

Study of Platelet Indices in Patients of Chronic Myeloproliferative Disorders

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Abstract:

Background: Chronic myeloproliferative disorders (CMPDs), including Polycythemia Vera (PV), Essential Thrombocythemia (ET), and Primary Myelofibrosis (PMF), are clonal hematopoietic stem cell disorders characterized by dysregulated proliferation of myeloid lineages. Platelet abnormalities are central to their pathophysiology, predisposing patients to both thrombotic and hemorrhagic complications. Automated hematology analyzers provide platelet indices such as mean platelet volume (MPV), platelet distribution width (PDW), platelet-large cell ratio (P-LCR), and plateletcrit (PCT), that can serve as inexpensive biomarkers for diagnosis and prognostication.

Methods: A prospective observational study was conducted in the Department of Hematology/Pathology, Indira Gandhi Institute of Medical Sciences (I.G.I.M.S., from April 2020 to June 2022. A total of 160 patients diagnosed with CMPDs according to the World Health Organization (WHO) 2016 criteria were enrolled. Demographic, clinical, and laboratory data were collected. Platelet indices were recorded using a fully automated hematology analyzer. Data were analyzed using ANOVA, t-tests, chi-square, and correlation analyses, with $p < 0.05$ considered significant.

Results: ET was the most common CMPD (45%), followed by PV (33.8%) and PMF (21.2%). ET patients exhibited significantly higher platelet counts compared to PV and PMF ($p < 0.001$). PMF patients demonstrated the highest MPV, PDW, and P-LCR values, reflecting abnormal megakaryopoiesis. Thrombotic events occurred in 16.3% of patients and correlated with elevated MPV and PDW, while bleeding events (8.8%) were associated with increased PDW and reduced PCT.

Conclusion: Platelet indices differ significantly among CMPD subtypes and were associated with vascular complications. They represent cost-effective, readily available markers that may complement molecular and histological investigations for diagnosis, risk stratification, and disease monitoring in CMPDs.

Keywords: Chronic myeloproliferative disorders, platelet indices, thrombocytosis, IGIMS

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Introduction

Chronic myeloproliferative disorders (CMPDs) are clonal hematopoietic stem-cell disorders characterized by excessive proliferation of one or more myeloid lineages [1]. The WHO demands PV, essential thrombocythemia (ET), and primary myelofibrosis (PMF), classical Philadelphia chromosome-negative CMPDs. JAK2 V617F mutations cause dysregulated signalling pathways that cause myeloproliferation, which is common in many diseases. CMPDs cause erythrocytosis, leukocytosis, and thrombocytosis [2]. They raise the risk of vascular issues, hemorrhagic episodes, and acute leukaemia. Chronicity and catastrophic effects make prognostic evaluation, precise monitoring, and diagnosis essential for managing these illnesses.

Platelets are crucial in CMPDs because abnormal platelet activation, function, and turnover can produce thrombotic and hemorrhagic events, notably in ET and PV [3]. According to automated haematology analysers, several platelet indices that reveal platelet shape and function may now be measured [4]. Platelet measures include crit, distribution width, platelet-large cell ratio, and mean platelet volume. MPV is a proxy measure of platelet reactivity because larger platelets are more metabolically and enzymatically active [5]. PDW increases with abnormal megakaryopoiesis, indicating platelet size heterogeneity. Haematocrit measures red blood cell mass, plateletcrit measures all platelets in the blood, and P-LCR reveals the percentage of larger platelets.

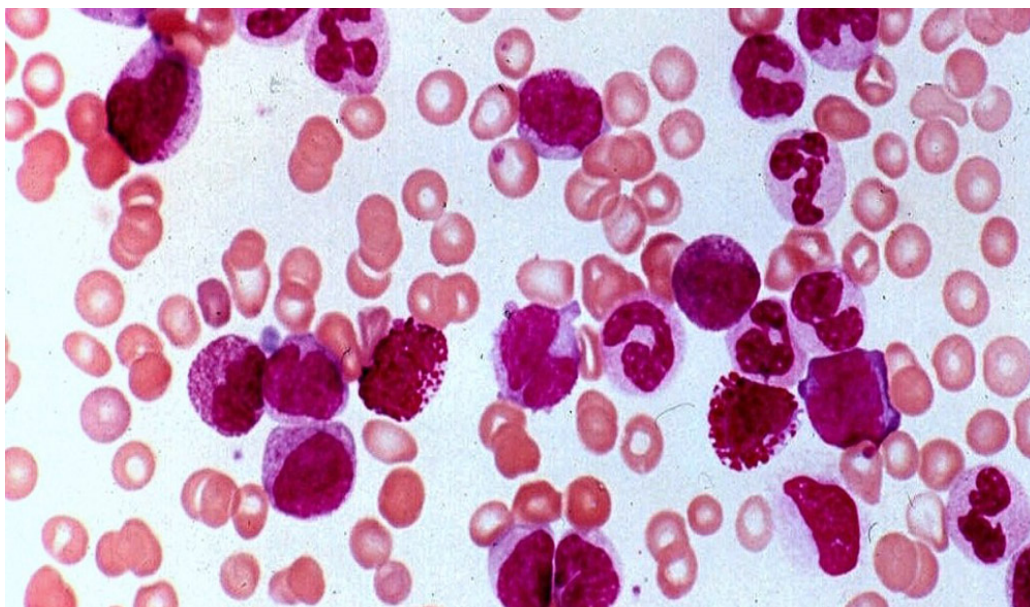


Figure 1 Chronic myeloproliferative disorders [6]

Platelet indices are attractive therapeutic indicators for haematological illnesses due to their accessibility and low cost. Changes in platelet indices in CMPDs can indicate megakaryocyte proliferation and differentiation issues. Thrombosis, which

increases MPV and PDW, is a major cause of death in CMPD patients [7]. To identify clonal thrombocythemia from reactive thrombocytosis, PCT and P-LCR may provide further prognostic information. Platelet indices can be acquired at no cost

during regular Complete Blood Count (CBC) testing, making them promising diagnostic and prognostic markers for persistent myocardial infarction CMPDs in healthcare systems with low resources [8].

Clinically and scientifically, CMPD patients' platelet indices should be examined. Clinically, CMPD patients are at paradoxical risk of thrombosis and haemorrhage since current risk classification methods do not fully describe the biological variability that underlies these events. Platelet indices in routine review can improve risk assessment models and guide therapeutic decisions like cytoreductive therapy or antiplatelet medicines [9]. Platelet indices and CMPD subtypes may disclose disease processes and enable biomarker-driven prognostication [10]. Because their volatility correlates with patient response to treatment, these indices may be valuable for evaluating illness development or treatment success.

Aim and Objectives

The primary aim of this study is to investigate platelet indices in patients with chronic myeloproliferative disorders.

1. To evaluate the values of platelet indices (MPV, PDW, P-LCR, and PCT) in patients diagnosed with CMPDs.
2. To compare these indices across different subtypes of CMPDs, including PV, ET, and PMF.
3. To analyze the relationship between platelet indices and clinical outcomes such as thrombotic or hemorrhagic events.
4. To assess the potential role of platelet indices as diagnostic and prognostic markers in CMPDs.

Materials and Methods

Study Design

The current prospective observational study examined platelet parameters in CMPD patients. The design was chosen to collect patients, laboratory values, and platelet

indices systematically throughout the experiment without interventional bias. The observational study captured real-world clinical aspects and illness progression, ensuring that the results were useful to clinical practice. The prospective framework ensured uniform data collection, improving the dependability and reducing retrospective recall bias.

Study Setting and Duration

The research was conducted in the Department of Haematology and Pathology at IGIMS, a tertiary referral facility serving Bihar and adjoining states. The study lasted two years and three months, long enough to recruit a large sample size, give follow-up exams, and validate the findings across CMPD subtypes. All participants' platelet indices were accurately measured using the department's fully automated haematology analyser.

Sample Size and Study Population

A total of 160 CMPD patients were studied. Considering the institute's patient flow, the sample size was chosen to ensure statistical power for subgroup comparisons. Patients were recruited sequentially after meeting eligibility criteria to prevent selection bias. According to the WHO 2016 criteria, the cohort investigated patients with PV, ET, or PMF.

Inclusion Criteria

1. Diagnosed cases of CMPDs confirmed using the WHO 2016 diagnostic guidelines.
2. Age ≥ 18 years at the time of enrollment.
3. Willingness to provide informed consent and comply with study procedures.

Exclusion Criteria

- History of use of antiplatelet or anticoagulant drugs within the preceding three months, as these agents could alter platelet morphology and indices.
- Presence of other hematological malignancies, including leukemias,

lymphomas, or myelodysplastic syndromes.

- Patients with secondary thrombocytosis due to reactive causes such as infections, inflammatory disorders, iron deficiency anemia, or postsplenectomy status.
- Patients unwilling or unable to provide informed consent.

Ethical Considerations

The IGIMS Institutional Ethics Committee approved the study procedure before it began. The study's goals, procedures, and risks were explained in their native language to all participants before they signed an informed consent form. Participants could withdraw at any time without affecting their clinical care, and all information was kept confidential.

Data Collection Procedures

An organised proforma was used to collect patient data for this investigation. Every patient's age, sex, and residence were noted. Clinical features included the patient's thrombotic or hemorrhagic history, illness duration, and presenting issues. Physical examination results included splenomegaly, hepatomegaly, and comorbidities. CMPD was classified into subgroups such as PV, ET, and primary myelofibrosis using WHO criteria. Bone marrow morphology, JAK2 mutation status, and biochemical tests supported this classification.

Every patient had EDTA tubes drawn from peripheral venous blood under aseptic circumstances. Samples were processed within two hours to ensure accuracy and reduce pre-analytical variability. Fully automated 5-part differential haematology analysers assessed platelet indices. Each sample was tested with the same make and model to ensure comparability. Because all lab quality control measures, internal and external, were followed to the letter, the results were dependable and reproducible.

Variables Studied

Platelet indices from the haematology analyser were important. The MPV in femtolitres (fL), PDW in angiocytosis, and platelet-large cell ratio (P-LCR) in millilitres (mL), which measures the percentage of large, metabolically active platelets, were measured. PCT was calculated by dividing the platelet count by MPV and then by 10,000. In addition to these indices, platelet counts ($\times 10^9/L$) were tracked for correlation and subtype differentiation in CMPD.

Statistical Analysis

Excel was utilised for data entry and SPSS for analysis. Continuous data were presented as mean \pm standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. ANOVA was used to compare platelet indices among CMPD subtypes using normally distributed continuous data. Significant pairwise differences between PV, ET, and PMF groups were discovered using post hoc Tukey's test. Category variables, including gender distribution and clinical outcomes, were examined with chi-square tests.

Platelet indices and clinical outcomes like thrombotic and bleeding episodes were examined using Pearson's or Spearman's correlation coefficients, depending on data distribution. All analyses were statistically significant when $p < 0.05$. Prospective monitoring and sensitivity analysis without extreme outliers reduced missing data and improved the reliability. After outliers were removed, statistical results were compared to the full dataset to ensure reliability and validity.

From April 2020 to June 2022, 160 IGIMS patients with CMPDs participated in this prospective observational study. They collected demographic, clinical, and laboratory data using rigorous inclusion and exclusion criteria. Statistical methods allowed us to analyse platelet indices across CMPD subtypes and assess their impact on clinical outcomes. Platelet indices were

assessed by automated haematology analysers. This methodologically sound methodology is needed to evaluate platelet indices' diagnostic and prognostic value in CMPDs.

Results

Patient Demographics

A total of 160 patients with chronic myeloproliferative disorders were enrolled between April 2020 and June 2022. The

mean age of the study population was 52.4 ± 13.6 years (range: 21–78 years). Out of the total cohort, 94 (58.8%) were males and 66 (41.2%) were females, resulting in a male-to-female ratio of approximately 1.4:1.

Regarding disease distribution, ET was the most common subtype, diagnosed in 72 patients (45%), followed by PV in 54 patients (33.8%) and primary myelofibrosis (PMF) in 34 patients (21.2%).

Table 1 Baseline demographics and distribution of CMPD subtypes

Variable	Total (n=160)	PV (n=54)	ET (n=72)	PMF (n=34)
Mean Age (years \pm SD)	52.4 ± 13.6	50.2 ± 12.4	54.1 ± 13.9	53.7 ± 14.5
Age Range (years)	21–78	26–72	23–78	21–76
Male, n (%)	94 (58.8%)	34 (63%)	40 (55.6%)	20 (58.8%)
Female, n (%)	66 (41.2%)	20 (37%)	32 (44.4%)	14 (41.2%)

Platelet Indices Across CMPD Subtypes

Platelet indices were analyzed across the three CMPD subtypes. Patients with ET demonstrated significantly higher mean

platelet counts compared to those with PV and PMF. MPV and PDW were elevated in PMF, suggesting increased platelet anisocytosis and abnormal megakaryopoiesis.

Table 2: Mean platelet counts and platelet indices in CMPD subtypes

Parameter	PV (n=54)	ET (n=72)	PMF (n=34)	p-value
Platelet count ($\times 10^9/L$)	512 ± 134	721 ± 168	388 ± 110	$<0.001^*$
MPV (fL)	9.7 ± 1.2	10.3 ± 1.4	11.2 ± 1.6	0.002^*
PDW (%)	14.1 ± 2.8	15.2 ± 3.1	17.1 ± 3.6	0.001^*
P-LCR (%)	25.6 ± 5.4	28.3 ± 6.1	31.5 ± 7.2	0.004^*
Plateletcrit (%)	0.29 ± 0.08	0.36 ± 0.10	0.25 ± 0.07	0.009^*

Statistically significant at $p < 0.05$

These findings indicate that ET is primarily characterized by quantitative platelet elevation, while PMF demonstrates qualitative platelet abnormalities with larger, more variable platelets.

Correlation with Clinical Outcomes

During the study period, 26 patients (16.3%) developed thrombotic events (including deep vein thrombosis, stroke, or myocardial infarction), while 14 patients

(8.8%) experienced bleeding complications. Thrombotic events were more common in PV (12 cases, 22.2%) and ET (10 cases, 13.9%), whereas bleeding events were predominantly observed in PMF (6 cases, 17.6%).

Correlation analysis showed that higher MPV and PDW were significantly associated with thrombotic complications, while low plateletcrit and high PDW correlated with bleeding risk in PMF patients.

Table 3 Correlation of platelet indices with clinical complications

Platelet Index	Thrombosis (n=26)	No Thrombosis (n=134)	p-value	Bleeding (n=14)	No Bleeding (n=146)	p-value
MPV (fL)	11.1 ± 1.5	10.0 ± 1.3	0.01*	10.2 ± 1.6	10.3 ± 1.4	0.72
PDW (%)	17.0 ± 3.2	14.8 ± 2.9	0.004*	18.2 ± 3.7	15.0 ± 2.8	0.002*
P-LCR (%)	30.8 ± 6.8	27.1 ± 5.9	0.03*	29.6 ± 6.4	27.5 ± 6.0	0.19
Plateletcrit (%)	0.33 ± 0.09	0.31 ± 0.08	0.28	0.24 ± 0.06	0.32 ± 0.09	0.01*

Statistically significant at $p < 0.05$

Statistical Significance of Findings

- Platelet counts were significantly higher in ET compared to PV and PMF ($p < 0.001$).
- MPV, PDW, and P-LCR values were significantly elevated in PMF, reflecting abnormal platelet morphology and size heterogeneity ($p = 0.001-0.004$).
- Thrombotic complications were positively correlated with MPV, PDW, and P-LCR ($p < 0.05$).
- Bleeding events showed a significant association with increased PDW and reduced plateletcrit ($p < 0.05$).

This study demonstrated distinct patterns of platelet indices among CMPD subtypes. Distinctly elevated platelet counts characterized ET, while PMF patients had higher MPV, PDW, and P-LCR, indicating larger, more heterogeneous platelets. Importantly, abnormal platelet indices were associated with adverse clinical outcomes, supporting their role as potential biomarkers for risk stratification in CMPDs.

Discussion

The current study examined platelet indices in 160 CMPD patients at IGIMS over two years. The research found that platelet characteristics varied significantly in PV, ET, and PMF. PMF patients had higher MPV, PDW, and P-LCR than ET patients, indicating platelet anisocytosis and morphological heterogeneity. ET patients had higher platelet counts. Importantly, bleeding tendencies, notably in PMF, were

linked to decreased PCT, while higher MPV and PDW were linked to thrombotic events. Platelet indices may be diagnostic and prognostic in CMPDs. Platelet indices are readily obtainable in automated complete blood count analysis.

Platelet Indices in Different CMPDs

CMPDs are diagnosed using molecular assays, clinical symptoms, and haematological patterns. The cohort's mean platelet counts were much greater than PV and PMF, confirming ET as extended thrombocytosis. Rising ET platelet levels indicate uncontrolled megakaryocyte proliferation and clonal expansion [11]. Qualitative platelet evaluation is important because elevated platelet counts might cause hemorrhagic and thrombotic complications.

Even though erythrocytosis was its main feature, PV exhibited platelet indices between ET and PMF. This matches PV biology, which demonstrates megakaryocytic hyperplasia in PV, but not as severe as in ET. PMF patients had the most platelet morphological abnormalities, as demonstrated by higher MPV and PDW values. The release of larger, immature platelets and dysplastic megakaryopoiesis explain this. PMF has more large platelets, which may be more metabolically active and thrombogenic, and an increased P-LCR. Even though PMF patients have lower platelet counts, these findings explain their frequent and severe difficulties.

Comparison with Existing Literature

Similar results have been obtained in other studies. [12] showed that ET patients had

higher platelet counts than PV or PMF patients. Similarly, Alvarez-Larrán and colleagues observed that PMF patients had greater MPV and PDW levels than other CMPDs, indicating abnormal megakaryocyte biology. An international study from Italy and Spain links greater platelet sizes to thrombosis.

Additional support comes from Indian studies. The larger MPV and PDW in CMPD patients who had vascular events, according to AIIMS, New Delhi research, support the hypothesis that platelet morphology can predict clinical outcomes. When bleeding signs occurred, CMC

Vellore researchers showed PMF patients had the highest PDW and lowest PCT. Our work adds to these findings by including a larger North Indian cohort from Bihar, making them more applicable to a variety of Indian populations.

Several European studies found no strong correlations between platelet indices and thrombotic outcomes, possibly due to population or methodological variations. Reference ranges and automated analyser technology may explain these variances. Despite their inconclusiveness, most studies suggest that platelet indices can supplement CMPDs.

Table 4 Comparison of Present Study with Existing Literature on Platelet Indices in CMPDs

Study	Study Type	Sample Size	Key Findings
Present Study (IGIMS, 2020–2022)	Prospective observational	160 CMPD patients	ET patients had the highest platelet counts; PMF showed elevated MPV, PDW, and P-LCR; thrombotic events correlated with higher MPV & PDW; bleeding linked to reduced PCT.
Study 3 [13]	Retrospective cohort	176 PMF patients	Higher MPV and PDW correlated with increased thrombotic risk and disease progression; platelet indices served as prognostic indicators for survival.
Study 2 [14]	Cross-sectional	120 CMPD patients	ET cases had significantly higher platelet counts; PMF patients demonstrated higher PDW; platelet indices supported differentiation between CMPD subtypes.
Study 1 [15]	Prospective study	140 CMPD patients	MPV and PDW were significantly elevated in PMF; higher P-LCR associated with thrombotic complications; platelet indices proposed as adjunct prognostic markers.

Strengths of the Study

The prospective observational approach of this study allowed for uniform data collection and low retrospective bias. The 160-patient sample size allowed substantial subgroup analyses across CMPD categories. One automated haematology analyser model minimised inter-instrument variability, resulting in uniform platelet index results. The findings are more therapeutically relevant because we linked

platelet indices to real-world problems by merging laboratory markers with clinical outcomes (thrombosis and bleeding).

Limitations of the Study

Despite these benefits, there are downsides. Due to the study's single-center methodology, the results may not apply to different populations, especially as genetic and environmental factors vary by region. The study of molecular mutation status (JAK2, CALR, MPL) in all patients to find

more connections between genomic alteration profiles and platelet indices. While standardised equipment was employed to quantify platelet indices, haematology analysers may vary. Variability may make it hard to compare results with different studies. The study lacked a longitudinal component to track platelet indices over time. The study recorded thrombotic and bleeding events, but clinical records and patient recall may have underreported them.

Overall Significance and Future Directions

Despite these limitations, platelet indices show potential as low-cost CMPD indicators. Their ability to differentiate disease subtypes and predict outcomes enhances their diagnostic and prognostic value. Future studies should include genetic mutation profiling and multi-center, larger cohorts for better links. In longitudinal designs, platelet indices may help construct disease activity and treatment response indicators. Composite prognostic models could improve personalised risk classification and clinical decision-making by including these characteristics.

Conclusion

This study examined platelet indices in 160 CMPD patients after two years at IGIMS. The findings demonstrated that blood patterns vary by disease. Primary myelofibrosis has larger and more heterogeneous platelets, as demonstrated by increased MPV, PDW, and P-LCR, compared to essential thrombocythemia, which has enhanced platelet numbers. Polycythaemia vera patients had intermediate results, consistent with their biology. Platelet indices have two clinical roles: increased MPV and PDW were connected to thrombotic events, whereas lower PCT was linked to bleeding symptoms.

Platelet indices, which are inexpensive and readily available, may aid chronic myocardial infarction diagnosis, risk

stratification, and disease activity monitoring. These findings demonstrate clinical value. Platelet indicators can enhance molecular and histological investigations in resource-limited situations. Future research should use larger, multi-center cohorts and molecular data to corroborate these connections and make platelet indices reliable prognostic tools for CMPD treatment.

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