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Original article

IPF Marker as A Differentiating Agent of Adult Thrombocytopenia Patients by Using Mindray Auto-Hematology Analyzer (1st attempt in Libya)

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Abstract
Platelets, crucial for hemostasis and thrombosis, are assessed in clinical practice using the immature platelet fraction (IPF), a marker reflecting bone marrow platelet production. IPF measurement, especially using automated hematology analyzers like Mindray BC-780[R], offers a non-invasive alternative for thrombocytopenia diagnosis. However, its utility in evaluating different thrombocytopenic conditions remains underexplored, particularly in Libya. The main aim is to evaluate the fraction of immature platelets in thrombocytopenia patients as compared with healthy individuals and the absolute immature count of platelets (AIPC). This descriptive, cross-sectional study included 228 participants (106 thrombocytopenic patients and 122 healthy controls) at Al-Wekaya Laboratory Center, Sabratha, Libya. Platelet counts and IPF were measured using the Mindray BC-780[R] analyzer. Statistical analyses were performed using SPSS v24 to evaluate differences in platelet parameters and IPF across various thrombocytopenia etiologies. Thrombocytopenic patients exhibited significantly lower platelet counts ($70.5 \times 10^3/\mu L$) and higher IPF (12.8%) compared to controls ($258.1 \times 10^3/\mu L$; IPF 4.23%; $p < 0.001$). Elevated IPF values (>10%) were prominent in hemolytic anemia (26.97%), acute myeloid leukemia (21.5%), and iron deficiency anemia (19.8%), indicating heightened bone marrow compensation. Pearson correlation revealed a significant inverse relationship between platelet count and IPF ($r = -0.246$, $p = 0.011$), highlighting compensatory platelet production in thrombocytopenic conditions. IPF measurement via Mindray BC-780[R] proves valuable for differentiating thrombocytopenia etiologies. Elevated IPF reflects active marrow compensation in conditions with heightened platelet turnover. This study, the first of its kind in Libya, emphasizes the clinical utility of automated IPF assessment in hematology. Further research is recommended to validate these findings across diverse populations and clinical settings.
Keywords. Platelets, Thrombocytopenia, Immature Platelets Fraction (IPF), Mindray BC-780[R].

Introduction

Platelets, which naturally play important roles in hemostasis, thrombosis, and inflammation, are nucleated cell fragments generated from megakaryocytes (MK) in the bone marrow (BM) and range in number from 150,000 to 400,000 cells/µL [1]. Thrombocytopenia, a reduction in the quantity of thrombocytes in peripheral blood, is a common hematological abnormality linked to serious bleeding consequences. Reduced thrombopoiesis and accelerated platelet breakdown in peripheral circulation are the two main categories of thrombocytopenia. The gold standard for determining the cause of thrombocytopenia is still bone marrow puncture (BMP), which is typically done to detect thrombopoiesis activity. However, the patient may experience discomfort from this very intrusive diagnostic technique. Therefore, assessing the immature platelet fraction (IPF) is one non-invasive method to help identify the cause of thrombocytopenia. The value of freshly released platelets in peripheral blood, known as reticulated platelets, is reflected in IPF. Their increased size and RNA content allow them to be differentiated from mature platelets [2]. Clinically, there are three primary categories of causes for thrombocytopenia: (i) decreased thrombopoiesis; (ii) increased platelet consumption or destruction; and (iii) aberrant platelet distribution (sequestration) [3, 4]. The quantity of megakaryocytes is connected with the number of reticulated platelets in the bone marrow, which are typically two to three times as many as those in the peripheral circulation. However, under typical circumstances, "mature" platelets remain in the bloodstream for seven to ten days [5].

As a result, the quantity of reticulated platelets (RPs) increases with rising platelet production and decreases with falling production, reflecting the rate of thrombopoiesis. One of the best prognostic markers for assessing thrombocytopenic patients is the measurement of reticulated platelets using an automated hematology analyzer in conjunction with blood counts. The percentage of RPs, or IPF, indicates how severely platelets have been damaged and how many platelets are being produced in bone marrow [6, 7]. When added to complete blood count (CBC), which reflects a population of freshly generated platelets with a higher proportion of residual RNA, the IPF marker has recently emerged as a crucial diagnostic tool for thrombocytopenia [8]. The IPF was first identified as reticulated platelets and evaluated by flow cytometry [9]. Recent research has documented the

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clinical value of employing automated hematology analyzers to measure immature platelets in clinical settings [10-12].
In this perspective, it is worth mentioning, that less is known about the IPF's use in the differential diagnosis of several conditions, including hematologic malignancies, bone marrow failure (BMF), hypersplenism, immune thrombocytopenia (ITP), microangiopathic hemolytic anemias, and hereditary macrothrombocytopenia (HM), even though its usefulness in the differential diagnosis between hypo- and hyperproliferative thrombocytopenia's has already been documented [13, 14]. More recently, Miyazaki et al. [15] found that HM patients had significantly higher IPF values than ITP in research including 15 HM patients. The IPF was assessed using an automated hematological analyzer. Fluorescent dyes that can bind to RNA and flow cytometers can be used to quantify RP, but their lack of consistency and fluctuating reference intervals restrict their clinical utility [16]. Conversely, automated hematology analyzers can estimate RP in peripheral blood by expressing it as IPF.
At the moment, automated IPF assessment is accessible and dependable in routine clinical practice [17, 18]. Using a patented asymmetric cyanine-based dye for nucleic acid substances staining, the Mindray auto-hematology analyzer may produce one of the most accurate IPF measurement results. The forward scatter vs. sideward fluorescence scatterplot is used to calculate IPF, and the findings are displayed as a percentage [19]. The PLT-O scattergram is used to derive the parameter for BC-760[R]/BC-780[R], whereas the PLT-H scattergram is used to derive the optional parameter for BC-760[B] [20]. During this study (as a first study about this subject in Libya using the Mindray auto-hematology analyser), the main aim is to evaluate the fraction of immature platelets in thrombocytopenia patients as compared with healthy individuals and the absolute immature count of platelets (AIPC). Different cases of thrombocytopenia patients and healthy individuals as control subjects will be included in this study.

Methods

Study design

A descriptive, cross-sectional study design was employed in this investigation. It was performed at Al-Wekaya laboratory center (Sabratha, Libya). Before gathering information about the thrombocytopenic patients and blood samples collection, special ethical permission was obtained from the Sabratha Oncology Institute's ethical council. Blood samples for CBC were analyzed at Al-Wekaya Laboratory Center.

Thrombocytopenic patients and the control group

In the present study, 228 participants were split into 106 thrombocytopenic patients 48(45.28%) males, 58(54.7%) females) and 122 healthy people (control group), consisting of 32(26.2%) males and 90(73.7%) females. Patients who had a platelet transfusion within the last 30 days were not included. Without regard to age, both male and female patients, as well as healthy persons, were admitted to the study. At the Sabratha Teaching Hospital (STH) and Sabratha Oncology Institute (SOI), thrombocytopenia was confirmed in two separate samples and by microscopic examination during routine clinical follow-up. All thrombocytopenic individuals had to have a confirmed diagnosis in order to be included. Worth mentioning, that all patients who participated in the current study with a platelet count below 150×10^9/L have been considered as thrombocytopenic patients.

Sample collection

Blood samples were drawn and placed in Becton Dickinson 4-mL (K2) EDTA vacutainers. Prior to testing, all samples were stored at ambient temperature (18°C to 25°C). After venipuncture, all samples were processed in four hours. The Mindray BC-780[R] was used to analyze all blood samples for IPF and all other related hematological parameters.

Measurement of IPF using Mindray Hematology Analyzer

The BC-780 (R) hematology analyzer from Mindray uses (Focusing Flow-DC Impedance Method) [20]. a semiconductor diode laser to perform fluorescent flow cytometry. Because IPF contains a significant amount of RNA that exhibits comparatively high FL (Fluorescent Light, RNA) and FS (Forward Scatter, size) signals, it studies IPF in the reticulocyte channel where an asymmetric cyanine dye measures RNA. In the optical platelet (PLT-O) measurement, IPF is gated inside a specific region of the platelet scatter plot that displays higher fluorescent signals. IPF can be stated as a percentage (IPF%) or as an absolute value (IPF#).

Statistical analysis

Statistical analyses were performed using SPSS Statistics version 24 (IBM Corporation, New York, NY). Quantitative variables were presented as the mean and standard deviation or median and interquartile range

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(IQR: 25th–75th percentiles). Student t-test was applied to compare parametric quantitative variables between two groups. Any other statistical measurements used for data analysis were mentioned in the context. IPF normal range of 0.7 – 5.7 % was used as the minimum and maximum limits of the fraction.

Results

Out of 122 individuals in the control group, 32 (26.2%) were male, and 90 (73.7%) were female. This indicates a higher proportion of females in the control group compared to males. Conversely, among the 106 thrombocytopenic patients, 48 (45.28%) were male, and 58 (54.7%) were female. While females remain the majority, the gender distribution among patients is more balanced compared to the control group (Table 1). The result of the Chi-Square test for independence indicated a statistically significant difference in gender distribution between the control group and the patient group, with a p-value (0.004).

Table 1. Gender distribution among the control group and thrombocytopenic patients.

Gender	Control group	%	Patients	%
Male	32	26.2	48	45.28
Female	90	73.7	58	54.7

Table 2 summarizes the frequency and percentage of different diagnoses among the participants in the study. It provides insights into the distribution of various medical conditions, highlighting the diversity of underlying diseases within the study population. In detail, Myelodysplastic syndrome (MDS), viral infections, idiopathic thrombocytopenic purpura (ITP), and unknown diagnoses each made up 7.27% of the individuals. These conditions are relatively common among the research population, as indicated by the fact that they form the largest single category. While Iron deficiency anemia (IDA) and Breast cancer each comprised 5.45% of cases, Colon cancer was slightly less common, accounting for 6.36%. on the other hand, diseases like Acute myeloid leukemia (AML)**,** Pregnancy-related thrombocytopenia**,** Vit. B12 deficiency and Chronic lymphocytic leukemia (CLL) each accounted for 3.64% of participants, which were fewer common diagnoses. Conditions like Von-Willebrand disease (VWD)**,** drug history**,** autoimmune diseases, and certain cancers (e.g., Prostatic cancer**,** Gallbladder cancer) were very infrequent, each comprising less than 1% of cases.

Table 2. Frequency and percent of different diseases among participants.

Diagnosis	Frequency	Percent %
Von Willebrand disease (VWD)	1	0.91
Myelodysplastic syndrome (MDS)	8	7.27
Hodgkin's and Non-Hodgkin's lymphoma (HL, NHL)	3	2.73
Viral infection	8	7.27
Hemolytic anemia (HA)	4	3.64
Idiopathic thrombocytopenic purpura (ITP)	8	7.27
Thyroidectomy and thyroid cancer	3	2.73
Acute lymphocytic leukemia (ALL)	2	1.82
Colon cancer	7	6.36
Disseminated intravascular coagulation (DIC)	3	2.72
Lung cancer	2	1.82
Drug history	1	0.91
Acute myeloid leukemia (AML)	5	4.55
Vit. B12 deficiency	4	3.64
Iron deficiency anemia (IDA)	6	5.45
Breast cancer	6	5.45
Pregnancy	5	4.55
Uterus cancer	1	0.91
Prostatic cancer	2	1.82
Liver disease	2	1.82
Chronic diseases	4	3.64
Chronic myeloid leukemia (CML)	1	0.92
Renal failure (RF)	2	1.82
Gallbladder cancer	1	0.91
Chronic lymphocytic leukemia (CLL)	4	3.64
Auto immune diseases	1	0.91

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Chronic kidney disease	1	0.91
Pancytopenia	2	1.82
Gastritis	2	1.82
Unknown diagnosis	8	7.27
Multiple myeloma	3	2.73

The platelet count (PLT) and IPF of the patients and the control group are contrasted in Table 3. In comparison to the patient group (70.5 × $10^3/\mu L$), the control group's mean platelet count (258.1 × $10^3/\mu L$) is noticeably greater. One of the main characteristics of the patients' clinical state, thrombocytopenia, is indicated by the significant decrease in PLT. Patients have a considerably greater mean IPF (12.8%) than controls (4.23%). Patients with elevated IPF are likely producing more platelets as a compensatory mechanism for their thrombocytopenia. With p-values < 0.001, both differences are statistically significant, suggesting that the observed variations are not likely to be the result of chance.

Table 3. Platelet count and IPF in patients and controls.

Category	PLT count (×10³/µL)				IPF (%)			
	Mean	**± SD**	**Median**	**SD-Error**	**Mean**	**SD**	**Median**	**SD- Error**
Control group	258.1	± 56.6	248	5.14	4.23	2.78	3.6	0.25
Patients	70.5	± 40.3	76	3.92	12.8	10.78	10.2	1.04
P-value	**< 0.001**				**< 0.001**			

Both platelet count and IPF differ significantly between the control and patient groups, with p-values far below the conventional significance level (p<0.05).

The IPF values for each patient's various illnesses are compiled in Table 4. The mean IPF varies by diagnosis; greater IPF levels, which indicate active platelet generation, are found in illnesses such as hemolytic anemia (26.97%), AML (21.5%), and IDA (19.8%). Interestingly, none of the patients had IPF less than 1%, indicating that there is a high platelet turnover in every case. In the majority of cases, particularly in disorders like NHL/HL (100%), IDA (83.3%), and AML (80%), there was significant compensatory production, as (40.9%) of patients had IPF in the intermediate range (1–10%) and (59.1%) had IPF >10%.

Table 4. Patients' diagnosis and IPF value

Diagnosis	Mean ± SD	IPF <1% N (%)	IPF 1-10% N (%)	IPF>10% N (%)
MDS	9.4±6.23	0 (0)	5 (62.5)	3 (37.5)
CLL	9.22±3.42	0 (0)	2 (50)	2 (50)
Breast cancer	12.01±14.7	0 (0)	4 (66.6)	2 (33.3)
Hemolytic anemia	26.97±18.79	0 (0)	1 (25)	3 (75)
AML	21.5±20.83	0 (0)	1 (20)	4 (80)
ITP	11.96±4.58	0 (0)	2 (25)	6 (75)
Multiple myeloma	3.96±2.32	0 (0)	3 (100)	0 (0)
Colon cancer	12.18±6.95	0 (0)	3 (42.8)	4 (57.1)
IDA	19.8±11.26	0 (0)	1 (16.6)	5 (83.3)
NHL, HL	16.2±8.15	0 (0)	0 (0)	3 (100)
Total number (%)		0 (0)	22(40.9)	32 (59.12)

There is a weak negative association between PLT and IPF%, as indicated by the Pearson correlation coefficient of -0.246 (Table 5). This indicates that IPF% tends to modestly increase when platelet counts decline. This correlation's p-value, which is less than the significance level of 0.05, is 0.011. Accordingly, it is doubtful that the statistically significant negative association between PLT and IPF% is the result of coincidence. A greater IPF% is linked to a decreased platelet count, indicating that the bone marrow makes up for this by making more immature platelets in thrombocytopenic patients, which is reflected in the elevated IPF.

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Table 5. The Pearson correlation coefficient between PLT and IPF%.

Correlations		PLT	IPF%
PLT	Pearson Correlation	1	-.246-*
	Sig. (2-tailed)		.011*
	N	106	106
IPF%	Pearson Correlation	-.246-*	1
	Sig. (2-tailed)	.011*	
	N	106	106
***. Correlation is significant at the 0.05 level (2-tailed).**			

Platelet count and IPF% have an inverse association, according to the scatter plot (Figure 1). IPF% rises when platelet counts fall, which may indicate that thrombocytopenia causes the bone marrow to produce more immature platelets in response. The data points are grouped based on various diagnoses; even at low platelet counts, certain illnesses (such as hemolytic anemia and iron deficiency anemia) exhibit a more noticeable increase in IPF%. Regardless of platelet count, other diseases, including MDS or CLL, could show less variation in IPF%.

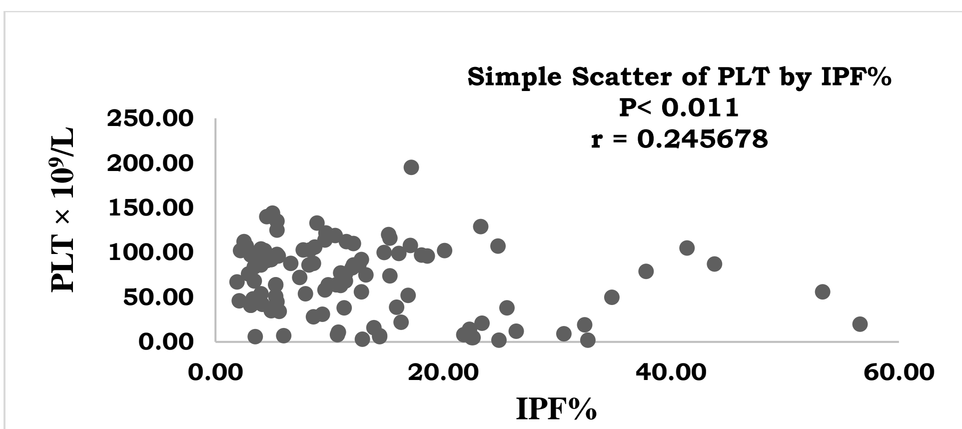

Figure 1. Scatter plots showing correlation between immature platelet fraction (IPF%) and platelet counts (X 10^9/L) of patients with different causes of thrombocytopenia.

Discussion

As bone marrow generates more platelets, IPF levels increase. As a result, measuring IPF represents bone marrow platelet synthesis from a peripheral blood sample, just like a reticulocyte count may indicate the creation of red blood cells. A contemporary metric for assessing youthful, more reactive platelets in peripheral blood is the immature platelet fraction. This study demonstrates that there is a considerable gender difference between the control group and thrombocytopenic patients, with a prevalence of females in both categories. However, thrombocytopenic patients are more likely to be male (45.28%) than the control group (26.2%), according to the Chi-square test (p = 0.004). Even while thrombocytopenia affects both sexes, this data suggests that the higher percentage of male patients may be caused by underlying biological or sociocultural factors that change how thrombocytopenic illnesses are diagnosed, reported, or prevalent. As a clinical syndrome, thrombocytopenia is diverse, as seen by the range of diagnoses seen among thrombocytopenic individuals. Together, MDS, viral infections, ITP, and uncertain diagnoses account for a significant percentage of patients (28.54%), highlighting the variety of etiologies that physicians deal with daily. It's interesting to note that diseases like IDA and breast cancer, which account for 5.45% of cases each, emphasize the part that systemic diseases and cancers play in the etiology of thrombocytopenia. Less prevalent disorders such as AML, vitamin B12 insufficiency, pregnancy-related thrombocytopenia, and CLL (each accounting for 3.64%) indicate that when dealing with thrombocytopenic individuals, a thorough diagnostic approach is necessary. Despite being less common, these disorders can have serious clinical repercussions and frequently require specific therapeutic techniques.

In this context, several studies found that patients with ITP had significantly more reticulated platelets (median IPF 7.7–25.2%, median ret-PLT 9.1%) when using automated hematolysis analyzers [21-28] and custom-made flow cytometry methods [29, 30]. Nonetheless, a number of studies have also reported elevated reticulated platelet counts in MDS and aplastic anemia (AA) patients [24, 31-34]. Notably, results of this study demonstrated that less than 1% of instances were related to specific illnesses, such as VWD, autoimmune disorders, and uncommon cancers (such as prostatic and gallbladder cancer). Even though they are uncommon,

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these disorders emphasize how crucial it is to take uncommon causes into account, especially when thrombocytopenia presents atypically or refractorily. The inclusion of "unknown diagnoses" (7.27%) further highlights the difficulties in diagnosing this patient population and the possibility of underdiagnosed or newly discovered illnesses. The findings of this study underscore the importance of demographic and diagnostic characterization in understanding the epidemiology and etiology of thrombocytopenia. The gender imbalance in the control group compared to the more balanced distribution among patients may indicate gender-specific susceptibility or healthcare-seeking behavior. Further research is warranted to explore the underlying factors contributing to these patterns and to evaluate the role of specific conditions, including hematological malignancies and systemic diseases, in the development of thrombocytopenia.

Furthermore, as anticipated and consistent with previous research [35, 36], examination of the IPF% in all thrombocytopenic patients (n=106) and normal samples (n=122) showed a significantly higher IPF% in thrombocytopenia than in the control group. This was also true when comparing cohorts within a particular gender. This supported more research on the parameter to determine its applicability as a marker of particular underlying thrombocytopenia causes. The results of this study demonstrate the usefulness of the IPF as a marker for assessing compensatory generation and platelet turnover across a range of hematologic and non-hematologic illnesses. High platelet turnover is always confirmed by elevated IPF values in all patients, confirming its use as a sensitive measure of thrombopoietic activity. Additionally, because the kinetics of platelet synthesis and consumption fluctuate depending on the underlying illness, the mean IPF values varied dramatically. The greatest mean IPF values were seen in hemolytic anemia, AML, and IDA; 26.97%, 21.5%, and 19.8%, respectively. These results are in line with the pathophysiological processes in these disorders, where heightened compensatory thrombopoiesis is required due to increased platelet consumption or destruction. Interestingly, none of the patients had an IPF < 1%, indicating that platelet generation is active, albeit to differing degrees, even in relatively stable conditions like multiple myeloma or CLL. We now know that inefficient thrombopoiesis and megakaryocyte hypoplasia are the causes of decreased platelet production. Reduced megakaryocyte growth, bone marrow replacement by cancerous cells, or fibrosis are the causes of megakaryocyte hypoplasia [37].

Further information was obtained by classifying the IPF values into three groups (<1%, 1–10%, and >10%). The majority of patients in conditions including AML, IDA, and NHL/HL have IPF >10% (100%, 83.3%, and 80%, respectively), indicating strong compensatory platelet production. In contrast, a higher percentage of patients with IPF in the intermediate range (1–10%) in diseases such as multiple myeloma and CLL had a less obvious requirement for compensatory thrombopoiesis. The distribution of IPF categories also lends credence to the idea that diseases like hemolytic anemia and ITP, which have accelerated platelet breakdown or consumption, are more likely to have elevated IPF values. For instance, IPF >10% was present in 75% of patients with hemolytic anemia and ITP, confirming the link between these disorders and elevated platelet turnover. These results were compatible with the results revealed by others [38-42]. Clinically, the constant lack of IPF <1% emphasizes how useful IPF is for identifying even minute thrombopoietic activity. Including IPF measures in standard diagnostic and monitoring procedures may be beneficial for disorders including NHL/HL, IDA, and AML, which have shown a high prevalence of IPF >10%. Elevated IPF, for example, may indicate the marrow's reaction to iron supplementation in IDA instances. Similarly, monitoring IPF may offer important information on marrow recovery following treatment for hematologic malignancies like AML. To distinguish ITP from other types of thrombocytopenia, computerized measurement of reticulated platelets is being carried out [43, 44]. Monitoring patient response can be aided by this measure, especially when immunosuppressive medication is switched [45]. IPF determination is a rapid and simple way to get information on bone marrow megakaryocytic activity and platelet generation. Additionally, it can be used as a gauge for platelet recovery in recipients of chemotherapy or hematopoietic stem cell transplants.

Results of the current study showed that the IPF% and PLT had a strong inverse relationship ($r = -0.246$, $p = 0.011$). This result lends credence to the theory that thrombocytopenia causes bone marrow activity to rise (Table 5), which in turn causes an increase in IPF% as a compensatory response. This association is further demonstrated by the scatter plot (Figure 1), which shows clear trends among disorders. Outliers, such as patients with abnormally high or low IPF% for a particular platelet count, may also be visible in the plot. These could point to particular clinical situations or measurement variability. The scatter figure illustrates how IPF% may be used as a diagnostic tool to distinguish between thrombocytopenia's various origins. These results are in agreement with others [46-48].

Conclusion

In thrombocytopenic situations, this study emphasizes the clinical relevance of the IPF as a sensitive indicator of bone marrow activity and compensatory platelet synthesis. When compared to controls, elevated IPF levels in thrombocytopenic patients show how useful it is for detecting active platelet turnover. The bone marrow's

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compensating reaction to thrombocytopenia is highlighted by the inverse relationship between PLT and IPF%. Moreover, disease-specific platelet production patterns are reflected in differences in IPF values among diagnoses. The conditions with the highest mean IPF levels, which indicate increased thrombopoietic activity, included hemolytic anemia, AML, and IDA. On the other hand, diseases like CLL and multiple myeloma displayed intermediate IPF levels, indicating less forceful compensatory mechanisms. The study also highlights the disparities in thrombocytopenia prevalence between genders and the difficulties in diagnosing a variety of underlying illnesses, such as systemic disorders and hematological malignancies. By using IPF in regular diagnostics, it is possible to quickly and non-invasively monitor bone marrow function, evaluate therapy response, and differentiate between thrombocytopenia causes, all of which can provide important information for patient management. Lastly, this study demonstrates the value of a comprehensive approach to data collection and analysis in revealing patterns that may guide future research and clinical practice. Therefore, future research should aim to validate these findings in larger, more diverse cohorts and explore the predictive value of IPF in treatment outcomes. Longitudinal studies could also elucidate the dynamic changes in IPF throughout disease progression and therapy.

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Jbireal M J; Writing, resources and supervision, S. R Alzzahani; Laboratory analysis; and review. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest
The authors declare no conflicts of interest.

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