Section: Pathology

Original Research Article

REVISITING THROMBOCYTOPENIA: DIAGNOSTIC INSIGHTS FROM PLATELET INDICES

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ABSTRACT

Background: Thrombocytopenia is a common hematological disorder caused by increased platelet destruction or reduced platelet production. Platelet indices help in differentiating the underlying mechanism of thrombocytopenia. **Materials and Methods:** This hospital-based observational study was conducted on 500 thrombocytopenic patients and 100 healthy controls over 18 months. EDTA blood samples were analyzed using a Automated 5 - part Mindray (BC- 760) automated hematology analyzer. Platelet indices including platelet count, Mean platelet volume (MPV), Platelet distribution width (PDW), Platelet large cell ratio (P-LCR), Immature platelet fraction (IPF) and Plateletcrit (PCT) were evaluated." **Results:** Most patients belonged to the fifth decade group comprising of (23.6%), with slight male predominance (51.6%), where thrombocytopenia was catergorized in 3 groups- Mild, Moderate and Severe. Moderate thrombocytopenia was most common (40.6%). Thrombocytopenia was divided in 2 groups – Hyperdestructive and Hypoproductive. Hyperdestructive thrombocytopenia constituted 60.6% of cases, while hypoproductive thrombocytopenia accounted for 39.4%. MPV, P-LCR, and IPF were significantly higher in hyperdestructive thrombocytopenia ($p < 0.001$). Immune thrombocytopenic purpura and infections were the main hyperdestructive causes, whereas megaloblastic anemia and pancytopenia were the major hypoproductive causes. **Conclusion:** MPV, P-LCR, and IPF are useful, rapid, and non-invasive markers for differentiating hyperdestructive from hypoproductive thrombocytopenia.

INTRODUCTION

A "platelet count of less than 150,000/mm³ is known as thrombocytopenia, and it is a frequent hematological anomaly linked to a number of clinical diseases.[1] Hemostasis, thrombosis, endothelial integrity, and vascular healing all depend on platelets, which are tiny cytoplasmic fragments produced from bone marrow megakaryocytes.[1] They have a volume of 4.5–11 fL, a diameter of roughly 3–5 µm, and a survival period of 7–10 days in circulation.[2]

Platelets play an important role in clot formation by adhering to injured vessel walls and providing phospholipid surfaces for coagulation factor activation.[3] In addition to hemostasis, platelets are involved in inflammation, immune defense, wound healing, angiogenesis, and tissue remodeling.[4] Activated platelets release cytokines, chemokines, growth factors, and antimicrobial substances that influence leukocytes, endothelial cells, and inflammatory responses.[5] They also promote leukocyte migration and generate reactive oxygen species during inflammation.[6,7]

The severity of thrombocytopenia is clinically important because severe reduction in platelet count, particularly below 10,000/mm³, can lead to life-threatening spontaneous gastrointestinal and intracranial hemorrhage.[1] Increased platelet destruction, reduced platelet synthesis, dilutional thrombocytopenia or aberrant splenic sequestration are the main causes of thrombocytopenia.[8] Conditions like immune thrombocytopenic purpura, dengue, malaria, and disseminated intravascular

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coagulation can cause hyperdestructive thrombocytopenia.[9]

Hypoproductive thrombocytopenia occurs in bone marrow disorders such as aplastic anemia, megaloblastic anemia, leukemia, myelodysplastic syndrome, and following chemotherapy or radiation.[8,10] The gold standard for determining the etiology of thrombocytopenia is still bone marrow aspiration, but this procedure is invasive, expensive, time-consuming, and bleeding-prone.[11]

Therefore, there is a need for simple and non-invasive methods for preliminary evaluation. Platelet indices including Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), Plateletcrit (PCT), Platelet Large Cell Ratio (P-LCR), and Immature Platelet Fraction (IPF) are provided by automated hematology analyzers.[12,13] Platelet size is represented by MPV, platelet size variation is indicated by PDW, the percentage of large platelets is measured by P-LCR, total platelet mass is represented by PCT, and newly released platelets from the bone marrow are represented by IPF.[15-18]

In hyperdestructive thrombocytopenia, increased marrow activity releases larger and younger platelets, resulting in higher MPV, PDW, P-LCR, and IPF values. In contrast, these indices are usually lower in hypoproductive thrombocytopenia.[8,15] Automated platelet indices are rapid, cost-effective, reproducible, and free from observer bias compared to manual methods.[14] Hence, these indices may serve as useful non-invasive markers in differentiating the underlying causes" of thrombocytopenia.

Therefore, the present study titled "Study of Platelet Indices in Evaluation of Thrombocytopenia" was undertaken to assess the role of platelet indices in differentiating the causes of thrombocytopenia and their utility in routine clinical practice.

MATERIALS AND METHODS

After receiving approval from the Institutional Ethics Committee, the current hospital-based observational study, "Study of Platelet Indices in Evaluation of Thrombocytopenia," was carried out over the course of eighteen months in the Department of Pathology at Muzaffarnagar Medical College in Muzaffarnagar, Uttar Pradesh. Regardless of whether clinical bleeding symptoms were present or not, the study included Muzaffarnagar Medical College patients with thrombocytopenia, which is defined as a platelet count of less than 1,50,000/cumm. 500 EDTA blood samples [cases] and 100 EDTA blood samples from healthy, normal people served as the control.

Inclusion Criteria

- All patients presenting with thrombocytopenia (platelet count <1,50,000/cumm).

Exclusion Criteria

- History of platelet transfusion within 15 days prior to blood sample collection.
- Pseudothrombocytopenia.
- Pregnant and lactating females.
- Patients receiving antiplatelet drugs or any medications causing thrombocytopenia.
- Patients undergoing chemotherapy or radiotherapy.

Study Procedure

Trained laboratory staff collected venous blood samples while adhering to stringent aseptic procedures. Blood was drawn into dipotassium EDTA vacutainers for complete blood count analysis. For phlebotomy, a tourniquet was applied above the venipuncture site and the area disinfected with 70% alcohol. Blood was drawn from a prominent vein using a syringe or evacuated tube system, with the tourniquet released promptly after venous access. Gentle withdrawal was ensured to prevent platelet activation. Anticoagulated samples were mixed by gentle inversion and properly labeled.[19] All samples were processed using an automated 5-part hematology analyzer, Mindray BC-760. Analysis was performed within 30 minutes of blood collection to minimize falsely elevated mean platelet volume values due to EDTA-induced platelet swelling

Peripheral blood smear examination was performed for all samples to assess the general blood picture and exclude pseudothrombocytopenia.[20] Blood films were prepared from direct blood sample and staining with Romanowsky (Leishman) stain using the wedge method.

Statistical Analysis

Microsoft Excel "was used to collate the gathered data, while SPSS version 25 was used for statistical analysis. Calculations were made for descriptive statistics such mean, standard deviation, frequency, and percentage. A p-value of less than 0.05 was deemed statistically significant, and appropriate" statistical tests were used as necessary.

RESULTS

The study included 100 healthy controls and 500 thrombocytopenic patients. Thrombocytopenia was seen in people ranging in age from 11 to 94. The age "group of 41–50 years old accounted for 118 cases (23.6%), followed by the age group of 31–40 years old with 94 cases (18.8%) and the age group of 21–30 years old with 86 cases (17.2%). Elderly patients above 70 years constituted a relatively smaller proportion of the study population [Table 1].

Male patients constituted 258 cases (51.6%), while females constituted 242 cases (48.4%), showing a slight male predominance. The gender distribution of thrombocytopenic patients is presented in [Table 1]. Thrombocytopenia was divided into three categories based on platelet count: mild, moderate, and severe. The most frequent manifestation, observed" in 203 individuals (40.6%), was moderate thrombocytopenia, which was followed by mild thrombocytopenia in 196 patients (39.2%). 101 patients (20.2%) had severe thrombocytopenia [Table 1].

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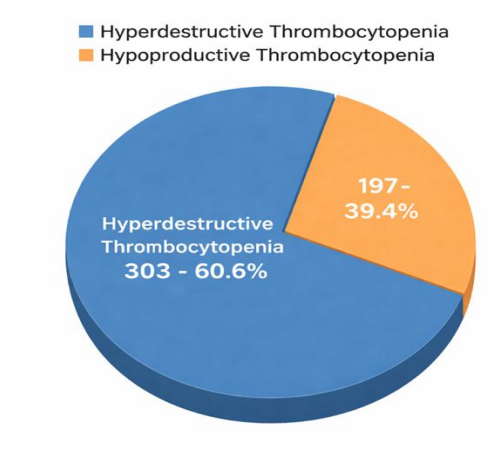
Table 1: Demographic and Clinical Profile of Patients with Thrombocytopenia (N = 500)

Age Group (Years)	Number of Patients	Percentage (%)
11–20	11	2.2
21–30	86	17.2
31–40	94	18.8
41–50	118	23.6
51–60	75	15.0
61–70	64	12.8
71–80	42	8.4
81–90	9	1.8
91–100	1	0.2
Gender		
Male	258	51.6
Female	242	48.4
Severity of Thrombocytopenia		
Mild thrombocytopenia	196	39.2
Moderate thrombocytopenia	203	40.6
Severe thrombocytopenia	101	20.2

When thrombocytopenia was categorized based on the underlying mechanism, 303 individuals (60.6%) had hyperdestructive thrombocytopenia, while 197 patients (39.4%) had "hypoproductive thrombocytopenia. The hyperdestructive group had a slightly lower mean platelet count (0.85 ± 0.39 $\times 10^1$/L) than the hypoproductive group (0.91 ± 0.37 $\times 10^1$/L), but this difference was not statistically significant (p = 0.07).The mean platelet volume was statistically considerably (p < 0.001) greater in hyperdestructive thrombocytopenia (13.36 ± 2.05 fL) than in hypoproductive thrombocytopenia (11.28 ± 1.34 fL). Additionally, the hyperdestructive group's platelet distribution width was somewhat smaller (16.44 ± 1.24%) than the hypoproductive group's (16.66 ± 0.69%), with statistical significance (p = 0.02). There was no discernible difference in plateletcrit between the two groups. In hyperdestructive thrombocytopenia, the mean plateletcrit was 1.26 ± 13.07%, while in hypoproductive thrombocytopenia, it was 0.10 ± 0.05% (p = 0.21). Hyperdestructive thrombocytopenia had a much greater platelet large cell ratio (52.12 ± 11.68%) than hypoproductive thrombocytopenia (37.43 ± 8.33%), with a very significant difference (p < 0.001).

Similarly, immature platelet fraction was significantly higher in hyperdestructive thrombocytopenia (16.45 ± 8.04%) than in hypoproductive thrombocytopenia (5.22 ± 2.16%), with a p-value of less than 0.001. These findings indicate that MPV, P-LCR, and IPF are" valuable markers in differentiating hyperdestructive from hypoproductive thrombocytopenia. [Table 2]



The etiological distribution of hyperdestructive thrombocytopenia (N = 303) in the present study revealed that Immune Thrombocytopenic Purpura (ITP) was the most common cause, accounting for 115 cases (37.9%), followed closely by 110 cases of bacterial infections (36.3%) that were blood culture positive. Viral infections (Dengue) contributed to 40 cases (13.2%), while malaria was identified in 22 cases (7.2%) and Disseminated Intravascular Coagulation (DIC) accounted for 16 cases (5.3%). Overall, immune-mediated and infectious causes constituted the majority of hyperdestructive thrombocytopenia, highlighting the significant role of both immunological mechanisms and infections in platelet destruction.

Among patients with hypoproductive "thrombocytopenia, megaloblastic anemia was the most common cause, seen in 125 patients (63.4%). Leukemia in 32 cases (16.2%), myelodysplastic syndrome in 17 cases (8.7%), myelofibrosis in 15 cases (7.7%), and bone marrow infiltration in 8 cases (4%). The etiological distribution highlights that immune-mediated and infection-related causes were predominant in hyperdestructive thrombocytopenia, whereas nutritional "deficiency and marrow-related disorders were the major causes of hypoproductive thrombocytopenia. [Table 4]

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Table 2: Statistical Comparison of Platelet parameters within groups

Variable	Hyper (Mean ± SD)	Control (Mean ± SD)	p-value	Hypo (Mean ± SD)	Control (Mean ± SD)	p-value	Hyper (Mean ± SD)	Hypo (Mean ± SD)	p-value
PLT	0.85 ± 0.39	2.42 ± 0.80	<0.001	0.91 ± 0.37	2.42 ± 0.80	<0.001	0.85 ± 0.39	0.91 ± 0.37	0.07
MPV	13.36 ± 2.05	11.90 ± 2.26	<0.001	11.28 ± 1.34	11.90 ± 2.26	0.003	13.36 ± 2.05	11.28 ± 1.34	<0.001
PDW	16.44 ± 1.24	16.32 ± 1.27	0.403	16.66 ± 0.69	16.32 ± 1.27	0.002	16.44 ± 1.24	16.66 ± 0.69	0.02
PCT	1.26 ± 13.07	0.29 ± 0.08	0.456	0.10 ± 0.05	0.29 ± 0.08	<0.001	1.26 ± 13.07	0.10 ± 0.05	0.21
P-LCR	52.11 ± 11.68	41.68 ± 12.60	<0.001	37.43 ± 8.33	41.68 ± 12.60	0.001	52.12 ± 11.68	37.43 ± 8.33	<0.001
IPF	16.45 ± 8.04	7.73 ± 5.01	<0.001	5.22 ± 2.16	7.73 ± 5.01	<0.001	16.45 ± 8.04	5.22 ± 2.16	<0.001

HYPER- Hyperdestruction, HYPO- Hypoproductive.

Table 3: Etiological Distribution of Hyperdestructive and Hypoproductive Thrombocytopenia

Etiology	Number of Patients	Percentage
Hyperdestructive Thrombocytopenia (N = 303)		
Immune Thrombocytopenic Purpura	115	37.9
Bacterial Infection	110	36.3
Viral infections (Dengue)	40	13.2
Malaria	22	7.3
Disseminated Intravascular Coagulation	16	5.3
Hypoproductive Thrombocytopenia (N = 197)		
Megaloblastic Anemia	125	63.4
Leukemia	32	16.2
Myelodysplastic Syndrome	17	8.7
Myelofibrosis	15	7.7
Bone Marrow Infiltration (Metastasis)	6	3.0
Bone Marrow Infiltration (Granuloma)	2	1.0

With an area under the curve (AUC) of 0.961, IPF exhibited the" best diagnostic accuracy for distinguishing hypoproductive thrombocytopenia, followed by P-LCR (AUC = 0.853) and MPV (AUC = 0.801), according to receiver operating characteristic curve analysis. PDW (AUC = 0.442) and PCT (AUC = 0.560) showed poor diagnostic ability. In hyperdestructive thrombocytopenia, inverse AUC values were observed for IPF (0.039), P-LCR (0.147), and MPV (0.199), indicating their higher values in hyperdestructive states [Figure 1 & 2].

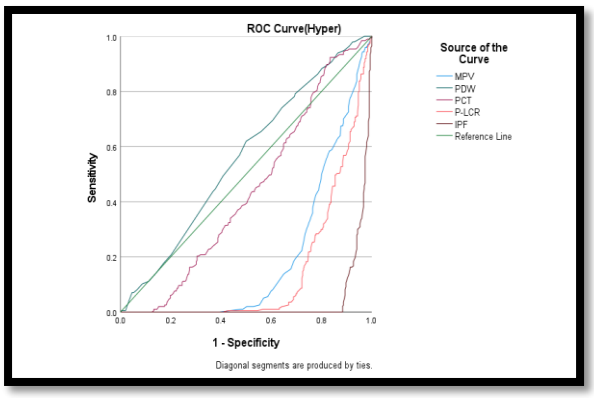

Figure 1: ROC CURVE analysis showing Hyperdestructive thrombocytopenia

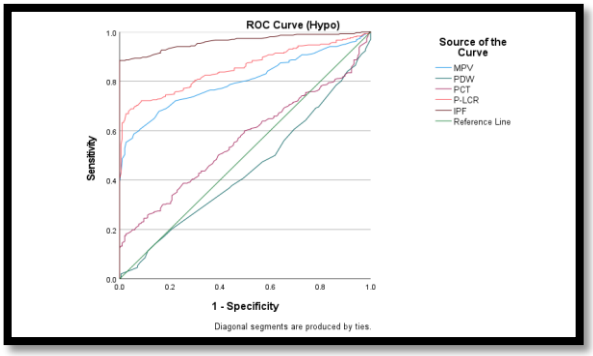

Figure 2: ROC CURVE analysis showing Hypoproductive thrombocytopenia

DISCUSSION

A frequent hematological "disorder, thrombocytopenia is caused by either diminished bone marrow synthesis of platelets or increased peripheral destruction.[2,9] Since platelet count alone does not indicate the underlying mechanism, platelet indices have emerged as useful non-invasive markers for differentiating hyperdestructive from hypoproductive thrombocytopenia.[21] The majority of patients in this study were between the ages of 41 and 50 (23.6%), followed by those between the ages of 31 and 40 (18.8%) and 21 and 30 (17.2%). Thrombocytopenia was more prevalent in young and middle-aged individuals, according to Mittal et al. and Negash et al.[8,3]

In the current study, there was a little male predominance, with 51.6% of participants being men and 48.4% being women. Borkataky et al. and

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Elsewefy et al. reported similar results, finding a virtually equal gender distribution with a slight male predominance.[9,22] Moderate thrombocytopenia was the most common presentation in the present study (40.6%), followed by mild thrombocytopenia (39.2%), while severe thrombocytopenia constituted 20.2% of cases. Similar observations were reported by Khairkar et al. and Parveen and Vimal, who found that most patients presented with mild to moderate thrombocytopenia.[4,23]

Hyperdestructive thrombocytopenia constituted 60.6% of cases, whereas hypoproductive thrombocytopenia accounted for 39.4%. Mittal et al., Borkataky et al., and Negash et al. reported a similar prevalence of hyperdestructive thrombocytopenia.[3,8,9] Hyperdestructive thrombocytopenia was more common in younger adults, while hypoproductive thrombocytopenia was more frequent in older age groups, likely due to a higher prevalence of marrow disorders.

In the current investigation, MPV dramatically rose as thrombocytopenia severity increased. Borkataky et al., Kaito et al., and Ntaios et al. found similar results, noting that MPV increases under circumstances linked to greater platelet release and increased platelet destruction.[9,24,25]

P-LCR also increased significantly with increasing severity of thrombocytopenia. Similar observations were made by Babu and Basu and Islam et al., who reported higher P-LCR values in hyperdestructive thrombocytopenia due to increased circulation of large platelets.[1,17]

IPF was much higher in hyperdestructive thrombocytopenia than in hypoproductive thrombocytopenia, and it varied significantly between severity groups. Jeon et al., Goel et al., and Naz et al. reported similar results, identifying IPF as one of the most helpful indices for distinguishing between decreased platelet production and increased platelet destruction.[18,26,27]

PDW and plateletcrit did not show significant differences" across severity groups in the present study. Khairkar et al. and Chandrashekar found similar results and came to the conclusion that PDW and PCT are not very useful in distinguishing the mechanism of thrombocytopenia.[4,28] Immune thrombocytopenic purpura was the most frequent cause of hyperdestructive thrombocytopenia, with bacterial infections coming in second. ITP was "identified by Kaito et al., Ntaios et al., and Islam et al. as a primary cause of hyperdestructive thrombocytopenia.[1,24,25]

Megaloblastic anemia was the most frequent cause of hypoproductive thrombocytopenia, followed by Leukemia. Carmel and Ishtiaq et al. found similar findings, identifying" marrow suppression and dietary insufficiency as the main reasons of hypoproductive thrombocytopenia.[29,30]

ROC analysis [Figure 1] showed that IPF had the highest discriminatory power for differentiating hypoproductive from hyperdestructive thrombocytopenia (AUC = 0.961), followed by P-LCR (0.853) and MPV (0.801), while PDW and PCT had limited utility. When hyperdestructive thrombocytopenia was set as the positive state, inverse AUCs were observed for IPF (0.039), P-LCR (0.147), and MPV (0.199), reflecting their higher values in hyperdestructive states rather than poor performance. Overall, IPF, P-LCR, and MPV are reliable markers for distinguishing the two categories.

ROC analysis - hypoproductive as positive (Figure 2) showed IPF had excellent discrimination (AUC = 0.961) with very high sensitivity and specificity. P-LCR (AUC = 0.853) and MPV (AUC = 0.801) demonstrated good diagnostic "performance, whereas PCT (0.560) and PDW (0.442) had limited utility. Similar findings were reported by Jeon et al.and Negash et al., who highlighted the strong diagnostic utility of these indices.[3,18]

CONCLUSION

Thrombocytopenia was more commonly due to hyperdestructive causes, particularly immune thrombocytopenic purpura and infections, while megaloblastic anemia and pancytopenia were the main hypoproductive causes. MPV, P-LCR, and IPF were significantly higher in hyperdestructive thrombocytopenia and proved useful in differentiating the underlying mechanism. Among these, IPF showed the highest diagnostic value. Overall findings indicated that platelet indices" can serve as rapid, inexpensive, and non-invasive adjuncts in the evaluation of thrombocytopenia.

Convenience sampling was used to perform the study at a single location, which may have limited generalizability and introduced selection bias. Not every instance had a bone marrow examination, which could have had an impact on the etiological classification. Platelet indices may also have been influenced by preanalytical factors such as EDTA-induced swelling and sample processing time. In addition, follow-up was not done, so the prognostic value of platelet indices could not be assessed.

Statements and declarations Conflicts of interest: The authors declare that they do not have conflict of interest.

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