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Original Research Article

Haematological abnormalities in transfusion dependent thalassemia patients: an observational study

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ABSTRACT

Background: Thalassemia is a common inherited hemoglobinopathy, with transfusion-dependent thalassemia (TDT) representing the most severe phenotype. Despite advances in transfusion and chelation therapy, patients remain at risk of hematological derangements arising from ineffective erythropoiesis, chronic anemia, and iron overload. Comprehensive evaluation of hematological indices in this population is essential for assessing transfusion adequacy and predicting complications. Objectives were to evaluate hematological abnormalities in transfusion-dependent thalassemia patients and to assess correlations between hematological indices and serum ferritin levels.

Methods: This hospital-based observational cross-sectional study was conducted in the departments of pediatrics and pathology, GMERS Medical College, Junagadh, over five months. A total of 43 children and adolescents with TDT, on regular transfusion for at least one year, were enrolled through universal sampling. Venous blood was collected prior to transfusion and analyzed using an automated hematology analyzer for hemoglobin (Hb), hematocrit (HCT), red blood cell (RBC) count, mean corpuscular indices (MCV, MCH, MCHC), red cell distribution width (RDW), total leukocyte count (TLC), and platelet count. Serum ferritin was measured using a fully automated biochemistry analyzer. Data were analyzed with SPSS v20; continuous variables were expressed as mean±SD, and correlations with ferritin were assessed by Pearson's coefficient.

Results: Patients had persistent moderate anemia with mean hemoglobin 7.48±1.24 gm/dl and hematocrit 22.53±4.21%. RBC counts averaged 2.93±0.51 $\times 10^6$/µl, with microcytosis (MCV 76.83±5.32 fl), low MCH (24.91±2.90 pg), and relatively preserved MCHC (32.39±3.01 gm/dl). RDW was markedly raised (18.68±4.33%). TLC showed wide variability (11,418.60±8,351.91/µl), and thrombocytosis was frequent (platelets 445,667±210,270/µl). Serum ferritin was markedly elevated (4,236.32±3,248.98 ng/ml). Correlations between ferritin and Hb, HCT, MCH, and platelets were negligible or weak and statistically non-significant ($p>0.05$).

Conclusions: Transfusion-dependent thalassemia patients exhibited persistent anemia, microcytosis, hypochromia, anisocytosis, and frequent thrombocytosis despite regular transfusions. Serum ferritin levels, although elevated, showed poor correlation with hematological indices, underscoring their independent role as markers of iron overload rather than hematologic status. Continuous monitoring of complete blood counts alongside ferritin remains crucial for guiding management and preventing complications.

Keywords: Anemia, Hematological abnormalities, Red cell indices, Serum ferritin, Thalassemia, Transfusion-dependent thalassemia

INTRODUCTION

Thalassemia represents one of the most common inherited hemoglobin disorders worldwide, with transfusion-dependent β-thalassemia (TDT) constituting the most severe form. Affected patients suffer from defective hemoglobin synthesis leading to chronic anemia, necessitating lifelong regular transfusion support to sustain adequate hemoglobin levels. Advances in transfusion and chelation therapy have significantly improved survival; however, these patients remain vulnerable to multisystem complications arising from iron overload and ineffective erythropoiesis.[1,2]

Hematological abnormalities in TDT are central to its clinical presentation and long-term course. Characteristically, these patients exhibit markedly reduced hemoglobin and hematocrit levels, reflecting the underlying ineffective erythropoiesis and increased peripheral hemolysis. Alterations in red blood cell indices, such as a low mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), often accompanied by a relatively preserved mean corpuscular hemoglobin concentration (MCHC), are typical of microcytic hypochromic red blood cells. In addition, increased red cell distribution width (RDW) reflects anisocytosis, a consequence of both marrow stress and repeated transfusions.[3,4]

White blood cell and platelet counts may also demonstrate variability. In some patients, splenic dysfunction, hypersplenism, or splenectomy contributes to altered leukocyte and platelet counts. Iron overload and marrow expansion further modify hematopoiesis, resulting in significant heterogeneity of hematological findings among patients. Such changes not only provide insight into disease severity but also influence clinical decision-making related to transfusion requirements and long-term monitoring.[5,6]

Serum ferritin, although primarily considered a biochemical marker of iron overload, also has clinical relevance when studied in relation to hematological parameters. Elevated ferritin levels correlate with iron deposition in various organs, yet their interplay with hematological indices may reflect the cumulative burden of transfusion, ineffective erythropoiesis, and tissue damage. Correlation analysis of ferritin with hematological parameters may therefore offer an additional dimension for understanding the disease burden in these patients.[7,8]

Given the paucity of studies that comprehensively describe hematological abnormalities in TDT patients within the same cohort and evaluate their correlation with iron overload, there exists a need for region-specific evidence. Early identification of deranged hematological indices can improve the monitoring of transfusion adequacy and predict long-term complications. The present study was undertaken to evaluate the hematological profile of transfusion-dependent thalassemia patients by assessing hemoglobin, hematocrit, red blood cell indices, total leukocyte count, and platelet count, and to determine their correlation with serum ferritin levels.

METHODS

Study setting, type, and duration

This hospital-based observational cross-sectional study was conducted in the department of pathology in collaboration with the department of pediatrics at GMERS Medical College and Hospital, Junagadh, Gujarat. The study was conducted retrospectively, following approval from the institutional ethics committee.

Study participants

The study population comprised children and adolescents diagnosed with transfusion-dependent thalassemia (TDT) who attended the pediatric thalassemia clinic for regular blood transfusions. Patients of either sex, aged between 1 and 18 years, who had been on regular transfusion therapy for a minimum duration of one year were included in the study. Exclusion criteria included patients with other hemoglobinopathies, such as sickle cell anemia, HbE disease, HbE-β thalassemia, or hereditary red cell disorders like G6PD deficiency and hereditary spherocytosis, as well as children with concurrent acute systemic illnesses or infections that could alter hematological results.

Sample size and sampling technique

A total of 43 transfusion-dependent thalassemia patients fulfilling the inclusion criteria were enrolled. All eligible patients who attended the thalassemia clinic during the study period were included by applying a universal sampling technique.

Data collection and laboratory investigations

Venous blood samples were collected under aseptic precautions before transfusion. For hematological evaluation, samples collected in EDTA vacutainers were analyzed using an automated hematology analyzer (Horiba ABX Micro). The parameters assessed included hemoglobin (Hb), hematocrit (HCT), red blood cell (RBC) count, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW), total leukocyte count (TLC), and platelet count. In addition, serum ferritin estimation was performed using a fully automated biochemistry analyzer (VITROS 5.1 FS Chemistry System) to assess iron overload and explore its correlation with hematological indices. All laboratory procedures were performed in accordance with strict internal and external quality

control measures to ensure the accuracy and reliability of the results.

Ethical issues

The study protocol was reviewed and approved by the institutional ethics committee of GMERS Medical College, Junagadh. Written informed consent was obtained from the parents or legal guardians of the participants before their inclusion. Patient confidentiality was maintained throughout the study.

Data analysis

All data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20. Continuous variables were expressed as mean and standard deviation. The correlation between serum ferritin and selected hematological indices was assessed using Pearson's correlation coefficient, with a p value of less than 0.05 considered statistically significant.

RESULTS

Table 1 underscores a mid-childhood skew: most patients were aged 6–10 years (37.2%), with adolescents (≥11 years) close behind (34.9%), while the ≤5-year group formed the remaining 27.9% of the sample (n=43).

Table 1: Age distribution of patients (n=43).

Age group (years)	Frequency	Percentage
≤5	12	27.9
6-10	16	37.2
≥11	15	34.9

Table 2 highlights a classic thalassemia pattern- moderate anemia with microcytosis and hypochromia- reflected by hemoglobin 7.48±1.24 gm/dl, hematocrit 22.53±4.21%, RBC 2.93±0.51×10⁶/µL, MCV 76.83±5.32 fL, MCH 24.91±2.90 pg, a relatively preserved MCHC (32.39±3.01 gm/dl), and a conspicuously raised RDW (18.68±4.33%).

Table 2: Hematological parameters in transfusion-dependent thalassemia patients.

Parameter	Mean±SD	Minimum	Maximum
Hemoglobin (gm/dl)	7.48±1.24	4.50	10.00
Hematocrit (%)	22.53±4.21	14.20	31.50
RBC count (M/µl)	2.93±0.51	2.00	4.41
MCV (fl)	76.83±5.32	64.00	86.00
MCH (pg)	24.91±2.90	18.80	35.20
MCHC (gm/dl)	32.39±3.01	28.00	49.50
RDW (%)	18.68±4.33	13.00	30.60
Serum ferritin (ng/ml)	4,236.32±3,248.98	6.10	11,349.00

Table 3: Leukocyte and platelet counts.

Parameter	Mean ± SD	Minimum	Maximum
Total leukocyte count (/µl)	11,418.60 ± 8,351.91	3,100	45,400
Platelet count (/µl)	445,666.67 ± 210,270.17	121,000	953,000

Table 4: Correlation of serum ferritin with hematological parameters.

Parameter	Correlation coefficient (r)	P value	Interpretation
Hemoglobin	–0.084	0.600	Negligible negative
Hematocrit	–0.013	0.938	Negligible negative
MCH	0.192	0.236	Weak positive
Platelets	–0.087	0.595	Negligible negative

From Table 3, variability in cell counts is notable: total leukocytes averaged 11,418.60±8,351.91/µl across a wide range, and platelet counts were frequently elevated (445,666.67±210,270.17/µl), indicating a common occurrence of thrombocytosis.

As depicted in Table 4, serum ferritin showed little to no meaningful association with key hematological indices- negligible, non-significant correlations with hemoglobin (r=−0.084; p=0.600) and hematocrit (r=−0.013; p=0.938), and only weak, non-significant links with MCH (r=0.192; p=0.236) and platelets (r=−0.087; p=0.595).

DISCUSSION

This study confirms the classic hematologic picture of TDT, despite regular transfusions: moderate to severe anemia, microcytosis, and hypochromia, with elevated

RDW, and frequent leukocytosis and thrombocytosis. The mean hemoglobin of 7.48 gm/dl in this cohort lies within the expected TDT range and aligns with contemporary series. For example, an Indian tertiary-center cohort of β-thalassemia major (BTM) reported a mean Hb of 6.3 gm/dl with an MCV of 71.4 fl and RBC count of 2.9×10^6/µl, closely mirroring our RBC count and microcytosis, albeit at a slightly lower Hb level than in our series.[9]

Hemoglobin and hematocrit

The present mean Hb (7.48 gm/dl) is higher than values reported by some pediatric BTM series from south Asia- e.g., mean Hb 6.28±1.75 gm/dl in thalassemia major patients within a pediatric anemia profile and ~5.1 gm/dl in β-thalassemia in a clinico-hematologic pediatric survey- both indicating more profound anemia than observed here, possibly reflecting differences in transfusion targets and clinic attendance intervals.[10,11] Microcytosis/hypochromia: Your MCV (76.83 fl) and MCH (24.91 pg) are directionally consistent with BTM, although several references describe typical MCV values lower than ours (often ~50-70 fl), underscoring that pre-transfusion timing and recent transfusion history can raise indices. GeneReviews summarizes BTM red cell indices as MCV ~50-70 fl and Hb generally <7-10 gm/dl; our data sit toward the milder end, again compatible with ongoing transfusion support.[12]

The elevated RDW in our cohort (18.68%) reinforces significant anisocytosis. While many RDW publications focus on trait/NTDT or the IDA vs thalassemia differential, they still support the principle that thalassemia groups have elevated RDW compared with healthy populations; Qurtom et al showed RDW elevation "in all patients with thalassemia major", and modern diagnostic papers highlight RDW's discriminatory utility (e.g., RDW>21% favoring NTDT over IDA), situating our mean RDW in the expected thalassemia spectrum but below NTDT-oriented cut-offs.[13,14]

The wide TLC dispersion we observed (mean ~11.4×10^3/µl; range 3.1-45.4 × 10^3/µl) is consistent with reports that many TDT patients exhibit leukocytosis, particularly when splenic function is impaired. In a previous study, 58.8% of BTM patients had high WBC counts, and 35.3% had thrombocytosis, particularly after splenectomy, which corroborates our frequent leukocytosis and high platelet counts.[15]

Platelet behavior is strongly influenced by splenic status; pediatric and surgical series show substantial post-splenectomy thrombocytosis- e.g., mean platelet count ~644,700±299,400/µl after splenectomy- values comparable to the upper range in our sample. Additional pediatric BTM work has also documented higher platelet counts among splenectomized patients compared to non-splenectomized patients.[16,17] These hematologic perturbations dovetail with the well-described prothrombotic milieu in thalassemia (platelet activation, endothelial dysfunction), lending clinical weight to monitoring and prophylaxis considerations where indicated.[18]

From a practice perspective, the constellation of low Hb/Hct, microcytosis, and high RDW underscores the need to optimize transfusion schedules to maintain guideline-consistent pre-transfusion Hb targets while minimizing viscosity-related risks. Elevated RDW, although nonspecific, remains a pragmatic indicator of ineffective erythropoiesis and optimal transfusion timing. Persistent thrombocytosis- especially in patients who have undergone splenectomy- has implications for hypercoagulability, as echoed in both mechanistic and clinical literature. Therefore, vigilance for thrombotic events and individualized antiplatelet strategies are prudent in high-risk phenotypes.[18]

In our cohort, serum ferritin correlated weakly or not at all with core hematologic indices (e.g., Hb r=–0.084; Hct r=–0.013; platelets r=–0.087), suggesting iron burden (as reflected by ferritin) is a poor surrogate for day-to-day hematologic status in TDT- a result that is biologically plausible because Hb levels are primarily transfusion-determined. Ferritin is both an iron load and an acute-phase marker. Population studies evaluating broad hematologic and biochemical profiles in BTM similarly emphasize that iron overload metrics and CBC parameters can behave semi-independently, with significant enzyme or organ changes alongside chronically low indices, rather than tight linear coupling between ferritin and Hb/indices.[19]

CONCLUSION

The study shows that transfusion-dependent thalassemia was marked by persistent moderate anemia (hemoglobin 7.48±1.24 gm/dl; hematocrit 22.53±4.21%), microcytosis and hypochromia (MCV 76.83±5.32 fl; MCH 24.91±2.90 pg) with relatively preserved MCHC, and pronounced anisocytosis (RDW 18.68±4.33%); leukocyte counts displayed wide dispersion (3,100-45,400/µl; mean 11,418/µl) and thrombocytosis was frequent (platelets 445,667±210,270/µl), while correlations between serum ferritin and core hematological indices were negligible and non-significant. These findings support integrating CBC-based surveillance- particularly red-cell indices, RDW, and platelet trends- with protocolized pre-transfusion hemoglobin targets, and interpreting ferritin primarily as an indicator of iron overload. Further longitudinal work, stratifying by splenic status and chelation intensity, is warranted to refine risk stratification.

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