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Immature Platelet Fraction as a Rapid Discriminator of Peripheral and Central Thrombocytopenia: A Cross-Sectional Observational Study

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ABSTRACT

Background: Immature platelet fraction (IPF) quantifies newly released, RNA-rich platelets and may provide a rapid indication of marrow thrombopoietic activity. This study evaluated whether IPF measured on an automated haematology analyser could help distinguish peripheral platelet destruction or consumption from central hypoproduction in patients with thrombocytopenia.
Methods: This cross-sectional descriptive observational study included 74 thrombocytopenic inpatients evaluated at the Central Diagnostic Laboratory, Department of Pathology, at a tertiary care centre in Rajasthan, from 1 December 2023 to 1 January 2024. EDTA-anticoagulated blood was analysed on a Sysmex XN-1000 for complete haemogram and IPF. Patients were assigned to peripheral or central aetiological groups from the final clinical diagnosis. The analyser reference interval used for IPF was 1.2%-8.6%.
Results: The cohort comprised 45 males (60.81%) and 29 females (39.19%); median age was 30 years. Fifty-five patients (74.32%) had peripheral and 19 (25.68%) had central thrombocytopenia. IPF exceeded 8.6% in 42/55 peripheral cases (76.36%) but only 1/19 central cases (5.26%). Using IPF >8.6% as a descriptive discriminator for peripheral thrombocytopenia yielded sensitivity 76.36%, specificity 94.74%, positive predictive value 97.67%, negative predictive value 58.06%, and accuracy 81.08%. Median IPF was highest in immune thrombocytopenia (27.5%) and lowest in aplastic anaemia (1.9%).
Conclusion: A raised IPF strongly favoured a peripheral destructive or consumptive mechanism and may serve as a rapid adjunct to the clinical evaluation of thrombocytopenia. A normal or low IPF did not reliably exclude a peripheral cause; consequently, IPF should complement, not replace, clinical assessment, smear review, and bone marrow examination when otherwise indicated.

Keywords*: immature platelet fraction; thrombocytopenia; thrombopoiesis; immune thrombocytopenia; bone marrow failure; Sysmex XN-1000.*

INTRODUCTION

Thrombocytopenia, generally defined as a platelet count below 150 × 10^9/L, is a common laboratory finding with causes that can be broadly classified as reduced platelet production, increased destruction or consumption, and sequestration. Determining the dominant mechanism is clinically important because investigation and treatment differ substantially among these categories.[1,2]

Bone marrow examination can directly assess megakaryopoiesis but is invasive, resource intensive, and not required in every patient. Routinely reported platelet indices such as mean platelet volume and platelet distribution width may support the differential diagnosis, although pre-analytical platelet swelling, analyser dependence, and incomplete standardisation limit their reliability.[3,4]

Immature platelets are newly released, RNA-rich platelets. Automated fluorescence analysers quantify them as the immature platelet fraction (IPF), which acts as an indirect measure of thrombopoietic activity. IPF typically increases when marrow production responds to peripheral platelet destruction or consumption and remains low when thrombopoiesis is impaired, although overlap occurs among diseases and across analytical platforms.[5-7]

Studies using automated IPF measurement have demonstrated higher values in consumptive disorders and immune thrombocytopenia than in hypoproductive states. However, local data across mixed adult inpatient aetiologies remain limited, and reference intervals and thresholds cannot be transferred uncritically between analysers or populations.[8-11]

The present study therefore evaluated the distribution and potential clinical utility of IPF among thrombocytopenic patients at a tertiary-care centre, with particular emphasis on differentiating peripheral from central causes.

MATERIALS AND METHODS

Study design and setting: A cross-sectional descriptive observational study was conducted in the Central Laboratory, Department of Pathology, Government Medical College, Kota, Rajasthan, India, over one month from 1 December 2023 to 1 January 2024.

Participants: Seventy-four inpatients presenting to different clinical departments with thrombocytopenia, defined as a platelet count <150 × 10⁹/L, were included irrespective of sex. Patients without thrombocytopenia were excluded. The final clinical diagnosis was used to group cases as peripheral destruction/consumption or central reduced thrombopoiesis.

Laboratory assessment: Venous blood collected in EDTA was analysed on the Sysmex XN-1000 automated haematology analyser. Complete haemogram and IPF were recorded. IPF values were interpreted using the laboratory/analyser reference interval of 1.2%-8.6% and categorised as low (<1.2%), within interval (1.2%-8.6%), or high (>8.6%).

Statistical analysis: The presentation dataset supplied aggregate counts, means with standard deviations, and medians with ranges. Categorical variables are reported as number and percentage; IPF is summarised as mean ± standard deviation and median (range). From the 2 × 2 aggregate distribution, sensitivity, specificity, predictive values, and accuracy of IPF >8.6% for identifying a peripheral mechanism were calculated descriptively. No hypothesis-test p values were generated because patient-level data were not available for independent reanalysis.

Ethical Consideration: Approval taken from departmental research committee, department of Pathology, Government Medical College, Kota.

RESULTS

Of 74 patients, 45 (60.81%) were male and 29 (39.19%) female; the median age was 30 years. A peripheral mechanism was assigned in 55 (74.32%) patients and a central mechanism in 19 (25.68%). Viral infection (29.72% of the whole cohort) and sepsis (20.27%) were the most frequent peripheral diagnoses. The central group included acute leukaemias, chronic myeloid leukaemia, aplastic anaemia, myelodysplastic neoplasm, and multiple myeloma.

Table 1. Distribution of IPF by final diagnosis (n=74)

Diagnosis	n	Mean ± SD (%)	Median (range) (%)	<1.2 %	1.2%-8.6%	>8.6%
Viral infection	22	11.32 ± 5.72	9.1 (5.2-28.5)	0	10	12
Sepsis	15	18.76 ± 5.85	20.0 (9.1-25.2)	0	0	15
Immune thrombocytopenia	9	26.43 ± 10.19	27.5 (7.5-40.4)	0	1	8
Consumptive coagulopathy	7	21.91 ± 12.81	22.6 (7.4-39.4)	0	2	5
Vaso-occlusive crisis	2	14.90 ± 0.42	14.9 (14.6-15.2)	0	0	2
Aplastic anaemia	3	2.43 ± 1.86	1.9 (0.9-4.5)	1	2	0
Chronic myeloid leukaemia	4	4.05 ± 2.13	3.25 (2.5-7.2)	0	4	0
Acute lymphoblastic leukaemia	4	2.33 ± 1.28	2.25 (0.9-3.9)	1	3	0

Acute myeloid leukaemia	4	4.83 ± 3.41	4.55 (1.1-9.1)	1	2	1
Multiple myeloma	2	8.35 ± 0.35	8.35 (8.1-8.6)	0	2	0
Myelodysplastic neoplasm	2	2.95 ± 2.76	2.95 (1.0-4.9)	1	1	0
Total	74	-	0.9-40.4	4	27	43

IPF ranged from 0.9% to 40.4%. It was highest in immune thrombocytopenia (median 27.5%) and lowest in aplastic anaemia (median 1.9%). All patients with sepsis and vaso-occlusive crisis had IPF >8.6%; one patient with acute myeloid leukaemia also had a raised IPF.

Table 2. IPF category according to mechanism of thrombocytopenia

Mechanism	n	IPF ≤8.6%, n (%)	IPF >8.6%, n (%)
Peripheral destruction/consumption	55	13 (23.64)	42 (76.36)
Central hypoproduction	19	18 (94.74)	1 (5.26)
Total	74	31 (41.89)	43 (58.11)

When IPF >8.6% was treated as a positive test for a peripheral mechanism, sensitivity was 76.36% (42/55), specificity 94.74% (18/19), positive predictive value 97.67% (42/43), negative predictive value 58.06% (18/31), and overall accuracy 81.08% (60/74). These estimates are descriptive and are not externally validated cut-offs.

DISCUSSION

The principal finding was the marked separation in the frequency of raised IPF between peripheral and central thrombocytopenia. More than three-quarters of peripheral cases had IPF >8.6%, compared with only one central case. The high specificity and positive predictive value suggest that, in this cohort, a raised IPF strongly supported active marrow compensation for peripheral platelet loss. Conversely, the modest negative predictive value means that an IPF within or below the reference interval cannot safely exclude a peripheral mechanism.

These findings are biologically plausible. RNA-rich immature platelets enter the circulation during accelerated thrombopoiesis and their proportion rises when marrow output compensates for shortened platelet survival. The IPF can therefore provide information that the platelet count alone cannot: the count reflects the net balance between production and clearance, whereas IPF more directly reflects recent platelet release.[5-7]

Ali et al. studied 637 thrombocytopenic patients and found higher IPF in increased-consumption disorders than in reduced thrombopoiesis, with particularly high values in immune thrombocytopenia. The present study showed the same pattern: immune thrombocytopenia had the highest median IPF (27.5%), while aplastic anaemia had the lowest median (1.9%). Other diagnostic studies have similarly reported utility for IPF-related parameters in distinguishing hyperdestructive from hypoproductive thrombocytopenia and in supporting an immune thrombocytopenia diagnosis.[8-11]

Infection-related groups also had high IPF: all sepsis cases and more than half of viral-infection cases exceeded 8.6%. Sepsis can combine consumption, immune activation, endothelial injury, and altered thrombopoiesis; serial studies have shown dynamic changes in immature platelet indices during critical illness. Thus, a raised IPF in infection should be interpreted as evidence of increased platelet turnover rather than as proof of a single aetiology.[12]

The single raised IPF result in acute myeloid leukaemia illustrates clinically important overlap. Central disorders may show residual or recovering megakaryopoiesis, concurrent infection or consumption, treatment-related temporal effects, or heterogeneous marrow involvement. Conversely, peripheral disorders may have a normal IPF early in their course or when compensatory production is inadequate. IPF should therefore be integrated with clinical context, medication and transfusion history, peripheral smear findings, other cell lines, and temporal platelet trends.

Analytical limitations are also important. IPF values are method dependent and may be influenced by platelet size, sample handling, time to analysis, and instrument-specific fluorescence gates. Published reviews recommend platform-specific reference intervals and caution against universal diagnostic thresholds.[6,7]

Clinically, automated IPF has several advantages: it is obtained from the same EDTA specimen as the complete blood count, requires little additional turnaround time, and may help prioritise subsequent investigations. A markedly raised IPF in an otherwise compatible presentation may support a peripheral process and reduce unnecessary immediate marrow examination. It should not, however, be presented as a replacement for bone marrow examination when there are blasts, pancytopenia, unexplained cytopenias, suspected marrow infiltration, or persistent diagnostic uncertainty.

LIMITATIONS

This was a small, single-centre study conducted over one month. Diagnostic groups were heterogeneous, and some categories contained only two to four patients. No healthy control group was taken. The threshold analysis was based on the analyser interval rather than an internally derived ROC cut-off, and confidence intervals or adjusted analyses could not be calculated from the aggregate data. These limitations restrict generalisability and preclude causal inference. Multi-centric studies with larger sample size with defined cohorts and control arm are recommended.

CONCLUSION

IPF measured on the Sysmex XN-1000 was substantially more often raised in peripheral destructive or consumptive thrombocytopenia than in central hypoproduction. IPF >8.6% had high specificity and positive predictive value for a peripheral mechanism in this cohort, while a non-raised value had limited exclusionary value. IPF is best used as a rapid, inexpensive adjunct within a structured diagnostic assessment. Larger prospective studies using explicit diagnostic adjudication, locally validated reference intervals, serial measurements, and patient-level statistical analysis are required before a universal decision threshold can be recommended.

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REFERENCES

1. Stasi R. How to approach thrombocytopenia. Hematology Am Soc Hematol Educ Program. 2012;2012:191-197. doi:10.1182/asheducation-2012.1.191.
2. Kaushansky K. Thrombopoiesis. Semin Hematol. 2015;52(1):4-11. doi:10.1053/j.seminhematol.2014.10.003.
3. Numbenjapon T, Mahapo N, Pornvipavee R, Sriswasdi C, Mongkonsritragoon W, Leelasiri A, et al. A prospective evaluation of normal mean platelet volume in discriminating hyperdestructive thrombocytopenia from hypoproductive thrombocytopenia. Int J Lab Hematol. 2008;30(5):408-414. doi:10.1111/j.1751-553X.2007.00969.x.
4. Noris P, Melazzini F, Balduini CL. New roles for mean platelet volume measurement in the clinical practice? Platelets. 2016;27(7):607-612. doi:10.1080/09537104.2016.1224828.
5. Hoffmann JJML. Reticulated platelets: analytical aspects and clinical utility. Clin Chem Lab Med. 2014;52(8):1107-1117. doi:10.1515/cclm-2014-0165.
6. Buttarello M, Mezzapelle G, Freguglia F, Plebani M. Reticulated platelets and immature platelet fraction: clinical applications and method limitations. Int J Lab Hematol. 2020;42(4):363-370. doi:10.1111/ijlh.13177.
7. Bodrova VV, Shustova ON, Khaspekova SG, Mazurov AV. Laboratory markers of platelet production and turnover. Biochemistry (Mosc). 2023;88(Suppl 1):S39-S51. doi:10.1134/S0006297923140031.
8. Ali I, Graham C, Dempsey-Hibbert NC. Immature platelet fraction as a useful marker in the etiological determination of thrombocytopenia. Exp Hematol. 2019;78:56-61. doi:10.1016/j.exphem.2019.09.001.
9. Jeon K, Kim M, Lee J, Lee JS, Kim HS, Kang HJ, et al. Immature platelet fraction related parameters in the differential diagnosis of thrombocytopenia. Platelets. 2020;31(6):795-801. doi:10.1080/09537104.2019.1678113.
10. Adly AAM, Ragab IA, Ismail EAR, Farahat MM. Evaluation of the immature platelet fraction in the diagnosis and prognosis of childhood immune thrombocytopenia. Platelets. 2015;26(7):645-650. doi:10.3109/09537104.2014.969220.
11. Ferreira FLB, Colella MP, Medina SS, Costa-Lima C, Fiusa MML, Costa LNG, et al. Evaluation of the immature platelet fraction contribute to the differential diagnosis of hereditary, immune and other acquired thrombocytopenias. Sci Rep. 2017;7:3355. doi:10.1038/s41598-017-03668-y.
12. Koyama K, Katayama S, Muronoi T, Tonai K, Goto Y, Koinuma T, et al. Time course of immature platelet count and its relation to thrombocytopenia and mortality in patients with sepsis. PLoS One. 2018;13(1):e0192064. doi:10.1371/journal.pone.0192064.