751-553X.2009.01212.x)
16. Xu RL, Zheng ZJ, Ma YJ, Hu YP, Zhuang SH: Platelet volume indices have low diagnostic efficiency for predicting bone marrow failure in thrombocytopenic patients. *Exp Ther Med*. 2013, 5:209-14. [10.3892/etm.2012.799](https://doi.org/10.3892/etm.2012.799)
17. Briggs C, Kunka S, Hart D, Oguni S, Machin SJ: Assessment of an immature platelet fraction (IPF) in peripheral thrombocytopenia. *Br J Haematol*. 2004, 126:93-9. [10.1111/j.1365-2141.2004.04987.x](https://doi.org/10.1111/j.1365-2141.2004.04987.x)