RJNMSR
Review Journal of Neurological & Medical Sciences Review

Volume 4, Issue 6, 2026

ROLE OF MEAN PLATELET VOLUME IN DIFFERENTIATING CAUSE OF THROMBOCYTOPENIA IN ADULTS: A STUDY FROM LAHORE, PAKISTAN

[1]Sameen Amjad, [2]Khadija Tariq, [3]Fiza Javed, [4]Mian Muhmmad Abu Bakar, [*5]Muhammad Ahsan Riaz, [6]Ayesha Riaz, [7]Muhammad Arshad

[1]Riphah College of Rehabilitation & Allied Health Sciences, Riphah International University, Lahore, Pakistan.

[2]Riphah College of Rehabilitation & Allied Health Sciences, Riphah International University, Lahore, Pakistan.

[3]Riphah College of Rehabilitation & Allied Health Sciences, Riphah International University, Lahore, Pakistan.

[4]Riphah College of Rehabilitation & Allied Health Sciences, Riphah International University, Lahore, Pakistan.

[*5]Department of Biotechnology, University of Okara, Okara, Pakistan.

[6]Department of Zoology, Government College, Women University Faisalabad, Faisalabad, Pakistan.

[7]Department of Biotechnology, University of Okara, Okara, Pakistan.

[*5]ahsanenv38@gmail.com

DOI: https://doi.org/10.5281/zenodo.21134296

Received	Accepted	Published
14 May, 2026	20 June, 2026	**22 June,** 2026

ABSTRACT

Background: Thrombocytopenia is a common clinical problem characterized by a low platelet count. Mean platelet volume (MPV), an indicator of platelet morphology and maturity, has shown potential for diagnosing the etiology of thrombocytopenia. However, its use in clinical practice remains limited.Objective: The objective of this study was to investigate the correlation between MPV and the etiology of thrombocytopenia, aiming to determine its usefulness as a diagnostic marker. Methods: A study was conducted, involving 80 thrombocytopenic patients. Clinical diagnosis, platelet counts, and MPV values were recorded for each patient. Peripheral blood smears were examined for the presence of mega-platelets. Cases were categorized into Group A (Hypo- production etiology) or Group B (hyper-destructive etiology). Results: There is significant difference seen in MPV values in all groups. Group B (Over-destructive) comprised 40 cases, with 35 cases demonstrating an MPV value above 10.5 fl. The remaining cases in Group B, although not prominently raised, showed the presence of mega-platelets on peripheral blood smears. Group A (Under-production) included 40 cases, with 38 which were not associated with a high MPV value. Receiver operating characteristic curve analysis determined a cutoff MPV value of 8.5 fl, offering maximum sensitivity and specificity in distinguishing hyper-destructive thrombocytopenia cases from others. Conclusion: MPV is an accurate platelet parameter that can aid in diagnosing the etiology of thrombocytopenia. It provides less invasive alternative to bone marrow examination for patients. Considering MPV, along with other platelet indices, may enhance diagnostic accuracy and patient management in thrombocytopenia cases.

Keywords: *Thrombocytopenia, mean platelet volume, hyper-destruction, hypo-production, diagnostic marker.*

Introduction

Platelets, also referred to as thrombocytes, are small fragments of irregularly shaped cells found in the blood. Their primary function is to maintain hemostasis, which is the process of preventing excessive bleeding after an injury[1]. Platelets are produced through thrombopoiesis in the bone marrow, where large precursor cells called megakaryocytes undergo maturation and release thousands of platelets into the bloodstream. Platelets have a circulating lifecycle of expected 7-10 days before they are eliminated by the spleen[2]

Despite not being complete cells, platelets possess crucial structures that are integral to the process of hemostasis. Surface proteins enable platelets to adhere to damaged blood vessel walls and to each other [3].Granules within platelets contain proteins necessary to form a solid clot that seals ruptured blood vessel. Additionally, Platelets possess muscle-like proteins that enable them to undergo shape changes when they adhere to surfaces and become adhesive[4].

Platelets are crucial for maintaining or keeping hemostasis. When a blood vessel sustains an injury, platelets adhere to the exposed collagen fibers of the vessel wall via specific receptors, such as the glycoprotein Ib/IX/V complex that interacts with von Wasebrand factor[5].This interaction triggers a series of events where platelets undergo changes in shape and release. various chemical substances, such as ADP and thromboxane A2. These substances further enhance platelet activation and aggregation. These processes culminate in the formation of a platelet plug that seals the injury site and prevents further blood loss[6]. Thrombocytopenia is a medical condition characterized by an abnormally low platelet count found in the blood, indicating a low number of platelets[7]. A platelet count below 150,000/µl is considered diagnostic for thrombocytopenia. This condition can arise from either reduced platelet production or increased platelet destruction, leading to an imbalance in the regulation of platelet levels. Accurately identifying and effectively managing thrombocytopenia relies on a comprehensive understanding of its underlying causes[8].

Disseminated intravascular coagulation (DIC) is a complex and potentially life-threatening condition characterized by widespread activation of the blood coagulation system, leading to the formation of blood clots throughout the body. Thrombocytopenia, or low platelet count, is commonly observed in DIC and plays a significant role in the underlying mechanism of the condition [9]. TTP is a rare and potentially life in threatening blood disorder characterized by abnormal blood clot formation throughout the body. Thrombocytopenia, or low platelet count, is a significant aspect of TTP. TTP primarily arises from a deficiency or dysfunction of ADAMTS13, responsible for breaking down von Wasebrand factor (vWF) in the blood [10].

It has been observed that mean platelet volume (MPV) is increased in thrombocytopenia associated with various cancers, including solid tumors and hematologic malignancies like lymphoma and leukemia. Multiple factors, such as increased platelet production and turnover, as well as the presence of cytokines and growth factors generated by tumor cells that promote platelet production, may contribute to the elevated MPV in these cases [11].

The rationale for conducting this study stems from the considerable challenge faced in accurately identifying the underlying cause of thrombocytopenia in adults. Thrombocytopenia, characterized by a reduction in platelet count, can originate from various sources such as immune-mediated disorders, bone marrow dysfunction, infections, drug- induced reactions, and systemic conditions. Present diagnostic methods for determining the cause of thrombocytopenia typically involve extensive laboratory investigations and clinical evaluations, entailing significant time and expense. MPV is a parameter that provides insights into the average size of circulating platelets and serves as an indicator of platelet production and activation. Recent research has suggested that MPV holds potential as a discriminatory tool for identifying the underlying cause of thrombocytopenia. However, the existing body of research focusing specifically on the diagnostic utility of MPV in adult patients with thrombocytopenia is limited. Consequently, the primary objective of this study is to explore the effectiveness of MPV in differentiating the causes of thrombocytopenia in adults. By conducting a comprehensive analysis of a large cohort of adult patients diagnosed with

thrombocytopenia, this study seeks to compare MPV values across different etiologies. The aim is to ascertain whether MPV can be established as a reliable and efficient diagnostic marker for identifying the underlying cause of thrombocytopenia. The potential clinical implications of these findings are substantial, as they could offer a non-invasive and readily accessible means of early and accurate differentiation of thrombocytopenia causes. Consequently, this could enable healthcare professionals to promptly implement targeted management strategies for affected patients. In conclusion, the primary goal of this study is to investigate the role of Mean Platelet Volume in distinguishing the causes of thrombocytopenia in adults. By advancing the existing knowledge base and potentially refining diagnostic practices in this patient population, this research holds the potential to enhance patient care and outcomes. It aims to facilitate more precise and timely interventions for individuals with thrombocytopenia, thereby contributing to improved medical practice.

Materials and Methods

Study Design and Setting

This is a descriptive, cross-sectional study carried out at Allama Iqbal Medical College and Mayo Hospital, Lahore, Pakistan for 4 months after getting approval from Institutional Review Board (IRB).

Selection and grouping of participants

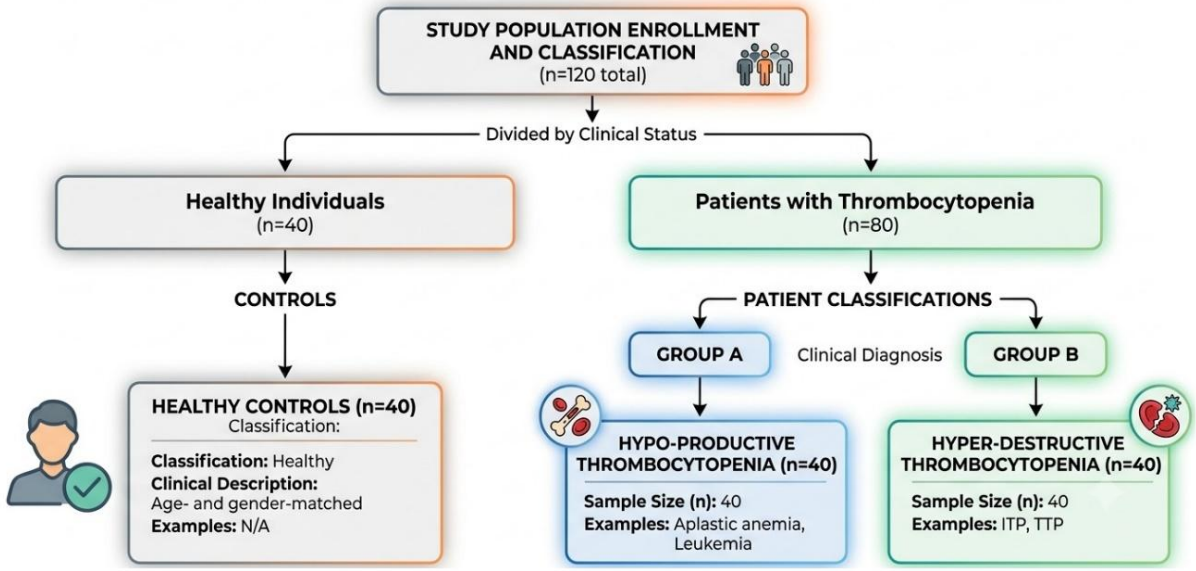


Figure 1. Flowchart illustrating the participant selection and clinical categorization of the study population (n=120) into healthy controls and subgroups of thrombocytopenia.

Exclusion Criteria: History of drug-induced thrombocytopenia, End-Stage Renal Disease (ESRD), Disseminated Intravascular Coagulation (DIC), or blood/platelet transfusion in the last month.

Laboratory and morphological analysis.

The venous blood samples (3 mL) were collected in EDTA-2K tubes and analyzed within 1-2 hours to prevent platelet swelling. Automated Parameters: Complete blood counts (CBC) and platelet profiles including platelet count, Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), and Platelet-Large Cell Ratio (PLCR)—were measured using a Sysmex XN-1000 automated hematology analyzer (Sysmex Corp., Japan).

For microscopic validation used methanol fixed peripheral blood films stained with Leishman's stain were examined under Olympus BX53 microscope (100× oil immersion) for morphology and to exclude the possibility of pseudothrombocytopenia.

For bone marrow evaluation if clinically indicated, bone marrow aspirates and trephine biopsies were performed to evaluate marrow cellularity and density of megakaryocytes as a means of confirming etiologies.

Statistical Analysis

SPSS version 26.0 and MedCalc version 20.0 were used for data analysis. Data for continuous variables were presented as Mean and Standard Deviation. Independent samples t tests or Mann Whitney U tests were used to test for inter-group differences depending on normality of the data, which was tested with P value < 0.05. The Receiver Operating Characteristic (ROC) curve analysis and Youden's index were used to identify the best cutoff of MPV for the distinction between hyper-destructive and hypo-productive thrombocytopenia.

Results

In the result of present s study a total of 120 subjects were evaluated, 80 patients with thrombocytopenia and 40 healthy controls (Figure.2). The control group was mainly male (62.5% vs 37.5% females) while the patient group was more evenly split with 53.8% males (n = 43) and 46.3% females (n = 38). When evaluated according to bone marrow smear, the patient group had moderate cellularity in 51.9%, hypo-cellularity in 7.4% and hyper-cellularity in 39.5% of the patients (Figure.2). Hemoglobin (HB) levels were comparable between Control (12.7 ± 1.0 g/dL) and Over-Destructive groups (12.6 ± 1.2 g/dL, p = 0.625) with significant reduction in Under-Production group (8.2 ± 1.0 g/dL, p < 0.0001). There was a large variation in the White Blood Cell (WBC) counts between the cohorts. Over-Destructive group had 14.0 ± 1.2 x 10^3/µL significantly higher while Under-Production group had 2.8 ± 2.8 x 10^3/µL significantly lower than the Control mean of 8.3 ± 1.7 x 10^3/µL (p < 0.0001) as shown in Figure (4.a).

Further differentiation of the underlying causes of the condition were made by platelet counts and indices. The mean Platelet count was 246.8 ± 88.3 x 10^3/µL for the Control group, which was slightly higher in the Over-Destructive group (281.2 ± 95.7 x 10^3/µL, p = 0.046) and markedly depleted in the Under-Production group (60.2 ± 36.5 x 10^3/µL, p < 0.0001). Importantly, there were highly significant differences (p < 0.0001, for both comparisons) in Mean Platelet Volume (MPV). The MPV of the Control group was 8.7 ± 1.0 fL and significantly lower in the Over-Destructive group (7.5 ± 0.5 fL), while the Under-Production group had a significantly higher MPV (9.7 ± 1.1fL) as shown in Figure (4.b).

There were also marked pathological alterations of the distribution and cell ratio parameters. The Control group had a mean PDW of 30.3 ± 50.3%, which was significantly decreased in the Over-Destructive group 12.3 ± 2.1%, p = 0.003 and the Under-Production group 10.7 ± 1.6%, p = 0.003. The Platelet Large Cell Ratio (PLCR) in the Control group was 29.6 ± 10.4%, increased significantly in the Over-Destructive group (41.7 ± 9.0%, p < 0.0001) and decreased significantly in the Under-Production group (22.7 ± 6.0%, p < 0.0001).These morphologically confirmed by peripheral blood films; films of patients showed an abundance of megakaryocytes and large megaplatelets while films of controls were uniform and normal as shown in Figure.5.

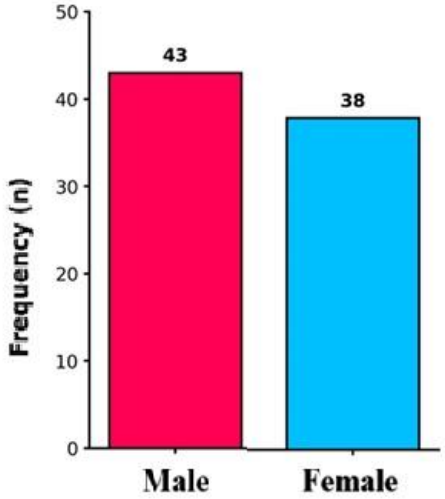


Figure 2. Gender Distribution in Thrombocytopenia Patients and Control Group.

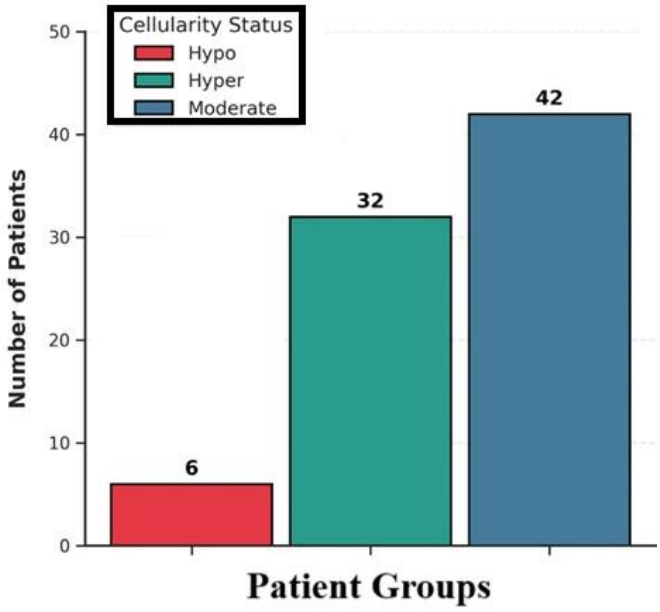


Figure 3: Distribution of cellularity in bone marrow smears across patient groups.

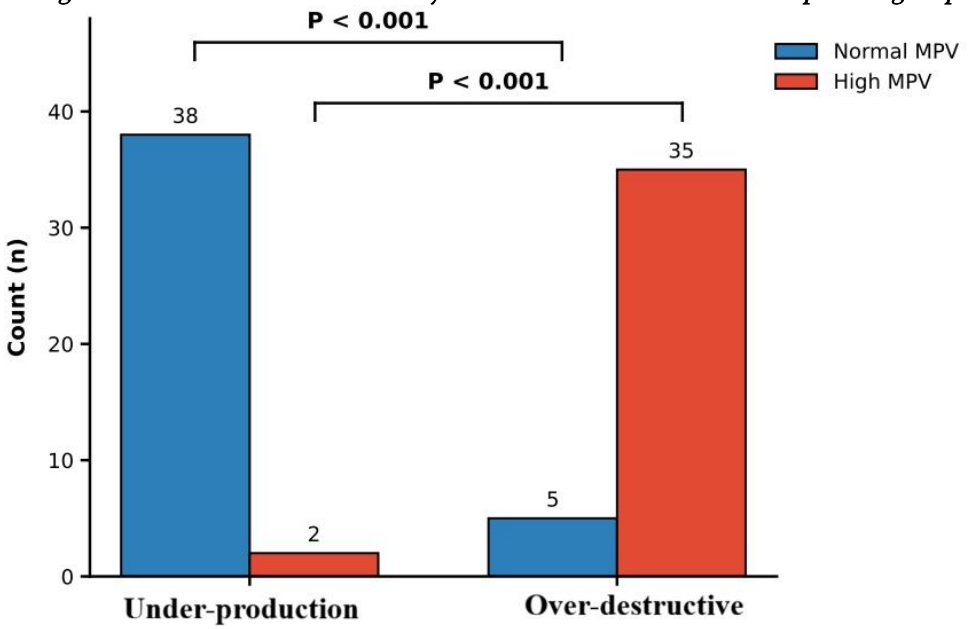
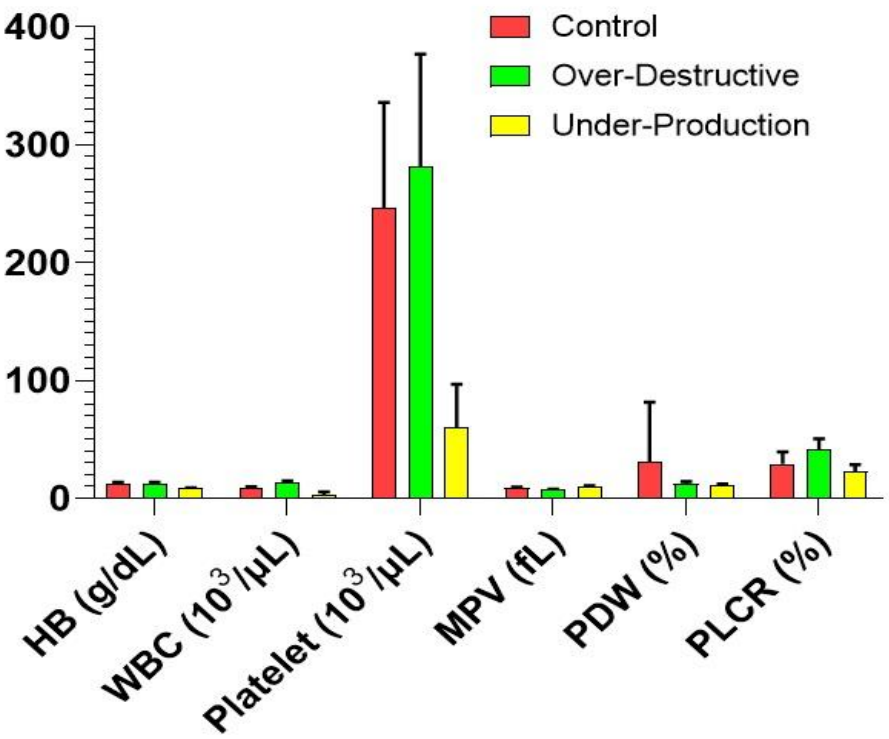


Figure 4: Distribution of Mean Platelet Volume (MPV) values in under-production and over-destructive patient groups.

a.



b.

Variable	Control	Over-Destructive		Under-Production	
	Mean and SD	Mean and SD	p-value	Mean and SD	p-value
HB (g/dL)	12.7±1.0	12.6±1.2	0.625	8.2±1.0	<0.0001
WBC (10^3/µL)	8.3±1.7	14.0±1.2	<0.0001	2.8±2.8	<0.0001
Platelet (10^3/µL)	246.8±88.3	281.2±95.7	0.046	60.2±36.5	<0.0001
MPV (fL)	8.7±1.0	7.5±0.5	<0.0001	9.7±1.1	<0.0001
PDW (%)	30.3±50.3	12.3±2.1	0.003	10.7±1.6	0.003
PLCR (%)	29.6±10.4	41.7±9.0	<0.0001	22.7±6.0	<0.0001

Figure 4. (a) Complete Blood Count (CBC) parameters across three groups: Under-Production, Over-Destructive, and Control in study cohorts. Data are presented as mean and SD. (b) Statistical analysis in table displays the hematological parameters indices between study groups.

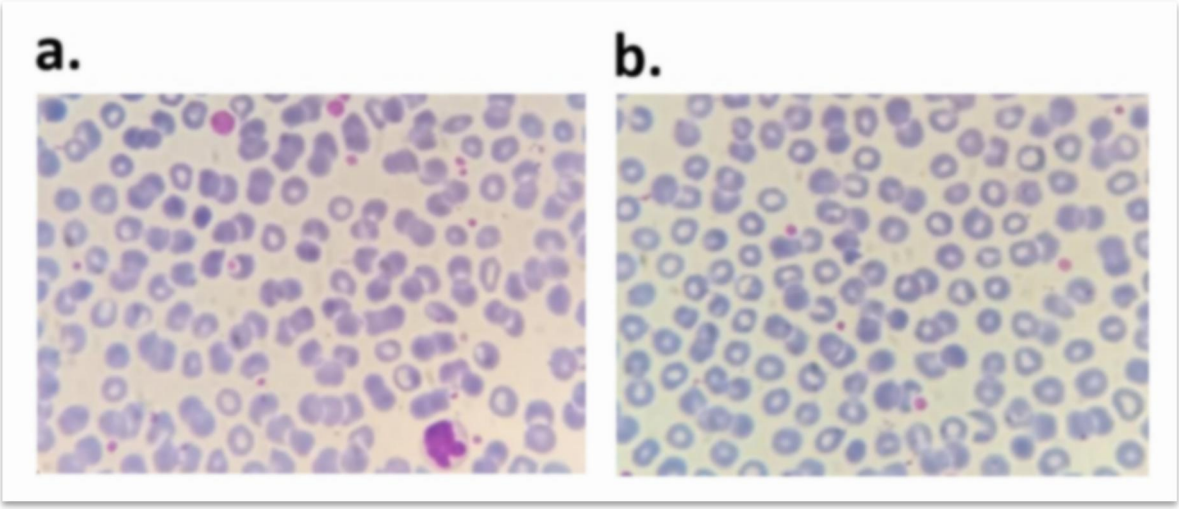


Figure 5. (a) Peripheral blood smear from a thrombocytopenic patient showing the presence of megakaryocytes and large platelets (megaplatelets). (b) Peripheral blood smear from a healthy individual showing normal platelet morphology. Scale bar = 10 µm.

Discussion

The differential diagnosis of thrombocytopenia (TCP) is crucial in determining the underlying cause, as it can result from various mechanisms such as platelet destruction, hypo- production. The current gold standard for diagnosing TCP is bone marrow (BM) examination, which is invasive and not recommended as a first-line diagnostic procedure. Therefore, there is a need for a simple, inexpensive, and non-invasive method to differentiate the types of TCP. Mean platelet volume (MPV) has emerged as a potential marker for TCP diagnosis, with several studies suggesting its usefulness in differentiating and categorizing TCP[12]. In this study, we aimed to evaluate various parameters and their significance in differentiating between under-production and over-destructive thrombocytopenia (TCP) in a cohort of 80 patients, along with 40 controls. Our analysis included demographic characteristics, age distribution, platelet counts, mean platelet volume (MPV), laboratory parameters, and diagnostic cases. According to previous studies had no significance difference seen on Hb parameters on both patient groups: Under-production and over-destructive[13]. In our study, we found a significant difference on both patient group: Group A (Under-production) vs Group B (Over- destructive), Group A vs Control. But there is no significant difference seen in Group B vs Control group presented in table 3 and 4.

Present study's findings revealed that the most frequently observed conditions in both the under- production and over-destructive groups were anemia, leukemia, immune thrombocytopenia (ITP), and thrombotic thrombocytopenic purpura (TTP). These results are consistent with existing knowledge on the common causes of thrombocytopenia, which adds credibility to the study's validity. Moreover, other studies conducted by Bhalara et al. [14] , Rehman et al., and Patel et al. [15] have also reported similar patterns in the most common causes of thrombocytopenia. Bhalara et al. identified Dengue fever as the leading cause among thrombocytopenia patients. Meanwhile, Rehman et al. and Patel et al. found malaria as the primary cause of thrombocytopenia, followed by other infections such as dengue [12]. The present study MPV shows significantly higher difference MPV values in the hyper-destructive group compared to the hypo-productive and normal group. These findings are consistent with previous studies that have shown an association between increased MPV and platelet destruction [16]. The mechanism behind the increased MPV in platelet destruction can be attributed to the active compensation of the bone marrow, resulting in the release of young and larger platelets. In contrast, patients with TCP due to lack of platelet production have similar MPV values to those with normal blood cell counts. These observations support the concept that larger platelets are functionally more active than smaller ones, as they are metabolically and enzymatically more active. This finding is in line with previous research emphasizing the

importance of MPV in reflecting platelet size and function [17]. The discrepancies in these findings may be attributed to differences in patient selection, the type of hematology analyzer used, and the inclusion of other platelet indices such as PDW and PLCR. Further studies combining various platelet indices may provide more promising results and a clearer understanding of their diagnostic value. Although MPV shows promise in distinguishing between hyper-destructive and hypo- productive thrombocytopenia, it is important to acknowledge the limitations of our study. We focused solely on MPV as a discriminating factor, disregarding other platelet indices. Additionally, difficulties in obtaining platelet indices in severe TCP or cases with red cell fragmentation may limit the applicability of MPV in certain situations.

Conclusion

Mean Platelet Volume (MPV) is a reliable, cost-effective, simple and non-invasive diagnostic parameter, which allows to distinguish between the primary causes of thrombocytopenia. The MPV is significantly elevated in hyper-destructive etiologies and normal in hypo-productive conditions. Combination of MPV with regular blood parameters minimizes the need for invasive BM examination, enabling prompt and focused patient management.

References

1. Lee, E.-J. and A.I.J.P.C.C.i.O.P. Lee, Thrombocytopenia. 2016. 43(4): p. 543-557.
2. Vanderschueren, S., et al., Thrombocytopenia and prognosis in intensive care. 2000. 28(6): p. 1871-1876.
3. Steéphan, F., et al., Thrombocytopenia in a surgical ICU. 1999. 115(5): p. 1363-1370.
4. Rf, B.J.N.E.J.M., Fetal thrombocytopenia and its relation to maternal thrombocytopenia. 1993. 329: p. 1463-1466.
5. Männel, D. and G.E.J.M.P. Grau, Role of platelet adhesion in homeostasis and immunopathology. 1997. 50(4): p. 175.
6. Zapata, J.C., D. Cox, and M.S.J.P.n.t.d. Salvato, The role of platelets in the pathogenesis of viral hemorrhagic fevers. 2014. 8(6): p. e2858.
7. Izak, M. and J.B.J.F.r. Bussel, Management of thrombocytopenia. 2014. 6: p. 45.
8. Lim, S.Y., et al., The incidence, causes, and prognostic significance of new-onset thrombocytopenia in intensive care units: a prospective cohort study in a Korean hospital. 2012. 27(11): p. 1418.
9. Kitchens, C.S.J.A.E.P.B., Thrombocytopenia and thrombosis in disseminated intravascular coagulation (DIC). 2009. 2009(1): p. 240-246.
10. Tsai, H.-M.J.H.o.c.o.N.A., Thrombotic thrombocytopenic purpura: a thrombotic disorder caused by ADAMTS13 deficiency. 2007. 21(4): p. 609-632.
11. Riedl, J., et al., Association of mean platelet volume with risk of venous thromboembolism and mortality in patients with cancer. 2014. 111(04): p. 670-678.
12. Gulati, I., et al., Diagnostic implication of mean platelet volume in thrombocytopenia. 2017. 10(4): p. 370-375.
13. Mala, K.G., B.J. Bhandari, and S.K.J.I.J.P.O. Kittur, Paramountcy of platelet parameters in thrombocytopenia our hospital experience. 2018. 5(4): p. 558-62.
14. Bhalara, S.K., et al., Clinical and etiological profile of thrombocytopenia in adults: A tertiary-care hospital-based cross-sectional study. 2015. 4(1): p. 7-10.
15. ur Rehman, Z., Clinical spectrum of thrombocytopenia in adult population of Karachi. 2001.
16. Shah, A.R., S.N. Chaudhari, and M.H.J.H. Shah, Role of platelet parameters in diagnosing various clinical conditions. 2011. 89: p. 11-13.
17. Ahmed, Y., et al., Retrospective analysis of platelet numbers and volumes in normal pregnancy and in pre-eclampsia. 1993. 100(3): p. 216-220.