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Original Research Article

PLATELET INDICES AS DIAGNOSTIC DISCRIMINATORS IN THROMBOCYTOPENIA: INSIGHTS FROM A 437-CASE RETROSPECTIVE STUDY

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ABSTRACT

Background: Thrombocytopenia is a common haematological disorder with heterogeneous underlying mechanisms, including hypoproductive, hyperdestructive, and combined etiologies. Accurate mechanistic differentiation is crucial for guiding management but often requires invasive bone marrow examination. Automated platelet indices may provide a non-invasive alternative for early stratification. **Aim:** To evaluate the diagnostic performance of platelet indices in differentiating hypoproductive, hyperdestructive, and combined thrombocytopenia and to assess their utility as non-invasive markers of thrombocytopenia mechanism. **Materials and Methods:** This retrospective cross-sectional study evaluated the diagnostic performance of platelet indices in differentiating hypoproductive, hyperdestructive, and combined thrombocytopenia. It included 437 patients with moderate-to-severe thrombocytopenia (<100 × 10^9 /L). Cases were categorized into hypoproductive (n=119), hyperdestructive (n=207), and combined (n=111) groups based on clinical and etiological evaluation. Platelet indices— the mean platelet volume (MPV), platelet distribution width (PDW), platelet-large cell ratio (P-LCR), and plateletcrit (PCT)—were analyzed. Intergroup comparisons were performed using Kruskal–Wallis and Mann–Whitney U tests. Correlations were assessed using Spearman's coefficient. Diagnostic performance was evaluated using one-vs-rest receiver operating characteristic (ROC) analysis. **Results:** Hyperdestructive thrombocytopenia demonstrated significantly higher MPV (12.10 ± 1.81 fL), PDW (19.32 ± 4.41 fL), and P-LCR (41.03 ± 12.64%) compared to hypoproductive cases ($p < 0.001$). MPV showed excellent discriminatory performance for hyperdestructive thrombocytopenia (AUC = 0.909), followed by P-LCR (AUC = 0.850). Platelet count alone demonstrated limited discrimination (AUC = 0.609). The combined group exhibited intermediate index values. PCT strongly correlated with platelet count ($\rho = 0.940$, $p < 0.001$). **Conclusion**: MPV and P-LCR demonstrate excellent diagnostic utility in differentiating thrombocytopenia mechanisms. Integration of platelet indices into routine evaluation may enhance non-invasive mechanistic classification and reduce reliance on invasive procedures.

INTRODUCTION

Thrombocytopenia, defined as a platelet count <150 × 10^9 /L, is a common haematological abnormality encountered in clinical practice and is associated with bleeding risk ranging from mucocutaneous hemorrhage to life-threatening intracranial bleeding at very low counts.[1,2] The underlying mechanisms of thrombocytopenia are broadly categorized into decreased marrow production (hypoproductive), increased peripheral destruction or consumption (hyperdestructive), and splenic sequestration.[3,4] Accurate identification of the underlying pathophysiology is essential, as management strategies differ significantly between these categories.

Conventionally, bone marrow examination has been regarded as the gold standard for differentiating hypoproductive from hyperdestructive thrombocytopenia. However, it is invasive, costly,

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time-consuming, and carries procedural risks, particularly in patients with severe thrombocytopenia.[5] Consequently, there has been increasing interest in non-invasive, readily available haematological parameters that can aid in early mechanistic stratification.

Modern automated haematology analyzers provide platelet indices such as mean platelet volume (MPV), platelet distribution width (PDW), platelet-large cell ratio (P-LCR), plateletcrit (PCT), and more recently immature platelet fraction (IPF). These indices reflect platelet size, heterogeneity, activation status, and marrow regenerative response. MPV, a marker of average platelet size, tends to increase in hyperdestructive states due to release of larger, younger platelets from the bone marrow.[6,7] PDW reflects variability in platelet size and is associated with active platelet turnover, while P-LCR represents the proportion of large platelets in circulation. PCT, analogous to hematocrit, represents total circulating platelet mass.[8,9] IPF has emerged as a promising marker of marrow response, with higher values indicating peripheral destruction and lower values suggesting impaired production.[10]

Several studies have demonstrated the diagnostic utility of platelet indices in differentiating thrombocytopenia mechanisms. Saran et al. reported significantly higher MPV, PDW, and PCT in hyperdestructive thrombocytopenia compared to hypoproductive cases in a 201-patient study.[11] Shetageri et al., in a large 369-case analysis, identified MPV as the most sensitive parameter for mechanistic discrimination.[12] More recently, Senthil Nathan et al. highlighted the prognostic and diagnostic significance of platelet indices in an 80-patient prospective study.[13] A 350-participant case–control study further demonstrated that IPF, MPV, and P-LCR were independent predictors of hyperdestructive thrombocytopenia.[14]

Despite growing evidence, most published studies are limited by small sample sizes or binary classification models. Comprehensive multi-categorical mechanistic evaluation integrating platelet indices with etiological stratification remains relatively underexplored.[15]

Therefore, the present study aims to evaluate the diagnostic performance of platelet indices in differentiating hypoproductive, hyperdestructive, and combined thrombocytopenia in a large cohort of patients, and to assess their potential role as practical, non-invasive tools for mechanistic classification in routine clinical practice.

MATERIALS AND METHODS

This retrospective cross-sectional analytical study included 437 patients diagnosed with moderate-to-severe thrombocytopenia at a tertiary care center. Thrombocytopenia was defined as a platelet count $<150 \times 10^9$ /L, based on automated haematology analyzer reports. Demographic details, complete blood count parameters, platelet indices, and etiological diagnoses were retrieved from institutional records. Patients with mild thrombocytopenia (platelet counts between 100 to 150×10^9 /L) were excluded from this cohort to focus on clinically significant disease where accurate mechanistic differentiation most directly impacts acute patient management and bleeding risk.

Inclusion Criteria

Patients of all ages and both sexes with moderate-to-severe thrombocytopenia (platelet count $<100 \times 10^9$ /L) were included in the study. Only those patients with a definitive clinical and/or etiological diagnosis enabling classification into hypoproductive, hyperdestructive, or combined thrombocytopenia were considered eligible.

Exclusion Criteria

Mild thrombocytopenia (100 to 150×10^9 /L) was excluded to eliminate diagnostic ambiguity from incidental or transient physiological variations, and to focus the evaluation on clinically significant cases where early mechanistic differentiation is critical for management. Patients were also excluded if the blood samples showed platelet clumping or pseudothrombocytopenia (such as EDTA-induced platelet aggregation), or if they had received platelet transfusion prior to sample collection, as these conditions could affect platelet indices and compromise accuracy.

Cases were categorized into three mechanistic groups—hypoproductive, hyperdestructive, and combined thrombocytopenia—based on clinical evaluation, peripheral smear findings, and underlying etiological diagnosis in accordance with standard haematological principles described in the literature, independent of platelet indices. Hypoproductive thrombocytopenia included disorders associated with decreased marrow production, such as hematologic malignancies, marrow suppression, or drug-induced suppression. Hyperdestructive thrombocytopenia comprised conditions characterized by peripheral platelet destruction or consumption, including sepsis, immune thrombocytopenia (ITP), hypersplenism, disseminated intravascular coagulation (DIC) etc. Combined cases like Dengue fever, systemic lupus erythematosus (SLE), and other acute viral infections, demonstrated overlapping clinical or etiological features suggestive of combined mechanisms.

Platelet count, Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), Platelet-Large Cell Ratio (P-LCR), and Plateletcrit (PCT) were obtained using automated haematology analyzers calibrated according to manufacturer standards. Thrombocytopenia severity was categorized as moderate or severe based on established platelet count thresholds. Continuous variables were expressed as mean ± standard deviation and median (interquartile range). Comparisons among the three groups were performed using the Kruskal–Wallis

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test, followed by Mann–Whitney U test with Bonferroni correction for pairwise analysis. Categorical variables were analyzed using the Chi-square test. Spearman's rank correlation coefficient (ρ) was used to assess relationships between platelet count and platelet indices. Diagnostic performance of platelet indices for mechanism discrimination was evaluated using one-vs-rest receiver operating characteristic (ROC) curve analysis with calculation of area under the curve (AUC). A p value <0.05 was considered statistically significant.

RESULTS

A total of 437 cases of thrombocytopenia were included in the analysis. Based on mechanism classification, cases were categorized into hypoproductive (n = 119), hyperdestructive (n = 207), and combined mechanism (n = 111) groups.

The mean age of the study population was 44.4 ± 24 years, comprising 230 males (52.6%) and 207 females (47.4%). The mean age of the study population did not differ significantly across the three mechanism groups (Kruskal–Wallis test, p = 0.783). Similarly, sex distribution was comparable between groups (Chi-square test, p = 0.492), with a slight male predominance overall.

Table 1 shows that platelet indices demonstrated significant variation across the three mechanism groups. Platelet count differed significantly among hypoproductive, hyperdestructive, and combined thrombocytopenia (Kruskal–Wallis test, p < 0.001). Mean Platelet Volume (MPV) also showed marked intergroup differences (p < 0.001), with the hyperdestructive group exhibiting higher MPV values compared to the hypoproductive group, while the combined group demonstrated intermediate values. Similarly, Platelet Distribution Width (PDW) and Platelet-Large Cell Ratio (P-LCR) varied significantly across the mechanism categories (both p < 0.001). Plateletcrit (PCT) likewise demonstrated a statistically significant difference among the three groups (p < 0.001), indicating distinct alterations in platelet mass and morphology according to the underlying pathophysiological mechanism.

Table 1: Comparison of platelet indices across hypoproductive, hyperdestructive, and combined thrombocytopenia (mean ± SD, median [IQR], Kruskal–Wallis test)

Variable	Hypoproductive mean±SD	Hypoproductive median (IQR)	Hyperdestructive mean±SD	Hyperdestructive median (IQR)	Combined mean±SD	Combined median (IQR)	p-value (Kruskal–Wallis)
Platelet count (/µL)	34069.11 ± 16912.75	35374.00 (21308.00–51065.00)	46229.25 ± 27265.81	45085.00 (23910.00–61415.00)	36488.77 ± 23310.42	32446.00 (18943.00–50209.50)	<0.001
MPV (fL)	7.59 ± 0.62	7.60 (7.10–8.10)	12.10 ± 1.81	12.50 (11.20–13.50)	10.07 ± 0.99	9.90 (9.30–10.85)	<0.001
PDW (fL)	12.94 ± 1.74	13.00 (11.40–14.35)	19.32 ± 4.41	20.20 (17.55–22.95)	17.08 ± 2.72	17.20 (15.25–19.60)	<0.001
P-LCR (%)	17.00 ± 5.23	17.00 (13.00–22.00)	41.03 ± 12.64	43.00 (32.00–51.00)	30.46 ± 8.41	30.00 (23.00–38.00)	<0.001
PCT (%)	0.03 ± 0.01	0.03 (0.01–0.04)	0.05 ± 0.03	0.06 (0.03–0.08)	0.04 ± 0.02	0.04 (0.02–0.05)	<0.001

Post-hoc analysis revealed p < 0.001 for all comparisons between hypoproductive and hyperdestructive groups.

Figures 1-3 boxplots illustrate the distribution of MPV, PDW, and P-LCR across the three mechanism groups, demonstrating clear separation particularly for MPV and P-LCR.

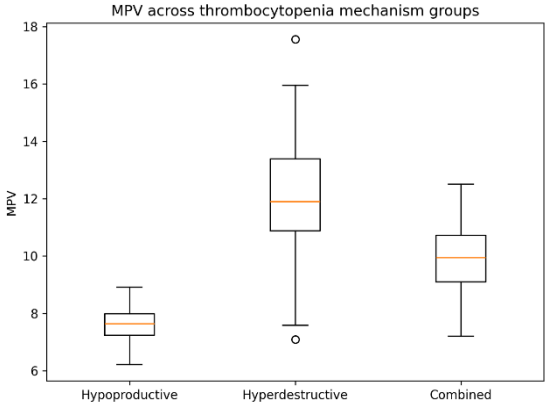

Figure 1: Boxplot depicting the distribution of Mean Platelet Volume (MPV) across hypoproductive, hyperdestructive, and combined thrombocytopenia, demonstrating significantly higher MPV values in the hyperdestructive group with intermediate values in the combined group

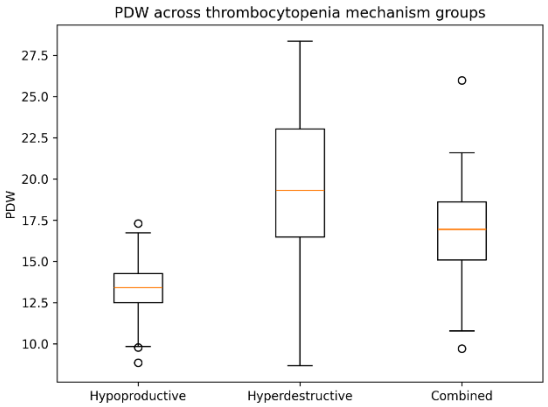

Figure 2: Boxplot showing the distribution of Platelet Distribution Width (PDW) across hypoproductive, hyperdestructive, and combined thrombocytopenia, demonstrating significantly higher PDW values in the hyperdestructive group and intermediate values in the combined group

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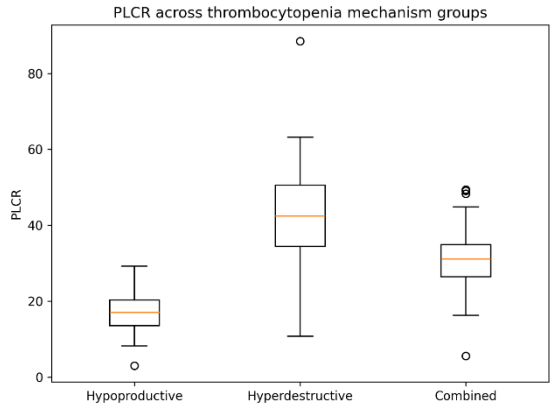

Figure 3: Boxplot illustrating the distribution of Platelet-Large Cell Ratio (P-LCR) across the three mechanism groups, with the highest values observed in hyperdestructive thrombocytopenia and intermediate values in the combined group.

The distribution of thrombocytopenia severity differed significantly among mechanism groups. Severe thrombocytopenia was more frequently observed in the 88 cases (73.9%) of hypoproductive and 82 cases (73.9%) in combined groups, whereas the hyperdestructive group showed a relatively higher proportion of moderate thrombocytopenia in 84 cases (40.6%) compared to the other two groups. The distribution of severity differed significantly across mechanism categories (Chi-square test, $p = 0.006$)

Table 2 depicts that in the overall cohort, infectious etiologies constituted the largest proportion of thrombocytopenia cases, followed by hematologic and immune causes. When stratified according to mechanism, a highly significant association was observed between etiologic group and mechanism category (Chi-square test, $p < 0.001$). Hyperdestructive thrombocytopenia was predominantly associated with infectious and immune causes, consistent with peripheral platelet destruction as the primary mechanism. In contrast, hypoproductive thrombocytopenia showed a stronger association with hematologic, malignant, and drug-related etiologies, reflecting impaired marrow production. The combined group demonstrated an overlapping etiologic distribution, suggesting combined or transitional pathophysiological processes.

Table 2: Distribution of etiological categories across hypoproductive, hyperdestructive, and combined thrombocytopenia

Group	Drug	Hematologic	Immune	Infectious	Liver/Systemic	Malignant	Systemic	p-value (Chi-square)
Hypoproductive	24 (20.2%)	85 (71.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	10 (8.4%)	0 (0.0%)	<0.001
Hyperdestructive	8 (3.9%)	0 (0.0%)	74 (35.7%)	70 (33.8%)	40 (19.3%)	0 (0.0%)	15 (7.2%)	
Combined	0 (0.0%)	0 (0.0%)	11 (9.9%)	100 (90.1%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	

Table 3 shows that the spearman correlation analysis revealed a very strong positive correlation between plateletcrit (PCT) and platelet count ($\rho \approx 0.93$, $p < 0.001$), consistent with the role of PCT as a measure of total circulating platelet mass. PDW and P-LCR demonstrated statistically significant but negative correlations with platelet count ($p < 0.05$), indicating partial association with platelet quantity and morphology. In contrast, MPV did not show a strong independent correlation with platelet count. These findings highlight that platelet indices provide additional morphometric and functional information beyond the absolute platelet count, supporting their complementary diagnostic value in evaluating thrombocytopenia.

Table 3: Spearman correlation of platelet indices with platelet count in patients with thrombocytopenia

Variable	Spearman ρ with platelet count	p-value
MPV	-0.078	0.103
PDW	-0.201	<0.001
P-LCR	-0.133	0.005
PCT	0.940	<0.001

Table 4 demonstrates multiclass diagnostic performance evaluated using a one-vs-rest ROC approach. MPV demonstrated the highest discriminative ability, particularly for identifying hyperdestructive thrombocytopenia, with excellent AUC values. P-LCR also showed strong discriminatory performance, while PDW and PCT demonstrated moderate accuracy. Platelet count alone showed comparatively lower discriminative power.

The one-vs-rest ROC curves for MPV in hyperdestructive and hypoproductive mechanism groups are shown in Figure 4, illustrating its robust ability to differentiate hyperdestructive thrombocytopenia from hypoproductive mechanisms.

Because the combined mechanism group represents an intermediate, overlapping pathophysiological state, binary one-vs-rest ROC analysis is mathematically unsuited to isolate it from the hypoproductive and hyperdestructive extremes.

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Therefore, ROC analysis was strictly utilized to evaluate the diagnostic utility of platelet indices in predicting the primary hyperdestructive and hypoproductive states.

Table 4: One-vs-Rest ROC analysis of platelet indices for discrimination of thrombocytopenia mechanism groups (AUC values)

Predictor	ROC (One-vs-Rest)	AUC
MPV (fL)	Hypoproductive vs Rest	0.994
MPV (fL)	Hyperdestructive vs Rest	0.909
PDW (fL)	Hypoproductive vs Rest	0.869
PDW (fL)	Hyperdestructive vs Rest	0.780
P-LCR (%)	Hypoproductive vs Rest	0.933
P-LCR (%)	Hyperdestructive vs Rest	0.850
PCT (%)	Hypoproductive vs Rest	0.734
PCT (%)	Hyperdestructive vs Rest	0.737
Platelet count (/µL)	Hypoproductive vs Rest	0.578
Platelet count (/µL)	Hyperdestructive vs Rest	0.609

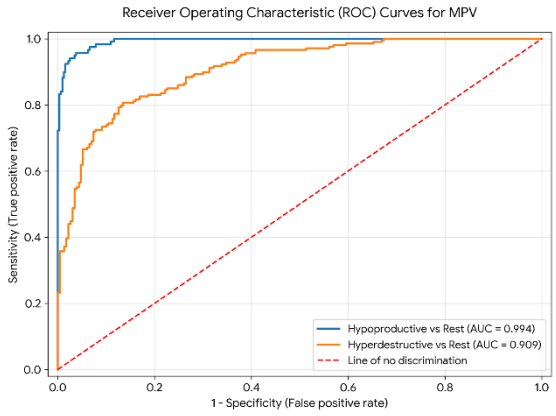

Figure 4: One-vs-rest receiver operating characteristic (ROC) curves for Mean Platelet Volume (MPV) in discriminating hypoproductive and hyperdestructive. MPV demonstrated excellent diagnostic performance for identifying hyperdestructive thrombocytopenia (AUC = 0.909), and for hypoproductive (AUC = 0.994). The diagonal dashed line represents the line of no discrimination.

Overall, platelet indices (MPV, PDW, P-LCR, and PCT) differed significantly across hypoproductive, hyperdestructive, and combined thrombocytopenia, underscoring distinct underlying pathophysiological mechanisms. Among these, MPV and P-LCR demonstrated the strongest discriminatory performance, particularly in identifying hyperdestructive thrombocytopenia. Etiological categories were significantly associated with mechanism classification, reinforcing the clinical relevance of mechanistic stratification. The combined group consistently exhibited intermediate haematological profiles, supporting its biological plausibility as a transitional or overlapping mechanism of thrombocytopenia.

DISCUSSION

The present study evaluated platelet indices in a large cohort of 437 patients with thrombocytopenia and demonstrated significant differences across hypoproductive, hyperdestructive, and combined mechanisms. The findings reinforce the growing body of evidence supporting the diagnostic utility of automated platelet parameters in mechanistic stratification of thrombocytopenia.

In our cohort, hyperdestructive thrombocytopenia demonstrated significantly higher MPV (12.10 ± 1.81 fL), PDW (19.32 ± 4.41 fL), P-LCR (41.03 ± 12.64%), and PCT (0.05 ± 0.03%) compared to hypoproductive thrombocytopenia (MPV 7.59 ± 0.62 fL; PDW 12.94 ± 1.74 fL; P-LCR 17.00 ± 5.23%; PCT 0.03 ± 0.01%; $p < 0.001$ for all comparisons). These results are pathophysiologically consistent with increased peripheral destruction leading to compensatory marrow release of larger, younger platelets into circulation. Other studies similarly identified MPV as the most sensitive discriminator between mechanisms.[16,17] The magnitude of MPV separation in our study appears more pronounced, possibly reflecting the predominance of severe thrombocytopenia cases within our cohort.

Our ROC analysis further strengthens this evidence, with MPV achieving an AUC of 0.994 for identifying hypoproductive and 0.909 for identifying hyperdestructive thrombocytopenia, indicating excellent discriminatory performance. Similar findings were reported by previous studies that demonstrated significant diagnostic accuracy of platelet indices, particularly MPV, in differentiating hypoproductive from hyperdestructive thrombocytopenia using ROC-based analysis.[18] P-LCR also demonstrated strong accuracy, followed by PDW and PCT, whereas platelet count alone showed comparatively inferior discrimination.

An important strength of this study is the inclusion of a combined mechanistic category. The combined group consistently exhibited intermediate MPV (10.07 ± 0.99 fL), PDW (17.08 ± 2.72 fL), and P-LCR (30.46 ± 8.41%) values, statistically distinct from both hypoproductive and hyperdestructive groups ($p < 0.001$ in most comparisons). Most prior studies, employed binary classification models. By incorporating a third category, our findings support the concept that platelet indices reflect a biological continuum rather than a strict dichotomy.

Although platelet count differed significantly between groups ($p < 0.001$), platelet count alone demonstrated limited mechanistic discrimination.

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Interestingly, severe thrombocytopenia was more frequent in hypoproductive (73.9%) and combined (73.9%) groups compared to hyperdestructive cases (59.4%) ($p = 0.006$). While immune-mediated destruction may produce profound thrombocytopenia, marrow suppression and hematologic malignancies frequently result in persistent severe reductions, which likely explains this distribution in our study population.

Spearman correlation analysis revealed a very strong positive correlation between PCT and platelet count ($\rho = 0.940$, $p < 0.001$), consistent with the mathematical derivation of PCT as a function of platelet count and volume. In contrast, MPV showed no significant independent correlation with platelet count ($\rho = -0.078$, $p = 0.103$), indicating that platelet size provides morphometric information beyond absolute quantity. PDW and P-LCR demonstrated weak but statistically significant negative correlations with platelet count, supporting the concept that increased platelet heterogeneity accompanies peripheral destruction.

The highly significant association between etiology and mechanistic classification ($p < 0.001$) validates our categorization model. Hyperdestructive thrombocytopenia was predominantly associated with infectious and immune causes, consistent with prior observations by Slichter,[3] and Provan et al.[5] In contrast, hypoproductive thrombocytopenia was strongly linked to hematologic and malignant etiologies, reflecting impaired marrow production. The combined group demonstrated overlapping features, most frequently infectious etiologies, suggesting evolving or combined pathophysiological mechanisms, possibly involving both consumption and transient marrow suppression.

Our findings are consistent with the conclusions of previous studies that emphasized the diagnostic and prognostic value of platelet indices and their potential to reduce unnecessary bone marrow examinations. Previous studies have also emphasised that the platelet indices remain underutilized despite being readily available through automated analyzers.[19] Given their non-invasive nature, cost-effectiveness, and rapid availability, incorporation of platelet indices into diagnostic algorithms appears rational and clinically feasible.

The present study has several strengths, including a relatively large sample size ($n = 437$), comprehensive multicategorical classification, and detailed ROC analysis. Few prior studies have incorporated a combined mechanistic category, and even fewer have performed one-vs-rest ROC evaluation across three groups. Nevertheless, limitations must be acknowledged. The retrospective design may introduce selection bias, IPF was not evaluated, and the study was conducted at a single center. Prospective multicenter studies incorporating IPF and validating standardized cut-off values are warranted to enhance generalizability.

Limitations: A limitation of this study is the exclusion of mild thrombocytopenia (100 to 150 × 10^9 /L). While this enriched our cohort for clear mechanistic pathology, it means our proposed diagnostic thresholds for MPV and P-LCR may not be directly extrapolatable to patients presenting with very mild platelet reductions.

CONCLUSION

In conclusion, platelet indices—particularly MPV and P-LCR—demonstrate excellent discriminatory performance in differentiating hyperdestructive from hypoproductive thrombocytopenia. The combined group exhibits intermediate morphometric characteristics, supporting the biological continuum model of thrombocytopenia mechanisms. Platelet count alone is insufficient for mechanistic classification, whereas integration of platelet indices offers a practical, non-invasive approach that may enhance diagnostic accuracy and reduce reliance on invasive bone marrow evaluation.

Conflict of Interest

The authors declared no conflict of interest.

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