Assessment of Iron Deficiency in Cyanotic Congenital Heart Diseases

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ABSTRACT

Background: Cyanotic congenital heart diseases (CCHDs) are structural heart anomalies resulting in systemic hypoxemia. While secondary erythrocytosis is a compensatory response to chronic hypoxia, iron deficiency may complicate this adaptation, potentially exacerbating morbidity and reducing oxygen-carrying efficiency. Aim of the study: To evaluate the prevalence of iron deficiency in children with CCHD and to identify the diagnostic utility of basic hematologic parameters in comparison to definitive biochemical tests. **Methods & Materials**: This cross-sectional observational study included 20 children with CCHD who had not undergone corrective surgery. Participants were divided into two groups based on serum ferritin levels: iron-deficient (Group A) and iron-sufficient (Group B). Hematological parameters (Hb, PCV, MCV, MCH, MCHC, RDW, peripheral smear) and biochemical markers (serum iron, TIBC, transferrin saturation) were analyzed. Statistical analysis included Chi-square tests and logistic regression. **Result**: Iron deficiency was confirmed in 50% of participants based on serum ferritin and transferrin saturation. Strong correlations were found between iron deficiency and microcytic hypochromic indices. RDW emerged as the only independent hematologic predictor of iron deficiency ($p = 0.04$). Clinical features such as underweight status and male predominance were more common among iron-deficient children. **Conclusion**: Routine hematologic tests, especially RDW, can be useful screening tools for iron deficiency in CCHD patients when biochemical testing is unavailable. Early detection and intervention are crucial to prevent complications related to iron-deficient erythropoiesis.

Keywords: *Cyanotic congenital heart disease, Iron deficiency, Serum ferritin, Red cell distribution width, Pediatric cardiology*

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INTRODUCTION

Congenital heart disease (CHD) encompasses a diverse range of structural abnormalities of the heart and great vessels present from birth, accounting for nearly one-third of all congenital anomalies globally [1, 2]. An estimated 8 per 1,000 live births are affected by CHD worldwide [3]. Cyanotic congenital heart disease (CCHD) is a subgroup of congenital heart diseases characterized by a structural defect in the heart that leads to poor oxygenation of blood, resulting in cyanosis (bluish discoloration of skin and mucous membranes) [4,5]. Affecting approximately 25% of all congenital heart defects, CCHD represents a significant burden on pediatric populations globally, particularly in low- and middle-income countries [6]. In CCHD, a right-to-left shunt of desaturated blood leads to decreased systemic oxygen saturation, triggering increased erythropoietin production and secondary erythropoiesis to enhance tissue oxygenation [7]. This compensatory mechanism, however, results in polycythemia and hyperviscosity, which manifest clinically as thromboembolic events in affected children [7,8]. Iron deficiency anemia (IDA) is a common complication in children with cyanotic congenital heart disease (CCHD). The presence of IDA worsens hyperviscosity symptoms due to microcytic erythrocytes that are less deformable within the microcirculation. This further increases the risk of morbidity, particularly cerebrovascular events and cyanotic spells [9,10]. While polycythemia raises hemoglobin and hematocrit levels in these patients, these elevated values can mask the underlying iron-deficient status. Despite the high hemoglobin levels caused by chronic hypoxia, many children with CCHD experience depleted iron stores, a condition known as iron-deficient erythropoiesis [10]. This paradox occurs because the heightened need for iron to support increased erythropoiesis outstrips the body's iron supply [11]. The mechanisms driving iron deficiency in CCHD are multifactorial. Chronic hypoxia stimulates erythropoiesis, increasing the demand for iron, while factors such as gastrointestinal blood loss, impaired nutrient absorption, and reduced dietary iron intake further limit iron availability [12]. Moreover, polycythemia and hyperviscosity, commonly associated with CCHD, place additional stress on cardiac function, intensifying the systemic effects of iron deficiency [13]. This deficiency not only worsens cyanosis but also impairs physical and cognitive development, reducing exercise tolerance and delaying growth in affected children [14]. Early detection and proper treatment of anemia in patients with cyanotic congenital heart disease (CHD), particularly those presenting with heart failure, are highly recommended. Additionally, the prevention of thromboembolic events and cyanotic spells in children with cyanotic CHD has been emphasized in recent studies [15]. In industrialized nations, most cyanotic CHD cases are surgically managed during the neonatal or infant period, coupled with optimal nutritional support. However, in many developing countries, a significant number of children with complex CHD remain either untreated or partially treated [16]. Despite the clinical importance of iron

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deficiency in cyanotic CHD, its diagnosis and management pose ongoing challenges [17]. Anemia, particularly in children with cyanotic CHD who often exhibit elevated hematocrit levels due to chronic hypoxia, is frequently overlooked or underdiagnosed in clinical practice [17]. Therefore, this study aims to evaluate the prevalence of iron deficiency anemia in children diagnosed with cyanotic congenital heart disease.

METHODS & MATERIALS

Study Design

This study was a cross-sectional observational study conducted at Department of Cardiac Surgery, Bangladesh Medical University, Dhaka, Bangladesh from January 2024 to December 2024. The study aimed to assess the prevalence of iron deficiency in patients diagnosed with cyanotic congenital heart diseases (CCHD) and to evaluate the relationship between iron deficiency and clinical parameters. A total of 20 patients with CCHD were included in the study. All patients were divided into two groups.

Group A (n=10): Patients with iron deficiency
Group B (n=10): Patients with sufficient iron

Inclusion Criteria

- Children aged between 3 months and 18 years diagnosed with CCHD and who had not undergone corrective surgery were included in the study.

Exclusion Criteria

- Exclusion criteria comprised postoperative patients, children already receiving iron therapy, and those with acute or chronic infections.

Ethical Considerations

The study was conducted following approval from the institutional ethics committee. Informed consent was obtained from parents or legal guardians before participation. All laboratory tests were performed following standard hospital procedures, and no additional invasive procedures were conducted.

Data Collection

Demographic data were collected for each participant, including registration number, age, date of birth, and sex. Clinical history was documented, focusing on symptoms such as breathlessness, cyanosis, cyanotic spells, feeding difficulties, signs of heart failure, neurological deficits, and other comorbid conditions. Dietary intake was assessed using a 24-hour recall method. Anthropometric measurements included weight, measured using an electronic scale, and height, assessed using an infantometer or stadiometer, depending on the child's age.

To aid in further evaluation, additional diagnostic tests, including chest X-rays, electrocardiograms (ECG), and echocardiograms (ECHO), were performed. Laboratory investigations involved collecting 8 ml of venous blood from each participant, which was equally distributed into plain and EDTA sample tubes for analysis. Definitive tests included serum ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin saturation, while basic hematological tests comprised a complete blood count (CBC) and peripheral smear examination. The complete blood count was conducted using a fully automated bi-directional analyzer, and serum ferritin levels were measured via a chemiluminescent immunoassay. Serum iron levels were determined using the ferrozine method without deproteinization, whereas TIBC was assessed through a spectrophotometric assay, and transferrin saturation was calculated using the (serum iron/TIBC)×100 formula. A pediatric hematologist reviewed the peripheral smear results, while a pediatric cardiologist analyzed the chest X-ray and ECG findings to ensure a comprehensive evaluation.

Reference Values for Iron Deficiency in CCHD

Due to the limited availability of specific reference values for iron deficiency in cyanotic congenital heart disease (CCHD), standard laboratory guidelines were used in conjunction with hospital-based reference values. The applied cutoffs included serum ferritin levels of less than 12 ng/mL for children aged five years or younger and less than 15 ng/mL for those older than five years. Transferrin saturation below 16% was considered indicative of deficiency. Hemoglobin (Hb) levels lower than 15 g/dL and packed cell volume (PCV) below 60% were also used as diagnostic criteria. Additionally, a mean corpuscular volume (MCV) of less than 80 fL was considered significant. Due to the absence of established CCHD-specific guidelines, values for mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDW) were interpreted based on hospital reference standards.

Statistical Analysis

Categorical variables were reported as percentages. Comparisons between categorical variables were performed using the Chi-square test or Fisher's exact test, as appropriate. Continuous variables were analyzed using an independent t-test. Univariate analysis followed by multivariate logistic regression was conducted to determine independent correlations between simple hematological tests and serum ferritin levels. Data entry and statistical analysis were conducted using SPSS software version 26.0, with statistical significance set at $p < 0.05$.

RESULT

Table I presented the demographic characteristics of the study population (N=20), equally distributed between Group A and Group B (n=10 each). In Group A, most participants were aged 1–5 years (40.0%), while in Group B, the majority were >5 years (50.0%), with a significant difference in age distribution (p=0.04). Males predominated in Group A (70.0%), whereas Group B included equal proportions of males and females (50.0% each), showing statistical significance (p=0.03). Severe underweight was common in both groups, observed in 60.0% of Group A and 50.0% of Group B, with a significant association (p=0.05). Normal height was reported in 50.0% of participants in both groups, while severe stunting occurred in 30.0% of Group A and 40.0% of Group B. Erythrocytosis was found in 40.0% of Group A and 60.0% of Group B, while normal RBC count was observed in 60.0% and 40.0%, respectively (p=0.4).

Table I
Demographic characteristics of the study population (*n*=20).

Variables	Group A		Group B		P-value
	n	%	n	%	
Age (years)					
<1	3	30.00	3	30.00	0.04
1-5	4	40.00	2	20.00	
>5	3	30.00	5	50.00	
Gender					
Male	7	70.00	5	50.00	0.03
Female	3	30.00	5	50.00	
Weight					
Normal	2	20.00	4	40.00	0.05
Underweight	2	20.00	1	10.00	
Severe underweight	6	60.00	5	50.00	
Height					
Normal	5	50.00	5	50.00	0.8
Stunted	2	20.00	1	10.00	
Severe stunted	3	30.00	4	40.00	
Erythrocytosis					
Erythrocytosis	4	40.00	6	60.00	0.4
Normal RBC	6	60.00	4	40.00	

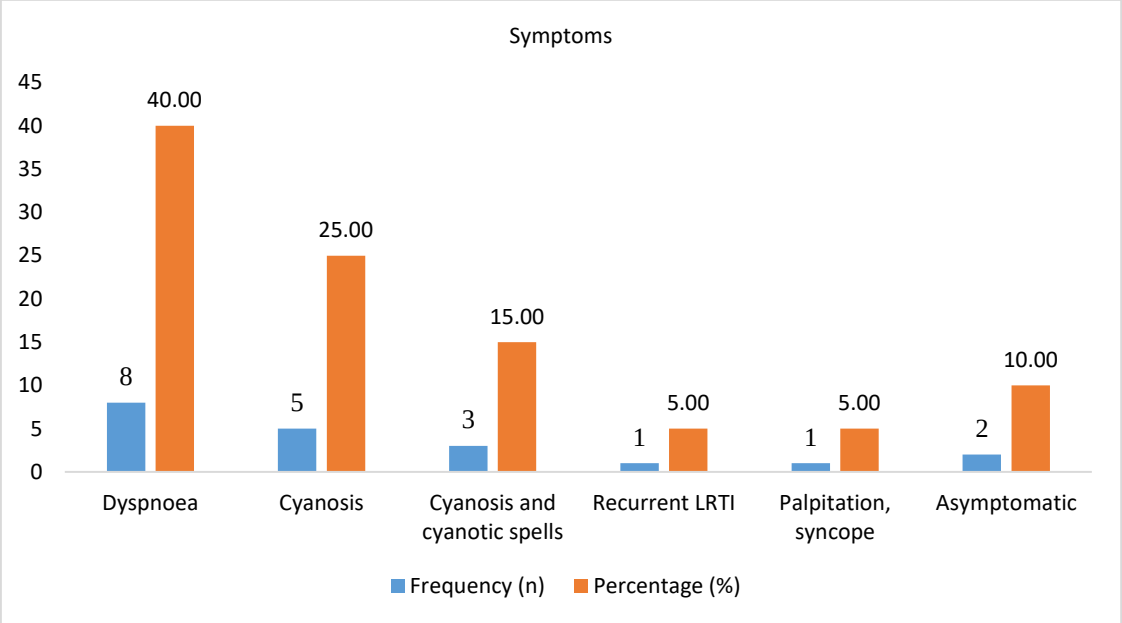

Figure 1 Symptoms presenting among study population (*n*=20)

Dyspnoea was the most common symptom, affecting 8 (40.0%) children, followed by cyanosis in 5 (25.0%) and cyanosis with cyanotic spells in 3 (15.0%). Recurrent LRTI and palpitation/syncope were reported in 1 (5.0%) child each, while 2 (10.0%) participants were asymptomatic (*Figure 1*).

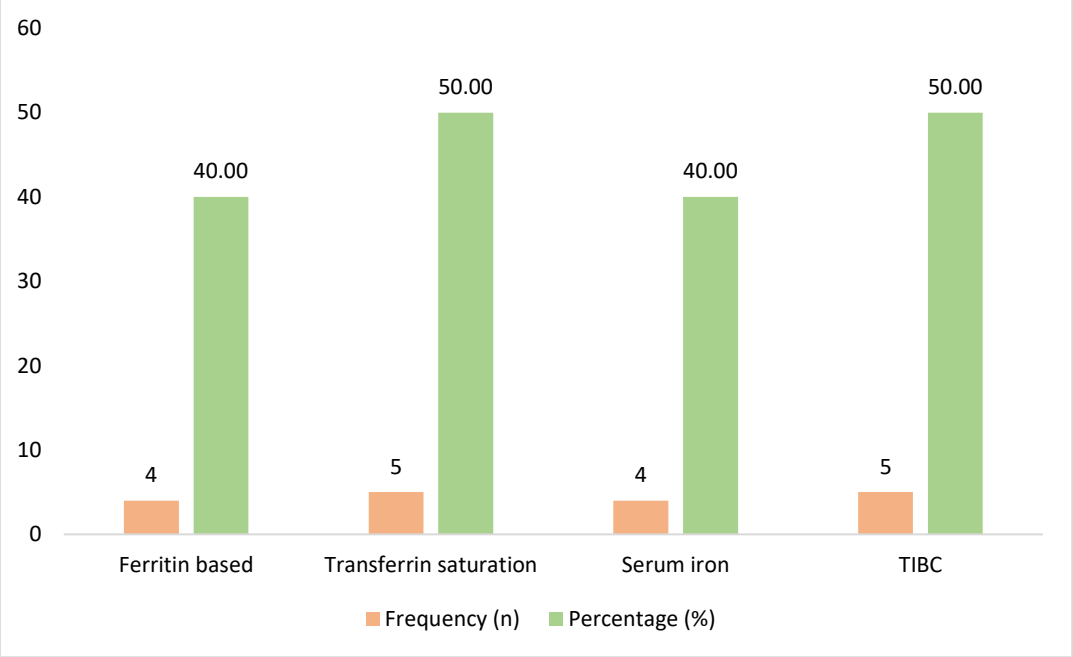

Figure 2 Iron deficiency based on blood test among CCHD children (*n*=10).

Transferrin saturation and TIBC identified iron deficiency in 50.0% children each, whereas serum ferritin and serum iron detected deficiency in 40.0% participants each (*Figure 2*).

Low serum iron was present in 90.0% Group A participants and 10.0% Group B participant (p<0.001). Similarly, low TIBC (<15) was observed in (80.0%) of Group A and (20.0%) of Group B participants (p<0.001) (*Table II*).

Table II
Showing comparison of serum ferritin with other definitive tests (*n*=20)

Variables	Group A		Group B		P-value
	n	%	n	%	
Serum iron					
Low	9	90.00	1	10.00	<0.001
Normal	1	10.00	9	90.00	
TIBC					
Low (<15)	8	80.00	2	20.00	<0.001
Normal (≥ 15)	2	20.00	8	80.00	

Low hemoglobin (<15) was found in (90.0%) Group A and (10.0%) Group B participants (p<0.001). Low PCV (<60%) occurred in (90.0%) and (20.0%) participants, respectively (p<0.001). Low MCV (<80) and low MCH were present in (80.0%) of Group A and (10.0%) of Group B participants (p<0.001). Low MCHC was observed in (70.0%) Group A and (30.0%) Group B participants (p<0.001). Low RDW occurred in (90.0%) and (20.0%) participants, respectively (p<0.001). Microcytic hypochromic smear findings were observed in (80.0%) of Group A, whereas normocytic normochromic findings predominated in Group B (90.0%) (p<0.001) *Table III*.

Table III
Showing comparison of serum ferritin with simple tests (*n*=20).

Variables	Group A		Group B		P-value
	n	%	n	%	
Hemoglobin					
Low Hb (<15)	9	90.00	1	10.00	<0.001
Normal (>15)	1	10.00	9	90.00	<0.001
PCV					
Low PCV (<60%)	9	90.00	2	20.00	<0.001
Normal PCV (≥60%)	1	10.00	8	80.00	<0.001
MCV					
Low MCV (<80)	8	80.00	1	10.00	<0.001
Normal MCV (>80)	2	20.00	9	90.00	<0.001
MCH					
Low MCH	8	80.00	1	10.00	<0.001
Normal MCH	2	20.00	9	90.00	<0.001
MCHC					
Low MCHC	7	70.00	3	30.00	<0.001
Normal MCHC	3	30.00	7	70.00	<0.001
RDW					
Low RDW	9	90.00	2	20.00	<0.001
Normal RDW	1	10.00	8	80.00	<0.001
Peripheral smear					
Microcytic hypochromic	8	80.00	1	10.00	<0.001
Normocytic normochromic	2	20.00	9	90.00	<0.001

Table IV shows logistic regression analysis, where only RDW was a significant predictor (B=2.121, SE=1.054, p=0.04). Hemoglobin (p=0.9), PCV (p=0.9), MCV (p=0.2), MCH (p=0.5), MCHC (p=0.2), and peripheral smear (p=0.2) were not statistically significant.

Table IV
Logistic regression analysis of the blood tests.

Variables	B	Standard error	P-value
Hemoglobin	19.874	15751.235	0.9
PCV	-18.743	15751.235	0.9
MCV	1.475	1.353	0.2
MCH	-1.231	1.332	0.5
MCHC	1.554	1.128	0.2
RDW	2.121	1.054	0.04
Peripheral smear	1.521	1.278	0.2

DISCUSSION

Cyanotic congenital heart diseases (CCHDs) are a group of structural heart defects that result in low oxygen levels in the blood, leading to chronic hypoxemia. This condition often triggers increased erythropoiesis, which in turn raises the body's iron demand. Iron deficiency in these patients can worsen symptoms and impact overall clinical outcomes. Therefore, assessing iron status is crucial for the effective management of individuals with CCHDs. In this study, a significantly higher proportion in Group A were aged 1–5 years (40% vs. 20%, $p = 0.04$) and were male (70% vs. 50%, $p = 0.03$). This findings align with the result of Bandyopadhyay et al [18]. Undernutrition was prevalent in both groups, with 60% of Group A severely underweight, compared to 50% in Group B ($p = 0.05$), and similar patterns in stunting though no statistical differences there. This finding was anticipated, as heart disease is known to negatively impact children's growth, overall health, and nutritional intake. Similar observations were reported by Lang'O et al [19]. In our study, erythrocytosis was observed in 40% of cases but showed no statistically significant association with iron deficiency (p=0.4), aligning with the findings of Onur et al [7]. However, a contrasting result was reported by Olcay et al., where erythrocytosis was significantly associated with iron deficiency [20]. Our cohort of patients with CCHD displayed symptoms such as dyspnoea (8/40%), cyanosis (5/25%), cyanotic spells (3/15%), recurrent lower respiratory infections (1/5%), palpitations/syncope (1/5%), and asymptomatic cases (2/10%). These findings highlight that while breathlessness and cyanosis predominate, a significant proportion present with varied or subtle clinical manifestations. This findings are consistent with the findings of Bandyopadhyay et al [18]. Additionally, functional consequences—growth delay and more frequent cyanotic spells—were noted with iron deficiency in previous reports [21]. According to our study, iron deficiency was detected in 40% of patients using ferritin levels, 50% using transferrin saturation (TSAT), 40% using serum iron, and 50% using total iron-binding capacity (TIBC). This variability reflects differing sensitivity and specificity of each marker in detecting iron deficiency in CCHD. We found that Biochemical markers—serum iron and TIBC—showed strong concordance with ferritin (90% low iron, 80% low TIBC in iron-deficient); all p-values were highly significant ($p < 0.001$). In our study, routine hematological indices—including hemoglobin (Hb), packed cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW), and peripheral blood smear findings—proved to be valuable in assessing iron status among children with cyanotic congenital heart disease (CCHD). We observed that 70% to 90% of children identified as iron deficient based on biochemical parameters exhibited classic hematological features of iron deficiency anemia. These included microcytic (small-sized) and hypochromic (pale-colored) red

blood cells on smear, as well as elevated RDW, which indicates a wide variation in red blood cell size. This correlation was found to be statistically significant ($p < 0.001$). Interestingly, among all hematologic markers analyzed, only RDW emerged as a statistically significant independent predictor of iron deficiency in logistic regression analysis ($p = 0.04$). This highlights RDW's potential as a sensitive early marker, even before overt anemia is present. Our findings are consistent with those reported by Animasahun et al., who also emphasized RDW as a reliable tool for early detection of iron deficiency in children with CCHD [22]. RDW may rise early in the course of iron depletion, preceding measurable drops in hemoglobin or hematocrit levels. Biochemical trends mirror broader studies: a large cohort noted significantly lower serum iron (~45 µg/dL), ferritin (~18 ng/mL), and elevated TIBC in cyanotic vs. acyanotic CHD, with microcytic indices and high RDW in iron-deficient patients [21].

LIMITATIONS

- The absence of a comparison group limits the ability to assess how iron deficiency prevalence in CCHD compares to that in acyanotic CHD or the general pediatric population.
- Inflammatory markers such as C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR) were not measured.
- The study did not assess long-term dietary habits, micronutrient supplementation, or socioeconomic status, all of which significantly impact iron intake and bioavailability.

CONCLUSION & RECOMMENDATIONS

Iron deficiency remains a common and clinically significant issue among children with cyanotic congenital heart disease. Despite elevated hemoglobin levels due to secondary erythrocytosis, many of these patients exhibit depleted iron stores, predisposing them to increased risks of morbidity. Biochemical markers such as serum ferritin and transferrin saturation remain gold standards for diagnosis. However, routine hematological parameters, particularly red cell distribution width (RDW), showed a strong correlation and can serve as valuable, cost-effective screening tools in resource-limited settings. Future studies with larger cohorts and longitudinal follow-up are recommended to better understand the clinical consequences and to guide therapeutic strategies.

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CONFLICT OF INTEREST

None declared

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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