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ORIGINAL ARTICLE

Correlation of thrombocytosis with markers of iron profile among patients diagnosed with iron deficiency anemia.

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ABSTRACT… Objective: To correlate thrombocytosis with markers of iron profile (serum iron, total iron binding capacity, serum ferritin and transferrin saturation). **Study Design:** Cross-sectional Prospective study. **Setting:** The Hematology Out-patient Department, Noor Thalassemia Foundation. **Period:** August 10, 2024 to August 10, 2025. **Methods:** A total of 142 patients having iron deficiency anemia were enrolled in the study by consecutive sample technique. Blood samples were drawn and tested for complete blood count and markers of iron profile (serum iron, total iron binding capacity, serum ferritin and transferrin saturation). The correlation between marker of iron profile and platelet count was observed using Pearson correlation. Our study cohort was divided in two groups: group A (platelet count >450x10^9/L) and group B (platelet count <450x10^9/L). Independent T test was applied to find the hematological and chemical findings of patients of both groups. **Results:** Platelet count showed significantly inverse relationship with serum iron (r = -0.192, p = 0.022), transferrin saturation (r = -0.213, p = 0.011) and serum ferritin (r = -0.178, p = 0.049) while a significantly positive correlation with TIBC (r = +0.165, p = 0.034). Transferrin saturation differs significantly in both groups, group A (4.67%) vs. group B (6.63%) (p=0.014). **Conclusion:** The inverse association between platelet count and markers of iron status confirms that thrombocytosis in IDA is a reactive process secondary to iron deficiency. Thrombocytosis in iron deficiency anemia reflects the severity of underlying iron deficiency.

Key words: Iron deficiency Anemia, Serum Ferritin, Transferrin Saturation, Thrombocytosis.

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INTRODUCTION

Iron deficiency anemia is the most prevalent form of nutritional deficiency disorder in developing countries, affecting a significant proportion of the population. The population groups affected by iron deficiency anemia particularly include women of reproductive age, children under age of five and individuals belonging to middle or low income group. Iron deficiency anemia is characterized by reduction in hemoglobin synthesis due to an inadequate supply of iron.[1]

Hematologically, iron deficiency anemia presents as hypochromic microcytic anemia.[2] Various biochemical markers of iron profile -such as serum iron, total iron binding capacity (TIBC), serum transferrin saturation and serum ferritin- help in diagnosis of iron deficiency anemia.[3] Serum iron represents the amount of total iron bound to transferrin circulating in the blood, total iron binding capacity represents the total capacity of transferrin to bind with iron, transferrin saturation indicates the fraction of iron bound to transferrin available for erythropoiesis and serum ferritin reflects total iron stores in the body.[4] In iron deficiency anemia, serum iron, transferrin saturation, serum ferritin are below 50 µg/dL, 14%, 15 ng/mL respectively while TIBC is above 450 µg/dL.[5]

The most common hematological observation associated with iron deficiency anemia is thrombocytosis, defined as the platelet count greater than 450x10^9/L.[6] Thrombocytosis is classified as primary thrombocytosis (clonal) in which platelet count increases due to bone marrow disorders such as myeloproliferative disorders or secondary (reactive) in which platelet count increases due to physiological or pathological conditions including inflammation, infections (particularly acute infections as part of the acute-phase response) or iron deficiency anemia.[7]

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In reactive thrombocytosis, platelet count usually returns to normal once the underlying cause is resolved.[8,9]

The mechanism of thrombocytosis in iron deficiency anemia is not fully known, though many hypotheses have been proposed. One possible explanation for this phenomenon is increase in level of erythropoietin (EPO) due to tissue hypoxia in iron deficiency anemia.[10] Since megakaryocytes and erythroid progenitors share a common precursor[11], high levels of erythropoietin and cytokine-mediated stimulation promote megakaryocytes proliferation and platelet production.[12,13] Cytokines involved in this process are mainly interleukin-6 (IL-6) and thrombopoietin.[14] Another proposed mechanism explains that iron plays a regulatory role in thrombopoiesis, iron deficiency limits its inhibitory role in thrombopoiesis, thus enhancing the platelets production.[15,16]

Since thrombocytosis is the hallmark manifestation of iron deficiency anemia, its correlation with parameters of iron profile (serum iron, TIBC, transferrin saturation and serum ferritin) is not well defined. Understanding this relationship will provide better insight into degree of iron deficiency reflected by platelets response. This will also assist in differentiating reactive thrombocytosis secondary to iron deficiency from thrombocytosis due to other causes.

Correlating thrombocytosis and other parameters of iron profile will improve understanding of hematological adaptation in iron deficiency. The results of this study will serve as an additional supportive marker for assessing iron profile in resource-limited settings where advanced iron studies are not routinely available.

METHODS

This cross sectional observational study was conducted at the Outpatient department of Noor Thalassemia Foundation, Lahore over a period of one year i.e., from August 10, 2024 to August 10, 2025 after approval from ethical review committee on 11-4-24. This study involved 142 patients with confirmed diagnosis of iron deficiency anemia. Inclusion criteria involved hemoglobin <12g/dL for females and <13g/dL for males, mean corpuscle volume (MCV) < 80fL, mean corpuscle hemoglobin (MCH) <27pg, serum ferritin <15 ng/dL.

Patients with other forms of anemia (e.g., anemia of chronic disorder), those with history of blood transfusion in recent one month, or taking iron supplements were excluded from study. Individuals with ongoing inflammation detected by using CRP were also excluded.

After obtaining informed consent from patients 5mL of blood was drawn and subjected to complete blood count (CBC) using Sysmex KX21 (Sysmex corporation, Kobe, Japan). Serum iron, TIBC, serum transferrin saturation, serum ferritin were analyzed using ARCHITECT i1000SR (Abbott Laboratories, Chicago, IL, USA,) immunoassays. Results of each patients were verified against internal quality control to ensure analytical accuracy.
Participants were classified into two groups,
Group A: participants with platelet count >450x10^9/L
Group B: participants with platelet count <450x10^9/L

Correlation between platelet count and four parameters of iron profile i.e., serum iron, TIBC, serum transferrin saturation and serum ferritin was analyzed using Pearson's correlation, a p-value <0.05 was considered statistically significant. Independent T-test was applied to evaluate hematological and biochemical differences between both groups. All data analysis was done using statistical package for social sciences SPSS version 26.0.

RESULTS

Our study consisted of 142 individuals with confirmed diagnosis of iron deficiency anemia ranging from 1 year to 88 years of age. The mean age of our participants was 28.11 ± 21 years. Majority of the participants belonged to 1 to 12 years of age group. Number of males in our study was 44 (31%) while that of females was 98 (69%). Male to female ratio in our study population was approximately 9:20. Number of males and females in each age group are enlisted in Table-I.

All the study parameters including red blood cells (RBC) count, hemoglobin (Hb) concentration, Mean corpuscular volume (MCV), Mean corpuscle hemoglobin (MCH), Mean corpuscle hemoglobin

concentration (MCHC), White blood cell count (WBC), Platelet count, serum iron, total iron binding capacity, serum transferrin saturation and serum ferritin of 142 participants are listed in Table-II as mean ± S.D.

TABLE-I

Number of males and females in each age group

Age Group	Number of Males	Number of Females	Total
1-12 years	28	16	44 (31%)
13-24 years	01	21	22 (15.5%)
25-36 years	5	18	23 (16.3%)
37-48 years	0	33	33 (23.2%)
49-60 years	04	05	09 (6.2%)
>60 years	06	05	11 (7.8%)
Total	44	98	142

TABLE-II

Mean ± S.D of all the parameters.

Parameters	Mean ± S.D
Number of Patients	142
RBC ($x10^{12}$/L)	4.9±3.5
Hb (g/dL)	8.9±2.40
MCV (fL)	62.72±8.5
MCH (%)	18.35±3.6
MCHC (%)	28.91±3.11
WBC ($x10^9$/L)	8.9±2.9
Platelets ($x10^9$/L)	406.11±140
Serum Iron (µg/dL)	21.96±13.6
TIBC (µg/dL)	431.4±108.6
Transferrin Saturation (%)	5.59±4.14
Ferritin (ng/mL)	8.8±6.85

Pearson correlation of Platelet count with parameters of iron profile revealed negative correlation with serum iron (r = -0.192, p = 0.022), transferrin saturation (r = -0.213, p = 0.011) and serum ferritin (r = -0.178, p = 0.049) while a positive correlation with TIBC (r = +0.165, p = 0.034). Platelet count has statistically significant correlation (p-value <0.05) with all the four parameters of iron profile (serum iron, TIBC, transferrin saturation, serum ferritin).

Out of 142 participants, 75 (53%) showed thrombocytosis i.e., platelet count >450x10^9/L, 4 (3%) had platelet count <150x10^9/L and 63 (44%) had normal platelet count 150 – 450 x10^9/L. All the participants were stratified into 2 groups: Group A and Group B. Group A had participants with platelet count >450x10^9/L while group B had participants with platelet count <450x10^9/L. Independent T-test was applied to calculated hematological and biochemical findings of both group and these are enlisted below in Table-IV.

Table-III

Pearson correlation platelet count with serum iron, TIBC, transferrin saturation, serum ferritin

	r-value	P-Value
Serum iron	-0.192	0.022
TIBC	+0.165	0.034
Transferrin saturation	-0.213	0.011
Serum ferritin	-0.178	0.049

TABLE-IV

Hematological and biochemical comparison of groups showing thrombocytosis and normal platelet count

	Group A (n=75)	Group B (n=67)	P-Value
RBC ($x10^{12}$/L)	5.45±4.86	4.3±0.64	0.114 NS
Hb (g/dL)	8.04±1.91	8.7±2.8	0.210 NS
MCV(fL)	60.62±8.75	65.07±7.71	0.284 NS
MCH (%)	17.21±3.47	19.62±3.44	0.973 NS
MCHC (%)	28.33±2.89	29.56±3.23	0.474 NS
WBC ($x10^9$/L)	9.54±2.79	8.35±3.0	0.496 NS
Platelets ($x10^9$/L)	607.31±104.87	342.82±71.81	0.007 S
Serum Iron (µg/dL)	19.47±11.24	24.76±15.49	0.084 NS
TIBC (µg/dL)	446±94.36	415±121	0.555 NS
Transferrin Saturation (%)	4.67±3.25	6.63±4.77	0.014 S
Ferritin (ng/mL)	7.5±6.1	10.4±7.34	0.099 NS

DISCUSSION

The demographic distribution of the study cohort revealed noteworthy insights, a strong predominance of females (69%) and a major representation of two age groups: young children (1-12 years) and women of reproductive (37-48 years). This pattern is highly consistent with the epidemiology of iron deficiency anemia, where the etiology is primarily nutritional among children and related to menstrual blood loss

and pregnancy among women of childbearing age.[17]

The present study confirms a high prevalence (53%) of reactive thrombocytosis in patients with iron deficiency anemia. The presence of thrombocytosis in hypochromic microcytic anemia can be used as a diagnostic clue pointing towards iron deficiency anemia as an underlying cause.[18] This observation is highly valuable in differentiating iron deficiency anemia from other causes of hypochromic and microcytic anemia such as anemia of chronic disorder and beta thalassemia trait.[19] Platelet count in anemia of chronic disorder is usually normal or below normal due to inflammatory suppression of platelets.[20]

In the current study, platelet count exhibited a significant inverse correlation with serum iron, (r = -0.192, p = 0.022), transferrin saturation (r = -0.213, p = 0.011) and serum ferritin (r = -0.178, p = 0.049) - a sensitive marker of iron status. These results suggest that the individuals with lower iron levels tend to exhibit more platelet count. Platelet count also showed a significant positive relationship with TIBC (r = +0.165, p = 0.034). When the iron deficiency increases- reflected by TIBC, the platelet count also tend to rise. Although the correlations were of weak strength, their statistical significance suggests that the relationship is consistent and not due to random variation. Similar observations were reported by Kuku et al. and Patel et al. who demonstrated that platelet count correlates with markers of iron deficiency among iron deficiency anemia patients.[21,22] These findings emphasize the need for evaluating the iron profile in patients with hypochromic microcytic anemia and thrombocytosis before considering primary thrombocytosis as a differential diagnosis.

The most striking findings that emerged from the comparison of group A (patients with thrombocytosis, mean platelets = 607.31 ± 104.87 x 10^9/L) and Group B (patients without thrombocytosis, mean platelets = 342.82 ± 71.81 x 10^9/L) revealed that there was no significant difference between the red cell indices (RBC, Hb, MCV, MCH, MCHC) or white blood cell count between both the groups. The lack of significant differences between these parameters suggests that variation observed in platelet count is not due to hematopoietic suppression or alteration in bone marrow. Instead, these findings point toward a specific alteration in the megakaryocytes lineage, likely influenced by iron status or thrombopoietic regulation instead of a broad effect on all blood cell lines. Similar findings described by Li et al. explains the iron deficiency anemia selectively affects platelet production through altered thrombopoietin dynamics and bone marrow micro environmental changes.[23]

Furthermore, patients of group A demonstrated a statistically significant lower serum transferrin saturation as compared to group B (4.67% vs. 6.63%, p = 0.014). Although the values of serum iron and serum ferritin were lower in group A than group B, they did not reach a statistically significant association: serum iron (19.47 µg/dL vs. 24.76 µg/dL p = 0.084) and serum ferritin (7.5 ng/mL vs. 10.4 ng/mL p = 0.099). This pattern is highly revealing that group A (showing thrombocytosis) has more profound functional iron deficiency. The lower transferrin saturation indicates a lower proportion of circulating transferrin is bound to iron in group A thus limiting iron for erythropoiesis. This iron starved erythropoiesis particularly reflected by lower transferrin saturation appears to be the stimulus for increased platelet production.[24]

Our findings support the pathophysiological link between iron and thrombocytosis. Chronic and severe iron deficiency changes the bone marrow environment by enhancing thrombopoiesis levels or reducing iron-mediated inhibition of megakaryopoiesis.

CONCLUSION

Thrombocytosis is a frequent hematological finding in patients with iron deficiency anemia and correlates significantly with the severity of iron depletion. Platelet counts showed inverse correlation with serum iron, serum ferritin and transferrin saturation, while positive correlation with TIBC, confirming association with iron status. The findings of our study confirm that thrombocytosis in iron deficiency anemia represents a reactive process secondary to iron depletion and may serve as a clinical indicator of more advanced iron store depletion, particularly characterized by critically low transferrin saturation.

Understanding this correlation will help in diagnosis, differentiation from primary thrombocytosis, and effective management of patients with iron deficiency anemia.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Saima Mansoor Bugvi:** Conceptualization of study.
2	**Kainaat Mahzaib John:** Literature search.
3	**Rabia Rasheed:** Data collection.
4	**Tayeba Ajmal:** Data entry.
5	**Shafqat Hussain Khan:** Data analysis.
6	**Muhammad Azeem:** Data collection.