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THE ROLE OF MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH, PLATELET COUNT AND PLATELETCRIT IN EVALUATION OF TYPE OF THROMBOCYTOPENIA

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INTRODUCTION

Platelets are anucleate cytoplasmic fragments derived from bone-marrow megakaryocytes that form the first line of defence against microvascular and macrovascular bleeding by adhering to injured endothelium and aggregating into a haemostatic plug.[1] Thrombocytopenia, defined as a platelet count below $150x10^9$/L, is one of the commonest haematological abnormalities encountered in clinical practice and predisposes patients to bleeding complications ranging from mild mucocutaneous haemorrhage to life-threatening internal bleeding.

Mechanistically, thrombocytopenia can be grouped under three broad headings: Hypo proliferative thrombocytopenia, arising from impaired bone-marrow production, as in aplastic anaemia, megaloblastic anaemia, myelodysplastic syndrome, and post-chemotherapy marrow suppression; Hyperdestructive thrombocytopenia, resulting from accelerated peripheral destruction or consumption of platelets, as in immune thrombocytopenic purpura (ITP), dengue, malaria, and sepsis; and splenic sequestration, in which platelets are pooled within an enlarged spleen secondary to portal hypertension, myeloproliferative disease, or haemoglobinopathy.[2,3] Because management, monitoring, and prognosis differ substantially across these mechanistic categories, accurate subtype classification carries direct clinical relevance.

Bone marrow aspiration and biopsy, which permit direct visualisation of megakaryocyte number and morphology, remain the traditional reference standard for this classification.[4] However, the procedure is invasive, costly, and carries bleeding risk in patients who are already thrombocytopenic, prompting interest in simpler, non-invasive alternatives.

Modern automated haematology analysers generate platelet indices like MPV, PDW, PCT, and platelet large cell ratio (PLCR) as part of every routine complete blood count, at no additional cost.[5] MPV reflects the average size of circulating platelets and rises when the marrow releases young, "stress" platelets in response to peripheral destruction, while it falls in states of impaired thrombopoiesis.[6] PDW quantifies the degree of anisocytosis among platelets and tends to increase when platelets of markedly differing size coexist, as occurs during active turnover.[6] Plateletcrit, analogous to haematocrit for red cells, expresses the total circulating

platelet mass and integrates both platelet number and size.[7]

Thrombocytopenia affects a substantial proportion of hospitalised and critically ill patients and is encountered across the age spectrum, from neonates with sepsis to elderly patients with myelodysplasia, reflecting the diversity of its underlying causes.[8] Reported subtype distributions vary considerably between populations, with infectious and immune-mediated (hyper destructive) causes predominating in regions with a high burden of dengue, malaria, and sepsis, while marrow-failure (hypo proliferative) causes are relatively more prominent in other referral settings, underscoring the value of region-specific data.[9]

Despite encouraging preliminary data[10], most published studies evaluating platelet indices are limited by small sample sizes, binary comparisons of thrombocytopenia mechanisms, and inconsistent assessment of multiple platelet indices simultaneously.[9,11] In addition, evidence from resource-limited settings, where bone marrow examination is often unavailable or impractical, remains scarce. Therefore, the present study was undertaken to determine whether routinely available platelet indices, namely mean platelet volume (MPV), platelet distribution width (PDW), platelet count, and plateletcrit (PCT), can reliably identify thrombocytopenia and differentiate its major mechanistic subtypes, including hypo proliferative, hyper destructive, and splenic sequestration thrombocytopenia. We also sought to determine which of these indices provides the greatest diagnostic accuracy and whether their combined interpretation could serve as a practical, low-cost, non-invasive first-line approach for the evaluation of thrombocytopenia.

MATERIALS AND METHODS

Study design and setting

The study was a prospective, hospital-based case-control study, which was conducted in the Department of Pathology, Sanjay Gandhi Memorial Hospital and associated hospitals of Shyam Shah Medical College, Rewa, Madhya Pradesh, India, which was conducted after approval from the Institutional Ethics Committee (S.No./IEC/M.C./2024/13824, dated 24/5/24) till next 12 months. Written informed consent was obtained from all participants (or guardians, for minors) before enrolment.

Study population, inclusion, and exclusion criteria

Study Group	Inclusion Criteria	Exclusion Criteria
Cases (Thrombocytopenia Group).	Patients of all age groups referred from inpatient (IPD) and outpatient (OPD) services for Complete Blood Count (CBC) and Peripheral Smear (PS)examination. • Platelet count $<100 \times 10^9$/L, confirmed on peripheral smear examination.	• Pseudo thrombocytopenia (EDTA-induced platelet clumping). •Borderline thrombocytopenia (platelet count $100–150 \times 10^9$/L) were excluded as they remain clinically asymptomatic and did not exhibit a significant bleeding tendency over time suggesting it may represent benign variant rather than true pathological thrombocytopenia. • History of blood or platelet transfusion within the preceding 7 days.
Controls (Control Group).	Age and sex-matched individuals referred for CBC and PS examination. • Platelet count $\geq 150 \times 10^9$/L. • No clinical or laboratory evidence of haematological or infectious disease.	• Acute or chronic infection. • Any haematological disorder. • Current treatment with corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs), or immunosuppressive agents. • History of blood or platelet transfusion within the preceding 7 days.

Patients referred from in-patient and out-patient departments for complete blood count (CBC) and peripheral smear examination (PS) were screened. The inclusion and exclusion criteria were divided into cases and controls.

Sample size

Using the formula $n = 4pq/d^2$, with an estimated thrombocytopenia prevalence of 13% (q = 87%) and an allowable error of 5%, the calculated sample size was 174; after adjusting for 10% attrition, 200 cases and 200 age and sex matched controls were enrolled.

Laboratory Methods

Three millilitres of venous blood were collected aseptically into EDTA vacutainers and processed within two hours on a fully automated three-part differential haematology analyser (Zybio Z3), which combines electrical-impedance (Coulter principle) and optical light-scatter technology, recording platelet count, MPV, PDW, and PCT for every subject; daily internal quality control was performed. Peripheral smears were prepared by the standard wedge method, stained with Leishman stain, and examined under oil immersion; platelet counts were also estimated by averaging counts across ten oil-immersion fields and multiplying by 15,000/microlitre as a confirmatory check.[12]

Classification of thrombocytopenia subtype

Cases were classified into hyper destructive, hypo proliferative, or splenic sequestration thrombocytopenia based primarily on the final clinical diagnosis established from the patient's history, relevant laboratory investigations. Platelet indices (MPV, PDW, platelet count, and plateletcrit) and peripheral smear findings were recorded independently and were evaluated for their ability to differentiate these clinically established mechanistic subtypes rather than being used as the sole basis for classification (Table 1).

Table 1: Characteristic clinical, peripheral smear, and platelet index findings observed in the three mechanistic subtypes of thrombocytopenia.

Feature	Hyperdestructive	Hypo proliferative	Splenic sequestration
Platelet count	Decreased	Decreased	Decreased
MPV	Increased	Decreased / low-normal	Normal / mildly increased
PDW	Increased	Normal / mildly increased	Normal / mildly increased
PLCR	Increased	Decreased	Normal / moderately increased
Plateletcrit	Normal / mildly decreased	Markedly decreased	Proportionately reduced
Giant platelets	Present or absent	Absent	Absent

Statistical analysis

Data were analysed using SPSS software. Categorical variables were expressed as frequencies/percentages and compared using the chi-square test; continuous variables were expressed as mean +/- standard deviation and compared using the independent-samples t-test (two groups) or one-way ANOVA (three subtypes). Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of platelet count, MPV, PDW, and plateletcrit in identifying thrombocytopenia. Because thrombocytopenia was defined using platelet count, the ROC analysis for platelet count was included only as a reference comparator for the other platelet indices.

RESULTS

200 confirmed thrombocytopenic cases and 200 sex matched controls with a comparable age distribution were studied. Cases were significantly older than controls (mean age 33.30 +/- 20.92 vs 25.45 +/- 21.27 years; t=3.72, p<0.001), while gender distribution was comparable (54.5% vs 57.0% female; chi-square=1.31, p=0.25) (Table 2). Jaundice (14.5% vs 4.0%; p<0.001) and hepatosplenomegaly (13.0% vs 6.0%; p=0.03) were significantly more frequent among cases, whereas fever was equally distributed (47.0% vs 46.0%; p=0.96).

Table 2: Baseline characteristics and comparison of platelet indices between cases and controls.

Variable	Cases (n=200)	Controls (n=200)	Test Statistic	p-value
Demographic & Clinical Characteristics				
Age (years), mean ± SD	33.30 ± 20.92	25.45 ± 21.27	t = 3.72	<0.001
Female sex, n (%)	109 (54.5)	114 (57.0)	χ^2 = 1.31	0.25
Fever, n (%)	94 (47.0)	92 (46.0)	χ^2 = 0.00	0.96
Jaundice, n (%)	29 (14.5)	8 (4.0)	χ^2 = 11.91	<0.001
Hepatosplenomegaly, n (%)	26 (13.0)	12 (6.0)	χ^2 = 4.91	0.03
Platelet Parameters				
Platelet count (/µL)	48,010 ± 27,783	317,740± 185,774	t = −19.87	<0.001
MPV (fL)	11.02 ± 2.46	10.16 ± 1.79	t = 3.99	<0.001
PDW (fL)	16.74 ± 1.16	16.49 ± 1.03	t = 2.33	0.02
Plateletcrit (%)	0.06 ± 0.04	0.30 ± 0.17	t = −20.52	<0.001
PLCC	19.76 ± 15.63	78.05 ± 45.49	t = −17.14	<0.001
PLCR (%)	37.20 ± 18.09	28.60 ± 13.15	t = 5.44	<0.001
Giant platelets, n (%)	45 (22.5)	13 (6.5)	χ^2 = 19.38	<0.001
Platelet clumps, n (%)	32 (16.0)	44 (22.0)	χ^2 = 1.97	0.16

All platelet parameters differed significantly between cases and controls (Table 2). Platelet count (48,010 +/- 27,783 vs 317,740 +/- 185,774/ microlitre; p<0.001) and plateletcrit (0.06 +/- 0.04 vs 0.30 +/- 0.17%; p<0.001) were markedly lower among cases, whereas MPV (11.02 +/- 2.46 vs 10.16 +/- 1.79 fL; p<0.001), PDW (16.74 +/- 1.16 vs 16.49 +/- 1.03 fL; p=0.02), and PLCR (37.2 +/- 18.1 vs 28.6 +/- 13.2%; p<0.001) were significantly higher among cases; PLCC was significantly lower in cases (19.76 +/- 15.63 vs 78.05 +/- 45.49; p<0.001). Giant platelets on smear were significantly more common among cases (22.5% vs 6.5%; odds ratio 4.17, 95% CI 2.14-8.12; p<0.001), while EDTA-induced platelet clumping did not differ between groups (16.0% vs 22.0%; p=0.16).

Among the 200 cases, hyper destructive thrombocytopenia was the commonest subtype (n=95,

47.5%), followed by hypo proliferative (n=56, 28.0%) and splenic sequestration (n=49, 24.5%) thrombocytopenia; no controls met criteria for thrombocytopenia (p<0.001). Presenting complaints among cases most often comprised non-specific systemic symptoms grouped as "others" (n=102), anaemia-related complaints (n=38), antenatal presentations (n=31), post-natal presentations (n=20), and fever (n=9), a distribution that differed significantly from controls (p=0.02). Among past-history variables, chronic liver disease was recorded exclusively among cases (n=5), while hypothyroidism and type 2 diabetes mellitus showed a similar, non-significant trend toward higher frequency in cases (p=0.34).

All four platelet indices varied significantly across the three subtypes (Table 4, Figure 1). MPV showed the strongest between-group separation (ANOVA F=38.62, p<0.001), rising progressively from hypo proliferative (8.90 +/- 1.50 fL) to splenic sequestration (11.20 +/- 2.00 fL) to hyper destructive thrombocytopenia (12.40 +/- 1.70 fL). PDW followed a parallel but more modest gradient (16.20 +/- 1.05 to 16.80 +/- 1.30 to 17.10 +/- 1.00 fL; F=9.12, p<0.001), as did platelet count (34,000 +/- 24,000 to 52,000 +/- 23,000 to 56,000 +/- 27,000/ microlitre; F=10.84, p<0.001) and plateletcrit (0.03 +/- 0.02 to 0.06 +/- 0.03 to 0.07 +/- 0.04%; F=32.22, p<0.001). Comparison of platelet indices across the three mechanistic subtypes of thrombocytopenia (one-way ANOVA) in elaborated in Table 3.

Table 3: Comparison of platelet indices across the three mechanistic subtypes of thrombocytopenia (one-way ANOVA).

Parameter	Hypo proliferative (n=56)	Splenic sequestration (n=49)	Hyperdestructive (n=95)	ANOVA F	p-value
MPV (fL)	8.90 ± 1.50	11.20 ± 2.00	12.40 ± 1.70	38.62	<0.001
PDW (fL)	16.20 ± 1.05	16.80 ± 1.30	17.10 ± 1.00	9.12	<0.001
Platelet count (/µL)	34,000 ± 24,000	52,000 ± 23,000	56,000 ± 27,000	10.84	<0.001
Plateletcrit (%)	0.03 ± 0.02	0.06 ± 0.03	0.07 ± 0.04	32.22	<0.001

Table 4: Diagnostic performance of platelet indices for identifying thrombocytopenia (ROC curve analysis).

Parameter	Cut-off	Sensitivity (%)	Specificity (%)	AUC	p-value
MPV	≥11.50 fL	40.0	79.0	0.60	0.04
PDW	≥17.00 fL	45.0	74.0	0.58	0.06
Plateletcrit	≤0.12%	95.5	92.5	0.99	<0.001
Platelet count	≤99,000/µL	100.0	100.0	1.00	<0.001

Figure 1. Comparison of Platelet Indices Across Thrombocytopenia Subtypes

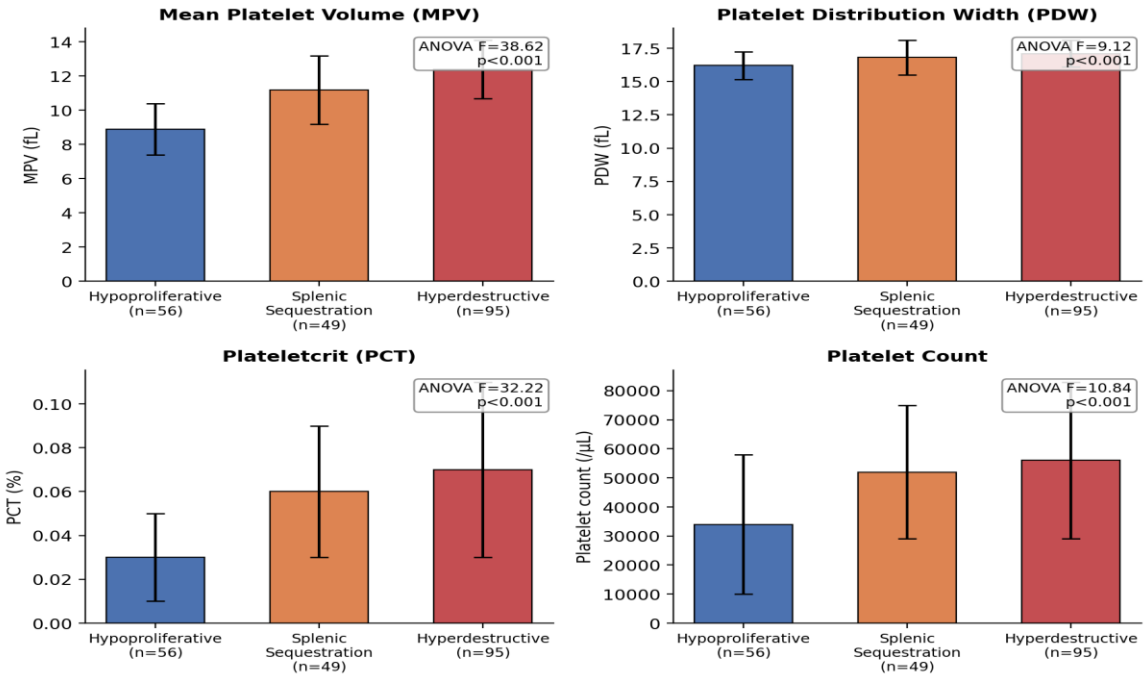

Figure 1. Comparison of platelet indices across thrombocytopenia subtypes (mean ± SD; error bars represent one SD).

On ROC analysis (Table 4, Figure 2), platelet count showed perfect discrimination between cases and controls (AUC 1.00; sensitivity and specificity 100% at a cut-off <=99,000/ µL). Plateletcrit performed nearly as well (AUC 0.99; sensitivity 95.5%, specificity 92.5% at cut-off <=0.12%; p<0.001). MPV (AUC 0.60; sensitivity 40%, specificity 79% at cut-off >=11.50 fL; p=0.04) and PDW (AUC 0.58; p=0.06) showed only modest and for PDW, non-significant ability to separate cases from controls as a single group, despite their strong subtype-discriminating performance on ANOVA (Table 3).

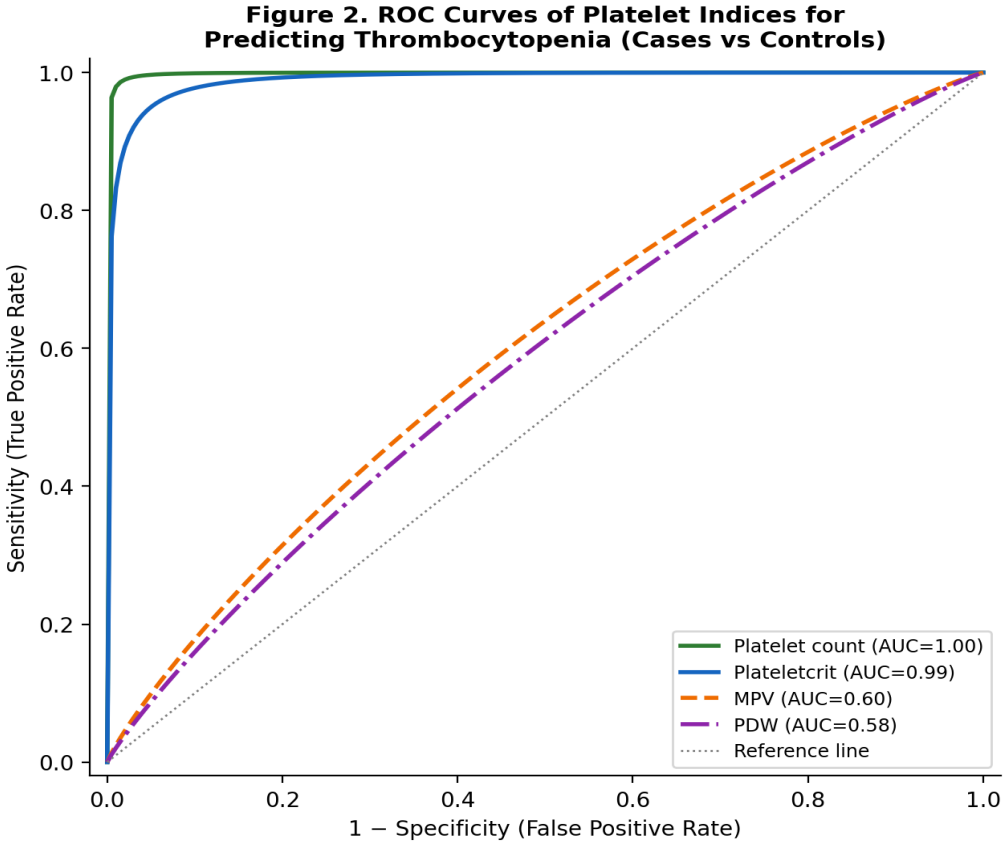

Figure 2: ROC curves of platelet indices for predicting thrombocytopenia (cases vs controls). Curves are representative, plotted from the reported AUC values under a binormal model.

DISCUSSION

The present study investigated whether routinely available platelet indices (MPV, PDW, platelet count, and plateletcrit) could accurately identify thrombocytopenia and differentiate its major mechanistic subtypes. Our findings demonstrate that platelet count and plateletcrit showed excellent diagnostic performance for confirming thrombocytopenia, whereas MPV and PDW were more informative in distinguishing hypo proliferative, hyper destructive, and splenic sequestration thrombocytopenia. Among the four indices, MPV exhibited the strongest discriminatory ability for differentiating the underlying mechanisms, while platelet count and plateletcrit were the most accurate markers for identifying thrombocytopenia itself. These findings indicate that the combined interpretation of routinely available platelet indices, together with peripheral smear examination, provides a practical, inexpensive, and non-invasive first-line approach for the evaluation of thrombocytopenia, particularly in settings where bone marrow examination is not readily available.

Two distinct diagnostic questions emerged from our data, with different answers. When the objective was simply to confirm the presence of thrombocytopenia, platelet count (AUC 1.00) and plateletcrit (AUC 0.99) performed near-perfectly, whereas MPV (AUC 0.60) and PDW (AUC 0.58, non-significant) performed only modestly. This is unsurprising as thrombocytopenia is, by definition, a disorder of platelet number, so platelet count and plateletcrit which is mathematically derived from count and volume, are expected to separate cases from controls almost completely. When the objective shifted to classifying thrombocytopenia by underlying mechanism, the picture reversed: MPV emerged as the strongest discriminator across subtypes (ANOVA F=38.62, p<0.001), followed by PCT (F=32.22), platelet count (F=10.84), and PDW (F=9.12). This apparent paradox modest ROC performance for MPV and PDW at the case control level, but strong ANOVA separation at the subtype level is explained by the mixed composition of the case group: high MPV values in hyper destructive cases and low values in hypo proliferative cases partially cancel each other when the case group is treated as a

single entity, diluting the overall case-control signal while preserving subtype-level discrimination.[13,14] As expected, platelet count tells diagnostic accuracy for identifying thrombocytopenia. However, this finding should be interpreted with caution because thrombocytopenia was defined using a platelet count threshold of $<100 \times 10^9$/L. Therefore, the excellent ROC performance of platelet count primarily reflects the study definition rather than an independent diagnostic capability. The more clinically relevant findings relate to the performance of MPV, PDW, and plateletcrit in differentiating the underlying mechanisms of thrombocytopenia.

Our finding of a progressive MPV gradient, lowest in hypo proliferative (8.90 +/- 1.50 fL), intermediate in splenic sequestration (11.20 +/- 2.00 fL), and highest in hyper destructive thrombocytopenia (12.40 +/- 1.70 fL) is biologically coherent and consistent with prior reports.[15,16,17,18] When platelets are consumed rapidly at the periphery, as in ITP, dengue, or sepsis, the marrow compensates by releasing larger, younger, more metabolically active "stress platelets," analogous to reticulocytes released during haemolysis, which raises MPV.[6] Conversely, in marrow failure states such as aplastic or megaloblastic anaemia, megakaryocyte output is diminished, and the few circulating platelets are uniformly small[19,20], Khan WS et al[13] and Nathan SS et al[14] both reported higher MPV in hyper destructive than hypo proliferative subtypes, while Khajuria A et al[11] identified MPV as an independent predictor of hyper destructive thrombocytopenia on multivariate analysis. Malladi SS et al[16] additionally demonstrated that MPV tracked disease progression serially in febrile thrombocytopenic patients, supporting its dynamic, mechanism-linked behaviour.

PDW mirrored this gradient, being highest in hyper destructive (17.10 +/- 1.00 fL) and lowest in hypo proliferative thrombocytopenia (16.20 +/- 1.05 fL; $F=9.12$, $p<0.001$), reflecting the coexistence of newly released large platelets alongside older, smaller ones during active turnover, versus the relatively uniform, small platelet population of marrow failure.[21] This pattern parallels findings from Khan WS et al [13], Jayanti Nayak DB et al[15], and Khairkar PS et al[22], the last of whom emphasised PDW as an under-utilised but diagnostically informative index best applied after thrombocytopenia has already been confirmed, rather than as a primary screening tool, an interpretation directly supported by our ROC data.

Plateletcrit, a composite measure of total circulating platelet mass, showed the expected profound reduction in cases versus controls, but the more clinically novel observation was that PCT also varied significantly across subtypes ($F=32.22$, $p<0.001$), rising from hypo proliferative through splenic sequestration to hyper destructive thrombocytopenia. This contrasts with earlier reports, including Jayanti Nayak DB et al[15] and Nathan SS et al[14] which found no significant subtype-level PCT variation, a difference we attribute to our larger subgroup sizes and the inclusion of a dedicated splenic-sequestration arm, which most published binary study designs omit. Data from other clinical contexts support the biological plausibility of PCT variation like Aydemir S et al[23] linked low PCT to higher mortality in critically ill COVID-19 patients, Talat F et al[24] reported comparable PCT values in neonatal sepsis regardless of culture positivity, and Mansour SM et al[25] found PCT unresponsive when the underlying pathology was not primarily production-based, together indicating that PCT variation is most informative when platelet production itself is the variable under study, precisely the contrast exploited by our three-arm design; similarly, Tungka EK et al[26] found PCT differences between survivors and non-survivors of acute kidney injury did not reach significance, reinforcing that PCT's discriminatory value is mechanism-specific rather than universal.

Platelet count showed the expected gradient across subtypes, being lowest in hypo proliferative thrombocytopenia, reflecting the absence of marrow reserve to compensate for reduced output, an observation corroborated by Jayanti Nayak DB et al[15] and Khan WS et al[13] the latter of whom directly correlated lower counts in hypo productive cases with reduced marrow megakaryocyte numbers on biopsy. This differential carries direct clinical implications, since patients with hypo proliferative thrombocytopenia and lower absolute counts are at greater risk of spontaneous haemorrhage and may more readily meet thresholds for prophylactic platelet transfusion.[27,28]

Giant platelets on peripheral smear were significantly more frequent among cases (22.5% vs 6.5%; odds ratio 4.17), consistent with the predominance of hyper destructive thrombocytopenia in our study and with reports from Shinde A et al[17] who showed that combining platelet histogram interpretation with smear review improves diagnostic precision. The absence of a significant difference in EDTA-induced platelet clumping between groups ($p=0.16$) supports the analytical integrity of our dataset, since pseudo thrombocytopenia is a recognised cause of spuriously low automated counts and artefactually elevated MPV.[12,29] PLCR was significantly higher, and PLCC significantly lower, among cases, consistent with a case group dominated by hyper destructive thrombocytopenia releasing large, immature platelets against a markedly reduced total platelet pool.[30,31]

Taken together, our findings support a practical, two-tier diagnostic approach: platelet count and plateletcrit should be used first to confirm true thrombocytopenia, after excluding pseudo thrombocytopenia by peripheral smear; MPV and PDW should then be applied to infer the likely underlying mechanism, with high values (MPV >=11.5 fL, PDW >=17.0) favouring a hyper destructive process warranting evaluation for ITP, dengue, malaria,

or sepsis[8], and low values favouring marrow failure warranting bone marrow examination. This staged approach, also advocated by Nathan SS et al[14] and Khajuria A et al[11] offers a cost-effective, non-invasive strategy that may reduce reliance on bone marrow biopsy, particularly in resource-limited settings.

Strengths of the study

1. This study included three major causes of thrombocytopenia (hyper destruction, hypo proliferation, and splenic sequestration), whereas most previous studies included some of them.
2. All four platelet indices (MPV, PDW, Platelet count, and PCT) were evaluated in all study groups, giving a more complete assessment.
3. The study had a large sample size with 200 cases and 200 age and sex-matched controls, making the results more reliable.
4. Every case of thrombocytopenia was confirmed by peripheral smear examination, reducing the chance of false diagnosis due to pseudo thrombocytopenia.
5. Age and sex matched controls helped reduce bias and made the comparison between groups more accurate.
6. Different statistical methods were used to assess both the ability of platelet indices to identify thrombocytopenia and their ability to differentiate its underlying causes.
7. The current study showed that different platelet indices have different clinical uses, rather than one index being useful for all situations.
8. Since platelet indices are automatically available in a routine CBC, the findings can be easily applied in day-to-day clinical practice without any extra cost or additional tests.

Limitations of the Study

1. This was a single-centre study conducted at a tertiary care hospital, so the findings may not represent patients in other hospitals or community settings.
2. Bone marrow examination was not performed in all patients. Therefore, the cause of thrombocytopenia was determined using clinical findings, peripheral smear examination, and platelet indices, which may have led to incorrect classification in some cases.
3. Immature Platelet Fraction (IPF) could not be evaluated because it was not available on the CBC analyser used in this study.
4. Some subgroups had a smaller number of patients, which may have reduced the accuracy of comparisons between different causes of thrombocytopenia.
5. Larger multicentre and longitudinal studies with longer follow-up, routine bone marrow examination, and IPF assessment are needed to confirm and strengthen these findings.

CONCLUSION

Platelet count and plateletcrit were near-perfect discriminators of thrombocytopenia itself, while mean platelet volume and platelet distribution width, though less useful for simple case identification, proved to be powerful markers of the underlying mechanistic subtype, rising progressively from hypo proliferative through splenic sequestration to hyper destructive thrombocytopenia. A combined interpretation of these four routinely available indices, together with peripheral smear examination, constitutes a practical, low-cost, non-invasive first-line strategy for both confirming thrombocytopenia and inferring its likely aetiology, potentially reducing dependence on invasive bone marrow examination in appropriate clinical settings.

Declaration of Generative AI and AI-assisted technologies in the writing process: The authors declare that no generative artificial intelligence (AI) or AI-assisted technologies were used in the writing, preparation, or editing of this manuscript.

DECLARATIONS

Ethical Approval

- The study was approved by the Institutional Ethics Committee of Shyam Shah Medical College, Rewa, Madhya Pradesh, India.
- Written informed consent was obtained from all participants before they were included in the study.

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- No funding was received for this study.

Conflict of Interest

- The authors declare that there is no conflict of interest.

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Author Contributions

- DK: Study concept and design, data collection, laboratory work, and manuscript writing.
- RQ: Study supervision, guidance, and manuscript review.
- LT: Statistical analysis, co-guidance, and manuscript review.
- All authors reviewed and approved the final version of the manuscript.

Data Availability

- The study data are available from the corresponding author upon request.

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