RESEARCH PAPER

Evaluation of Thrombocytopenia Using Platelet Indices

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ABSTRACT

Aim & Objective: To evaluate and compare the diagnostic utility of platelet indices plain differentiating hypo-productive versus hyper-destructive thrombocytopenia and to identify which indices show statistically significant differences between groups.

Materials & Methods: This observational, prospective, comparative study was conducted in the Department of Pathology, Chettinad Hospital & Research Institute, Chennai, from November 2025 to January 2026. A total of 214 patients with thrombocytopenia (platelet count <150 × 10^9/L), confirmed on peripheral smear, were enrolled. Cases were classified into hypo-productive (aplastic anemia, megaloblastic anemia, marrow infiltration) and hyper-destructive (immune thrombocytopenic purpura, disseminated intravascular coagulation, hypersplenism) groups based on clinical data, peripheral smear findings, and, where available, bone marrow examination and relevant laboratory investigations. Peripheral venous blood samples were collected in K_2EDTA tubes and analyzed within 2–4 hours using an automated hematology analyzer. Platelet indices including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) were recorded. Statistical analysis was performed using the independent t-test, with significance set at $p < 0.05$.

Results: Among 214 participants, the mean age was predominantly 21–40 years (40.2%) with a slight male predominance (57.9%). The two etiologic groups were equally represented (n = 107 each). Megaloblastic anemia (43%) and immune thrombocytopenic purpura (37.4%) were the most common causes of hypo-productive and hyper-destructive thrombocytopenia, respectively. MPV and PDW were significantly higher in the hyper-destructive group ($p < 0.001$), reflecting marrow compensatory release of larger, younger platelets, whereas PCT showed no significant difference ($p = 0.09$). "Mean platelet volume (MPV) and platelet distribution width (PDW) were significantly higher in the hyper-destructive group compared to the hypo-productive group ($p < 0.001$), whereas plateletcrit (PCT) showed no significant difference ($p = 0.09$)."

Conclusion: MPV and PDW are reliable, rapid, and cost-effective indices for differentiating hypo-productive from hyper-destructive thrombocytopenia. Elevated MPV and PDW are indicative of hyper-destructive mechanisms, whereas lower values suggest impaired marrow production. PCT has limited diagnostic value. Incorporating these indices into routine CBC reporting can aid early classification and guide further diagnostic evaluation, potentially reducing the need for invasive procedures.

Keywords: Thrombocytopenia, Mean Platelet Volume, Platelet Distribution Width, Plateletcrit, Hypo-productive, Hyper-destructive

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INTRODUCTION

Thrombocytopenia, defined as a platelet count below 150 × 10^9/L, is a common hematological abnormality frequently identified in routine complete blood count (CBC) reports, peripheral smear examinations, and during evaluation of patients presenting with bleeding

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manifestations, infections, or systemic illnesses. It represents a heterogeneous group of conditions with varied etiologies and prognoses [1]. Prompt and accurate determination of the underlying mechanism is essential, as therapeutic approaches differ substantially between hypoproductive thrombocytopenia (caused by decreased bone marrow platelet production) and hyperdestructive thrombocytopenia (resulting from increased peripheral destruction or sequestration) [2]. Traditionally, clinical evaluation and bone marrow examination have been used for etiologic classification; however, these methods may be invasive, resource-intensive, and not immediately available in all settings [3]. As a result, there is growing interest in **platelet indices** derived from automated hematology analyzers as potential non-invasive biomarkers that can aid in distinguishing between thrombocytopenia subtypes.

MPV, a measure of average platelet size, is often elevated in hyper-destructive thrombocytopenia due to increased marrow turnover and release of larger, immature platelets [5]. Similarly, PDW, which reflects variability in platelet size, has been proposed to increase in conditions with active platelet destruction [6]. PCT, representing the total platelet mass in circulation, may correlate with disease severity and bone marrow output [7]. Several studies have suggested significant differences in these indices between patients with hypoproductive versus hyperdestructive thrombocytopenia, supporting their potential utility as diagnostic aids [8–10].

From a clinical perspective, thrombocytopenia requires prompt evaluation, as management depends on the underlying etiology. Hypoproductive conditions may require nutritional supplementation (e.g., vitamin B12 or folate), bone marrow–directed therapy, or treatment of underlying marrow pathology, whereas hyperdestructive causes often require immunosuppressive therapy, treatment of infections, or management of systemic conditions. In cases of severe thrombocytopenia, particularly when platelet counts fall below critical levels or when active bleeding is present, urgent intervention such as platelet transfusion may be lifesaving. Therefore, early differentiation between these mechanisms is essential for timely and appropriate management.

Despite accumulating evidence, there is considerable variability in reported diagnostic performance of platelet indices across populations and analyzer technologies, underscoring the need for further validation in diverse clinical settings [11].

A study by Khushboo et al. demonstrated that MPV and PDW values differed significantly between etiologic groups and could contribute to improved classification of thrombocytopenic patients [12]. Thrombocytopenia can be broadly classified based on underlying mechanisms into hypoproductive (decreased bone marrow production) and hyperdestructive (increased peripheral destruction or sequestration). It may also be categorized based on clinical severity, etiology (infectious, immune-mediated, nutritional, or malignant), and bone marrow findings. Platelets play a crucial role in hemostasis by contributing to primary clot formation, maintaining vascular integrity, and participating in inflammatory and immune responses.

However, few prospective comparative studies have systematically evaluated all three indices MPV, PDW, and PCT in the same cohort with rigorous clinical categorization. In this context, the present study was designed to evaluate and compare the diagnostic utility of MPV, PDW, and PCT in differentiating hypoproductive from hyperdestructive thrombocytopenia in an observational prospective cohort at a tertiary care center

Severe thrombocytopenia can lead to life-threatening complications such as spontaneous bleeding, intracranial hemorrhage, and mucosal bleeding, necessitating urgent diagnosis and management. Early differentiation between hypo-productive and hyper-destructive causes is critical as treatment strategies vary from bone marrow stimulation to immunosuppressive therapy.

AIM & OBJECTIVE

To evaluate and compare the diagnostic utility of MPV, PDW, and PCT in differentiating hypo-productive versus hyper-destructive thrombocytopenia, and to identify which indices show statistically significant differences between groups.

MATERIALS & METHODOLOGY

Study design and setting

This will be an observational, prospective, comparative study conducted in the Department of Pathology, Chettinad Hospital & Research Institute, Chennai. The study period will extend from November 2025 to January 2026. The platelet indices for all samples/cases were evaluated using automated hematology analyzer, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT), for differentiating hypo-productive from hyper-destructive thrombocytopenia.

Clinical classification of thrombocytopenia

Each enrolled case was classified into one of two etiologic groups hypoproductive or hyperdestructive thrombocytopenia using a combination of clinical assessment, peripheral smear examination, bone marrow findings (where available), and relevant ancillary investigations. Clinical assessment included detailed history, presenting symptoms (such as bleeding manifestations), and associated systemic illnesses.

Peripheral smear evaluation was performed to assess platelet number, size (including presence of giant platelets), platelet clumping, and associated red cell or white cell morphological abnormalities. Bone marrow examination, when available, was used to evaluate megakaryocyte number and morphology, thereby helping to differentiate decreased production from increased peripheral destruction.

Relevant ancillary investigations included serum vitamin B12 and folate levels (for megaloblastic anemia),

coagulation profile including prothrombin time (PT), activated partial thromboplastin time (aPTT), and D-dimer (for disseminated intravascular coagulation), liver function tests and ultrasound abdomen (for hypersplenism), infection markers such as malarial parasite test, dengue serology, and other relevant microbiological investigations, as well as autoimmune markers where indicated.

Hypoproductive thrombocytopenia was diagnosed in conditions associated with decreased bone marrow platelet production (e.g., aplastic anemia, megaloblastic anemia, marrow infiltration), characterized by reduced or ineffective megakaryopoiesis. Hyperdestructive thrombocytopenia was diagnosed in conditions with increased peripheral platelet destruction or sequestration (e.g., immune thrombocytopenic purpura, disseminated intravascular coagulation, hypersplenism), typically showing normal or increased megakaryocytes in the bone marrow.

Only cases with adequate clinico-hematological and laboratory evidence supporting the assigned category were included in the final comparative a

Specimen collection and laboratory methods
Peripheral venous blood (3–5 mL) was collected into K_2EDTA tubes under aseptic precautions and mixed gently to prevent clotting. Samples were processed within 2 hours of collection using an automated hematology analyzer.The analyzer operates on the electrical impedance principle for platelet counting and volume measurement, with additional optical/fluorescence methods where applicable for improved accuracy. Platelet count and platelet indices mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) were obtained as part of the complete blood count (CBC).

Peripheral blood smears were prepared and stained using Leishman's stain and examined microscopically under oil immersion objective (100X) to assess platelet morphology and to exclude platelet clumping, giant platelets, or or any other significant morphology and presence of hemoparasite

All samples were processed within 2 hours to minimize EDTA-induced platelet swelling and ensure accuracy of MPV measurements

Data collection
A structured proforma was used to record demographic details, presenting complaints, relevant clinical diagnosis, platelet counts, and platelet indices (MPV, PDW, PCT). Where available, results of bone marrow examination and relevant ancillary investigations were also recorded to support etiological classification. These included serum vitamin B12 and folate levels, coagulation profile (prothrombin time, activated partial thromboplastin time, and D-dimer), liver function tests, infection markers (such as malarial parasite testing and dengue serology), and imaging studies such as ultrasound abdomen for assessment of splenomegaly. Only one sample per participant was used for analysis.

Sample size and sampling technique
Based on a study conducted by Khushboo et al. [4], it was anticipated that the proportion of platelet indices would be approximately 60% in one group and 40% in the other group. The sample size was calculated for comparison of two independent proportions using the standard formula:

$$n = [(Z\alpha/2 + Z\beta)^2 \times \{p1(1 - p1) + p2(1 - p2)\}] / (p1 - p2)^2$$

where $p1 = 0.6$ and $p2 = 0.4$, $Z\alpha/2 = 1.96$ for 95% confidence interval, and $Z\beta = 0.84$ for 80% power. The calculation was performed using OpenEpi software for validation. Based on this, the estimated sample size was 107 participants in each group, yielding a total of 214 participants for the study.

Inclusion and Exclusion Criteria

Inclusion criteria:
Patients diagnosed with thrombocytopenia (platelet count $<150 \times 10^9$/L) by automated CBC and confirmed by peripheral smear.

Cases for which the analyzer successfully reports MPV, PDW, and PCT.

Patients who provide informed consent for participation.

Exclusion criteria:
Patients who have received antiplatelet therapy (e.g., aspirin) or blood transfusion within 14–15 days prior to sampling.

Patients receiving anticoagulant therapy (e.g., heparin, warfarin, or direct oral anticoagulants) were excluded, as these agents can influence platelet count, activation, and platelet indices.

Statistical analysis
"Independent t-test was used for comparison. ROC curve analysis was performed. SPSS version XX was used."

RESULTS
The demographic data for the 214 participants reveals a predominantly young-to-middle-aged cohort with a slight male majority. The etiological classification of thrombocytopenia among the 214 participants shows a perfectly even split between the two primary mechanisms of the condition. Specifically, hypo-productive thrombocytopenia, characterized by decreased platelet production in the bone marrow, and hyper-destructive thrombocytopenia, involving the accelerated breakdown of platelets in the bloodstream, each accounted for exactly 50% of the cases (n = 107). This identical distribution suggests that, within this specific study population, the underlying causes are just as likely to stem from production failures as they are from peripheral destruction or consumption.

Table 1: Age distribution of study population

Age Group (years)	Number (n)	Percentage (%)
< 20	38	17.8
21–40	86	40.2
41–60	58	27.1
> 60	32	14.9
Total	**214**	**100**

The majority of study participants belonged to the 21–40 years age group (40.2%), followed by the 41–60 years group (27.1%), indicating that thrombocytopenia was most commonly observed in young and middle-aged adults. Individuals below 20 years (17.8%) and above 60 years (14.9%) constituted smaller proportions of the study population, suggesting relatively lower representation in the extremes of age. Overall, the findings demonstrate a predominance of thrombocytopenia in the economically productive age group.

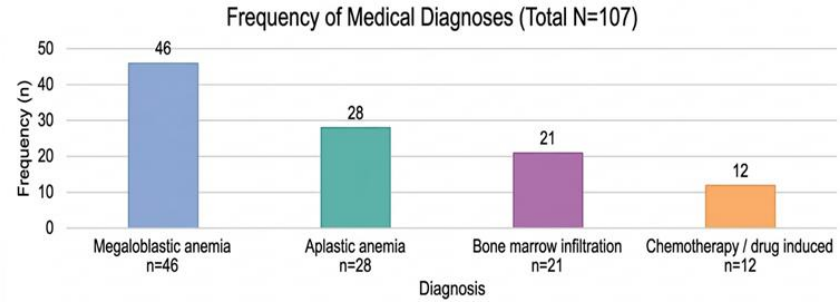

Figure 1: Causes of Hypo-productive Thrombocytopenia (n = 107)

The primary causes of hypo-productive thrombocytopenia within a study group of 107 patients, highlighting that megaloblastic anemia is the most prevalent etiology, accounting for 43% (n = 46) of the cases. This is followed by aplastic anemia at 26.2% (n = 28) and bone marrow infiltration at 19.6% (n = 21), indicating that structural or nutritional deficiencies in the marrow are significant drivers of reduced platelet production. The least common cause identified in this cohort was chemotherapy or drug-induced suppression, representing 11.2% (n = 12) of the total. Collectively, these data emphasize that while various factors can impair marrow function, nutritional megaloblastic changes and primary bone marrow failure syndromes constitute the majority of hypo-productive platelet disorders in this specific population.

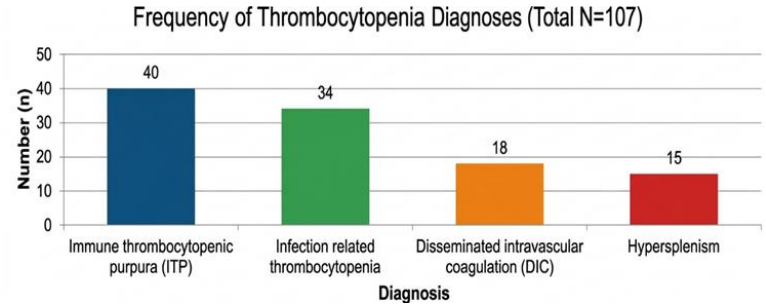

Figure 2: Causes of Hyper-destructive Thrombocytopenia (n = 107)

The causes of hyper-destructive thrombocytopenia in the same cohort of 107 patients demonstrated that immune thrombocytopenic purpura (ITP) was the leading cause, accounting for 37.4% (n = 40) of cases. This was followed by infection-related thrombocytopenia at 31.8% (n = 34), while disseminated intravascular coagulation (DIC) and hypersplenism accounted for 16.8% (n = 18) and 14% (n = 15), respectively.

Hypersplenism refers to a condition characterized by splenic enlargement (splenomegaly) and increased sequestration and premature destruction of blood cells, including platelets, within the spleen. Under normal conditions, the spleen stores approximately one-third of the body's platelets; however, in hypersplenism, excessive pooling and destruction lead to thrombocytopenia. Common causes of hypersplenism include chronic liver disease with portal hypertension, hematological malignancies (such as lymphoma and leukemia), infections (e.g., malaria, kala-azar), and autoimmune disorders.

In contrast to hypo-productive causes, these findings highlight that accelerated peripheral destruction or sequestration often driven by immune-mediated mechanisms, systemic infections, or splenic pooling is a major mechanism responsible for thrombocytopenia in this group.

Table 2: Diagnostic Utility of Platelet Indices

Parameter	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
MPV	82.2	76.6	78.9	80.4	79.4
PDW	79.4	72	75.1	76.8	75.5
PCT	55.1	50.4	52.3	53.2	52.7

Table 2 evaluates the diagnostic utility of platelet indices in differentiating between hypo-productive and hyper-destructive thrombocytopenia, demonstrating that both Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) serve as effective diagnostic tools with relatively high sensitivity, specificity, and accuracy across all metrics, generally exceeding 70%. In contrast, Plateletcrit (PCT) shows significantly lower performance, with sensitivity and specificity hovering near 50–55%, suggesting that it lacks the clinical robustness required to reliably discriminate between these two underlying mechanisms of thrombocytopenia. Consequently, these findings support the prioritize use of MPV and PDW in clinical settings to assist in the rapid classification of platelet disorders.

Table 3: Comparison of Platelet Indices between Hypoproductive and Hyperdestructive Thrombocytopenia

Parameter	Hypo-productive (Mean ± SD)	Hyper-destructive (Mean ± SD)	p-value
Platelet count (×10⁹/L)	62.4 ± 18.6	58.9 ± 20.3	0.21
MPV (fL)	8.1 ± 1.2	11.2 ± 1.6	<0.001
PDW (%)	14.8 ± 2.3	18.6 ± 3.1	<0.001
PCT (%)	0.07 ± 0.02	0.06 ± 0.02	0.09

MPV and PDW were significantly higher in hyperdestructive thrombocytopenia ($p < 0.001$), indicating increased platelet production and variability due to peripheral destruction. Platelet count and PCT showed no statistically significant difference between the groups, suggesting limited value in differentiating the underlying mechanism.

DISCUSSION

Thrombocytopenia has a variety of causes that can be broadly divided into two categories: hypo-productive (reduced marrow production) and hyper-destructive (increased peripheral destruction). Because these two groups have quite different diagnostic work-ups and therapeutic approaches, it is clinically crucial to distinguish between them. Although it is intrusive and not always accessible, bone marrow testing has historically been regarded as the gold standard for differentiating these mechanismsAdditional platelet parameters such as mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT), generated by automated hematology analyzers, have emerged as useful **non-invasive markers**, as they can provide indirect information about platelet production and destruction using routine blood samples, thereby reducing the need for invasive procedures like bone marrow examination [13].The current study shows that platelet indices MPV and PDW in particular are useful metrics for distinguishing between hyper-destructive and hypo-productive thrombocytopenia.

In the present study, MPV and PDW were significantly higher in patients with hyper-destructive thrombocytopenia compared to those with hypo-productive thrombocytopenia ($p < 0.001$), whereas PCT did not show a statistically significant difference ($p = 0.09$). These findings indicate that platelet size and platelet size variability increase in conditions associated with peripheral platelet destruction because of compensatory bone marrow response and release of larger immature platelets into circulation.

The findings of the present study are consistent with the observations made by Numbenjapon et al. [14], who reported significantly elevated MPV values in hyper-destructive thrombocytopenia compared to hypo-productive causes. Similarly, Shetageri et al. [15] demonstrated that MPV showed high sensitivity and specificity in differentiating the two etiological categories of thrombocytopenia. Comparable findings regarding elevated PDW values in hyper-destructive thrombocytopenia were also reported by Baig et al. [17], supporting the role of platelet size variability as a useful diagnostic marker.

In our study, MPV demonstrated the highest diagnostic performance with sensitivity of 82.2%, specificity of 76.6%, and overall diagnostic accuracy of 79.4%, followed by PDW with an accuracy of 75.5%. These findings are in agreement with previous studies that identified MPV and PDW as reliable platelet indices for preliminary differentiation of thrombocytopenia. In contrast, PCT showed relatively poor diagnostic utility, which is consistent with earlier reports suggesting that plateletcrit mainly reflects total circulating platelet mass rather than the underlying mechanism of thrombocytopenia.

Numerous investigations that highlight the diagnostic significance of platelet volume indices have produced similar results. MPV was considerably higher in hyper-destructive thrombocytopenia than in hypo-productive causes, according to a study by Numbenjapon et al. [14]. This suggests that increased platelet size is a result of increased marrow activity and the release of younger platelets into the circulation in response to peripheral destruction. When platelet breakdown is expedited, platelet indices associated with platelet size and variability

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tend to rise, which can be explained by this physiological mechanism.

MPV is one of the most accurate platelet metrics for differentiating between these two etiological categories, according to a number of researchers. MPV was discovered by Shetageri et al. to have the highest sensitivity and specificity among platelet indices in distinguishing between hyper-destructive and hypo-productive thrombocytopenia [15]. This finding supports MPV's role as a useful preliminary diagnostic indicator in routine hematological evaluation. Similarly, Chintalapudi et al. reported that platelet volume parameters, including MPV and platelet large cell ratio (P-LCR), demonstrated superior discriminatory power between hypo-productive and hyper-destructive thrombocytopenia [16**].** This can be explained by the fact that P-LCR reflects the proportion of larger, immature platelets (>12 fL), which are released into circulation during increased bone marrow activity in response to peripheral platelet destruction. Therefore, higher P-LCR values are typically observed in hyper-destructive thrombocytopenia, enhancing its ability to differentiate between the two mechanisms. [16] The idea that bigger platelets are usually generated from the bone marrow during enhanced platelet turnover a feature of peripheral destruction illnesses such immune thrombocytopenic purpura is supported by these data.

It has also been demonstrated that platelet distribution width (PDW), which represents the variation in platelet size, might offer helpful diagnostic data. In hyper-destructive thrombocytopenia, studies assessing platelet indices have shown noticeably higher PDW values, indicating increased heterogeneity in platelet size due to rapid platelet synthesis and discharge of immature platelets [4]. PDW's usefulness as a supportive marker in differentiating between hypo-productive and hyper-destructive groups was further supported by Baig et al.'s observation of a substantial difference in PDW between these groups [17]. Therefore, the greater PDW values seen in these individuals may be related to the increased variability in platelet shape seen in destructive diseases.

Plateletcrit (PCT) seems to have a more restricted role in differentiating between various reasons of thrombocytopenia, despite the diagnostic utility of MPV and PDW. Platelet count, not platelet size or variability, is the main factor influencing PCT, which is the total platelet mass in the blood. According to earlier research, PCT's diagnostic use in etiological distinction is limited since it does not consistently demonstrate substantial differences between hypo-productive and hyper-destructive thrombocytopenia [16]. Therefore, rather than identifying the fundamental cause of thrombocytopenia, PCT might be more helpful in evaluating total platelet biomass.

Platelet indices should not be used in place of final diagnostic investigations, even though they provide a quick and affordable method for the initial classification of thrombocytopenia. Variability in these indices' sensitivity and specificity across various populations and clinical situations has been observed in some research. For instance, Aslam et al. noted that bone marrow testing is still the most reliable way to determine the origin of thrombocytopenia when clinical doubt exists and found that MPV alone may have limited diagnostic accuracy [18 "As a result, peripheral smear analysis, clinical observations, and other laboratory investigations should be considered when interpreting platelet indices [18]. In addition, platelet indices are significantly influenced by pre-analytical variables. The type of anticoagulant used during sample collection plays a crucial role; EDTA, the most commonly used anticoagulant, is known to cause time-dependent platelet swelling, which may lead to falsely elevated MPV values. It may also induce platelet clumping, resulting in pseudothrombocytopenia. Citrate, on the other hand, can produce relatively lower MPV values due to dilutional effects. Furthermore, delays between sample collection and analysis can further alter platelet morphology and indices [19–21].

In the clinical context, anticoagulant therapies such as heparin may also affect platelet counts and function, particularly in conditions like heparin-induced thrombocytopenia, thereby influencing platelet indices. Hence, these factors must be carefully considered to avoid misinterpretation. Therefore, platelet indices should always be interpreted in conjunction with peripheral smear findings and clinical correlation

The accuracy and repeatability of platelet counts may be impacted by several variables. However, platelet indices can be useful supplements in the first assessment of thrombocytopenic patients and may assist determine whether more invasive tests are necessary when read properly in a suitable therapeutic setting.

Overall, the results of this study are consistent with previous research showing that platelet indices, specifically MPV and PDW, can help distinguish between hyper-destructive and hypo-productive thrombocytopenia. They are practical and affordable instruments for early diagnostic evaluation because they are available as part of standard complete blood count testing. By using these indices in routine hematological evaluation, physicians may be able to reduce the need for needless intrusive procedures, improve patient care, and narrow the differential diagnosis.

CONCLUSION

This study shows that the assessment of thrombocytopenia can be aided by platelet indices derived from standard automated hematology analyzers. The utility of mean platelet volume (MPV) and platelet distribution width (PDW) in differentiating between hypo-productive and hyper-destructive thrombocytopenia was demonstrated. Because the bone marrow releases more big, immature platelets, hyper-destructive diseases were more often linked to elevated MPV and PDW values. On the other hand, hypo-productive diseases showed comparatively lower values, indicating decreased platelet production. However, plateletcrit (PCT) had little diagnostic use and

did not demonstrate a meaningful difference. According to these results, MPV and PDW may be helpful supporting indicators for the first assessment of thrombocytopenia. They offer a quick and economical method for preliminary evaluation because they are easily accessible in regular complete blood count results. In patients with thrombocytopenia, platelet indices can generally aid in better early diagnostic decision-making.

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