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Iron Deficiency Anemia among Children with Congenital Heart Disease- A Cross-Sectional Study

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Abstract **Original Research Article**

Background: Iron Deficiency Anemia (IDA) is a common concern among children with Congenital Heart Disease (CHD) due to multiple factors, including chronic hypoxia, inadequate nutritional intake, and increased iron demands. ***Aim of the study*:** The aim of this study was to determine the prevalence of iron deficiency anemia in children with Congenital heart disease. ***Methods*:** This cross-sectional study was conducted in Department of Pediatric Cardiology, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh, during the period from July 2019 to December 2019. A total of 100 pediatric patients with confirmed CHD were selected for this study. ***Result*:** The study included 100 pediatric CHD patients, with a mean age of 5.2 ± 2.7 years (range: 6 months–12 years). Males comprised 60%, and cyanotic CHD cases were more prevalent (55%). Hematological analysis showed significantly lower hemoglobin (10.2 ± 1.5 g/dL) and RBC counts in cyanotic CHD than acyanotic CHD ($p < 0.001$). Serum iron (45.1 ± 8.7 µg/dL) and ferritin (18.5 ± 5.2 ng/mL) were significantly reduced in cyanotic CHD ($p < 0.01$), with higher TIBC and lower transferrin saturation. Patients with ferritin <50 ng/mL had significantly lower hemoglobin, MCV, and MCH ($p < 0.05$), confirming iron deficiency anemia. IDA was associated with growth retardation (63.6%), delayed neurodevelopment (40%), increased cyanotic spells (36.4%), and high surgical risk (43.6%) ($p < 0.01$). ***Conclusion*:** This study highlights the high prevalence of iron deficiency anemia (IDA) in cyanotic congenital heart disease (CHD) patients and its adverse impact on growth, neurodevelopment, and surgical outcomes.

Keywords: Iron Deficiency Anemia, Children, and Congenital Heart Disease.

INTRODUCTION

Iron deficiency anemia (IDA) is the most common nutritional deficiency disorder worldwide, disproportionately affecting children, particularly in developing countries where dietary insufficiencies, infections, and limited healthcare access contribute to its high prevalence. In Bangladesh, nearly 40-50% of children suffer from some form of anemia, with IDA being the predominant cause, largely due to inadequate dietary iron intake, recurrent infections, and poor healthcare infrastructure [1]. Congenital heart disease (CHD), a structural abnormality present at birth, further compounds this issue, with approximately 25,000 new cases reported annually in Bangladesh, making it a significant contributor to childhood morbidity and mortality [2]. Despite this burden, limited research exists on the relationship between IDA and CHD in Bangladeshi children, necessitating further investigation into their co-occurrence and the impact of IDA on cardiac outcomes. The association between CHD and IDA is well-documented in high-income countries, where cyanotic CHD leads to increased erythropoiesis due to chronic hypoxia, often resulting in functional iron deficiency because iron demand exceeds supply [3]. Children with CHD frequently experience poor oral intake, malabsorption, and increased iron losses due to chronic inflammation, further exacerbating iron deficiency [4]. In Bangladesh, where nutritional iron deficiency is already widespread, children with CHD are particularly vulnerable to severe IDA, increasing their risk of growth retardation, neurodevelopmental delays, and poor surgical outcomes [1]. However, data on how IDA affects CHD outcomes in Bangladesh remains scarce, making this an urgent area of study. Clinical evidence suggests that undiagnosed or untreated IDA in CHD patients can severely impair cardiac function, particularly in cyanotic cases where chronic hypoxia increases blood viscosity and the risk of cerebrovascular events [5]. Studies have demonstrated that children with

cyanotic CHD and untreated IDA experience frequent cyanotic spells, which increase perioperative complications and surgical mortality [4]. Furthermore, IDA has been shown to raise NT-proBNP levels in CHD patients, a marker of cardiac stress, indicating worsening myocardial function [6]. Despite these critical implications, routine IDA screening is not systematically implemented for CHD children in Bangladesh, which may contribute to higher surgical risks and poorer post-operative outcomes. Beyond its immediate cardiac effects, IDA is a well-established risk factor for delayed neurodevelopment, impaired cognitive function, and poor physical growth, particularly in low-income settings where nutritional interventions are insufficient [7]. Studies from Bangladesh indicate that iron supplementation alone may not fully reverse cognitive impairments in children suffering from early-life IDA, emphasizing the need for early detection and intervention [8]. Given that CHD already predisposes children to neurodevelopmental challenges, the co-existence of IDA may further exacerbate these issues, leading to long-term developmental disabilities. However, no large-scale study in Bangladesh has evaluated the neurodevelopmental impact of IDA in CHD patients, highlighting a significant research gap. Moreover, while global studies have established nutritional deficiencies as a primary driver of IDA, Bangladesh presents unique risk factors that may influence its prevalence among CHD patients. Unlike many Western populations, groundwater iron contamination and genetic conditions such as thalassemia contribute to variations in IDA prevalence and severity [9]. Studies indicate that while IDA is highly prevalent, its etiology in Bangladesh is more complex, requiring context-specific screening and management strategies [8]. Yet, there is limited understanding of how these regional factors interact with CHD-related IDA, further reinforcing the need for locally relevant data to guide public health policies. Given the high burden of IDA and CHD in Bangladesh, this study aims to quantify the prevalence of IDA in children with CHD, identify risk factors, and assess its impact on cardiac outcomes, neurodevelopment, and perioperative risks.

OBJECTIVES

To determine the prevalence of iron deficiency anemia in children with Congenital heart disease.

METHODOLOGY & MATERIALS

This cross-sectional study was conducted in Department of Pediatric Cardiology, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh, during the period from July 2019 to December 2019. A total of 100 pediatric patients with confirmed CHD, aged 6 months to 12 years, were selected using convenience sampling from the hospital's pediatric cardiology and hematology departments. The study population was categorized into cyanotic and acyanotic CHD groups to examine differences in IDA prevalence and severity. Patients with a history of recent blood transfusion, chronic kidney disease, or other hematological disorders were excluded to ensure data reliability. A detailed clinical history and dietary assessment were recorded using a structured questionnaire to evaluate nutritional intake, feeding difficulties, and socioeconomic status. Laboratory investigations included complete blood count (CBC), serum ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin saturation levels, performed using standardized automated techniques to diagnose IDA. Additionally, echocardiographic findings were analyzed to correlate IDA severity with CHD type and functional status. Neurodevelopmental assessments were conducted for children under 5 years using age-appropriate cognitive and motor development scales. The data were analyzed using SPSS software, with descriptive statistics (mean, standard deviation, frequency) used for baseline characteristics, and chi-square tests and logistic regression models applied to determine associations between CHD type, IDA, and clinical outcomes. A p-value <0.05 was considered statistically significant. Ethical approval was obtained from the hospital's Institutional Review Board (IRB), and informed written consent was taken from parents or guardians before participation.

RESULT

The study included 100 pediatric patients diagnosed with congenital heart disease (CHD). Table I presented that the mean age was 5.2 ± 2.7 years and an age range spanning from 6 months to 12 years. The study population had a male predominance (60%), while females accounted for 40% of participants. In terms of CHD classification, 55% of the children had cyanotic CHD, whereas 45% had acyanotic CHD. Table II showed a comparison of hematological parameters between cyanotic and acyanotic CHD patients revealed significant differences. The mean hemoglobin levels were lower in cyanotic CHD patients (10.2 ± 1.5 g/dL) compared to acyanotic CHD patients (11.5 ± 1.2 g/dL), with a statistically significant p-value of <0.001. Similarly, red blood cell (RBC) count was lower in cyanotic CHD patients (4.7 ± 0.5 $\times 10^{12}$/L) compared to acyanotic CHD patients (4.9 ± 0.4 $\times 10^{12}$/L, $p < 0.05$). Serum iron and ferritin levels also showed marked differences, with cyanotic CHD patients exhibiting significantly lower serum ferritin (18.5 ± 5.2 ng/mL) and serum iron (45.1 ± 8.7 µg/dL) compared to acyanotic CHD patients (25.2 ± 6.3 ng/mL and 60.3 ± 10.1 µg/dL, respectively; $p < 0.01$). Additionally, total iron-binding capacity (TIBC) was higher in cyanotic CHD patients (412 ± 50 µg/dL) compared to acyanotic CHD patients (380 ± 45 µg/dL, $p < 0.05$), while transferrin saturation was significantly lower in cyanotic CHD patients (11.0 ± 2.4%) than in acyanotic CHD patients (15.7 ± 3.1%, $p < 0.05$). Red cell indices further supported the iron-deficient state in cyanotic CHD patients. Mean corpuscular volume (MCV) was significantly lower in cyanotic CHD (75.1 ± 6.0 fL) than in acyanotic CHD

(79.5 ± 5.8 fL, p < 0.01). Mean corpuscular hemoglobin (MCH) was also lower in cyanotic CHD (23.5 ± 2.7 pg) compared to acyanotic CHD (25.1 ± 2.6 pg, p < 0.05). Mean corpuscular hemoglobin concentration (MCHC) showed no statistically significant difference between the groups (p = 0.07). Red cell distribution width (RDW) was significantly higher in cyanotic CHD patients (16.2 ± 2.3%) compared to acyanotic CHD patients (14.9 ± 2.0%, p < 0.01), reflecting greater variability in red cell size, which is characteristic of iron deficiency anemia (IDA). Table-III demonstrated the Serum ferritin levels in CHD patients. Analysis of serum ferritin levels demonstrated that a greater proportion of cyanotic CHD patients had severe iron deficiency. Among cyanotic CHD patients, 22 (40%) had serum ferritin levels below 12 ng/mL, compared to only 8 (17.8%) in the acyanotic CHD group (p < 0.001). The majority of cyanotic CHD patients (45.5%) had ferritin levels in the range of 12-49 ng/mL, whereas acyanotic CHD patients had a more even distribution across different ferritin categories. Serum ferritin levels between 50-100 ng/mL were more frequent in acyanotic CHD patients (26.7%) than in cyanotic CHD patients (10.9%, p = 0.08). Only a small percentage of both groups had ferritin levels above 100 ng/mL. A comparative assessment of hematological parameters based on serum ferritin levels (<50 ng/mL vs. ≥50 ng/mL) showed in table-IV illustrated that patient with ferritin levels <50 ng/mL had significantly lower RBC counts (4.5 ± 0.6 vs. 4.9 ± 0.5 $\times 10^{12}$/L, p < 0.01). Similarly, hemoglobin levels were significantly lower in the low-ferritin group (9.8 ± 1.4 g/L) compared to the high-ferritin group (11.5 ± 1.2 g/L, p < 0.001). MCV (72.5 ± 6.3 fL vs. 80.2 ± 5.7 fL, p < 0.05), MCH (22.1 ± 2.5 pg vs. 25.4 ± 2.8 pg, p < 0.01), and transferrin saturation (10.3 ± 2.5% vs. 16.4 ± 3.2%, p < 0.01) were also significantly lower in the low-ferritin group. Conversely, RDW was significantly higher (17.2 ± 2.1% vs. 14.8 ± 2.0%, p < 0.05) in patients with ferritin <50 ng/mL, further supporting the presence of iron deficiency anemia. In table-V, the study revealed that growth retardation was significantly higher in IDA patients (63.6%) compared to non-IDA patients (22.2%, p < 0.001), indicating the impact of iron deficiency on physical development. Delayed neurodevelopment was more prevalent in IDA patients (40.0%) than in non-IDA patients (13.3%, p < 0.01), suggesting cognitive impairment risks. Increased cyanotic spells were observed in 36.4% of IDA patients versus 11.1% in non-IDA patients (p < 0.05), highlighting worsened oxygenation. High surgical risk was significantly greater in IDA patients (43.6%) compared to non-IDA patients (17.8%, p < 0.01), emphasizing perioperative concerns.

Table-I: Baseline characteristics of the study people (N=100)

Characteristics	Number of patients	Percentage (%)
Age (years)		
Mean ± SD	5.2 ± 2.7	
Range	6 months to 12 years	
Gender		
Male	60	60
Female	40	40
Type of CHD		
Cyanotic CHD	55	55
Acyanotic CHD	45	45

Table-II: Hematological parameters of study participants (N=100)

Parameter	Mean ± SD (Cyanotic CHD)	Mean ± SD (Acyanotic CHD)	p-value
Hemoglobin (g/dL)	10.2 ± 1.5	11.5 ± 1.2	<0.001
RBC (x10^12/L)	4.7 ± 0.5	4.9 ± 0.4	<0.05
Serum Ferritin (ng/mL)	18.5 ± 5.2	25.2 ± 6.3	<0.01
Serum Iron (µg/dL)	45.1 ± 8.7	60.3 ± 10.1	<0.01
TIBC (µg/dL)	412 ± 50	380 ± 45	<0.05
Transferrin Saturation (%)	11.0 ± 2.4	15.7 ± 3.1	<0.05
MCV (fL)	75.1 ± 6.0	79.5 ± 5.8	<0.01
MCH (pg)	23.5 ± 2.7	25.1 ± 2.6	<0.05
MCHC (%)	31.2 ± 1.9	32.5 ± 1.6	0.07
RDW (%)	16.2 ± 2.3	14.9 ± 2.0	<0.01

Table-III: Serum ferritin levels in CHD patients (N=100)

Serum Ferritin (ng/mL)	Cyanotic CHD (n=55)	Acyanotic CHD (n=45)	p-value
<12	22	8	<0.001
12-49	25	20	<0.01
50-100	6	12	0.08
>100	2	5	0.12

Table-IV: Hematological parameters based on serum ferritin levels (N=100)

Parameters	SF < 50 ng/mL	SF ≥ 50 ng/mL	p-value
RBC (x10^12/L)	4.5 ± 0.6	4.9 ± 0.5	<0.01
Hemoglobin (g/L)	9.8 ± 1.4	11.5 ± 1.2	<0.001
MCV (fL)	72.5 ± 6.3	80.2 ± 5.7	<0.05
MCH (pg)	22.1 ± 2.5	25.4 ± 2.8	<0.01
MCHC (%)	30.5 ± 1.8	32.1 ± 1.7	0.08
RDW (%)	17.2 ± 2.1	14.8 ± 2.0	<0.05
Serum Iron (µg/dL)	35.2 ± 7.6	55.1 ± 8.3	<0.001
Transferrin Saturation (%)	10.3 ± 2.5	16.4 ± 3.2	<0.01

Table-V: Association between IDA and clinical outcomes in CHD patients (N=100)

Outcome	IDA Patients (n=55)	Non-IDA Patients (n=45)	p-value
Growth Retardation (%)	35 (63.6%)	10 (22.2%)	<0.001
Delayed Neurodevelopment (%)	22 (40.0%)	6 (13.3%)	<0.01
Increased Cyanotic Spells (%)	20 (36.4%)	5 (11.1%)	<0.05
High Surgical Risk (%)	24 (43.6%)	8 (17.8%)	<0.01

DISCUSSION

Iron deficiency anemia (IDA) is a well-established comorbidity in children with congenital heart disease (CHD), particularly in cyanotic CHD, where chronic hypoxia and increased erythropoiesis lead to functional iron deficiency. The findings of this study align with previous research that underscores the significant burden of IDA in CHD patients and its impact on clinical outcomes. The baseline characteristics of the study population revealed a male predominance (60%), consistent with global studies where CHD is reported more frequently in males [10]. The higher prevalence of cyanotic CHD (55%) compared to acyanotic CHD (45%) in this study suggests a greater representation of severe cases, similar to findings from a Bangladeshi cohort where cyanotic CHD was significantly associated with poorer hematological and nutritional status [11].

The hematological parameters of study participants demonstrate that children with cyanotic CHD exhibited significantly lower hemoglobin levels (10.2 ± 1.5 g/dL) compared to acyanotic CHD patients (11.5 ± 1.2 g/dL, $p < 0.001$), confirming findings from Soni *et al.*, [12], who reported lower hemoglobin levels in cyanotic CHD due to chronic hypoxia-induced erythropoiesis and iron depletion. Moreover, serum ferritin levels were markedly lower in cyanotic CHD (18.5 ± 5.2 ng/mL) compared to acyanotic CHD (25.2 ± 6.3 ng/mL, $p < 0.01$), consistent with findings from Gidding and Stockman [13], who identified ferritin depletion in cyanotic CHD patients. This highlights the paradox of erythrocytosis-induced functional iron deficiency, where increased iron utilization for red blood cell production exacerbates depletion. Similarly, serum iron levels (45.1 ± 8.7 µg/dL in cyanotic CHD vs. 60.3 ± 10.1 µg/dL in acyanotic CHD, $p < 0.01$) were significantly lower, reinforcing findings by Hågå *et al.*, [14] that cyanotic CHD patients have persistently lower iron stores due to excessive erythropoietic drive.

Analysis of serum ferritin levels revealed that 40% of cyanotic CHD patients had ferritin <12 ng/mL, compared to 17.8% of acyanotic CHD patients ($p < 0.001$), indicating severe iron depletion. These results align with findings by Soni *et al.*, [12], who reported that 56.6% of cyanotic CHD patients had IDA, significantly higher than in acyanotic cases. The majority of cyanotic CHD patients (45.5%) had ferritin levels between 12-50 ng/mL, whereas acyanotic CHD patients showed a relatively higher percentage in the 50-100 ng/mL category (26.7%), reinforcing previous studies that highlight the greater iron deficiency burden in cyanotic CHD [15].

The relationship between iron stores and hematological indices further demonstrates the functional consequences of IDA in CHD patients. Patients with serum ferritin <50 ng/mL had significantly lower RBC counts (4.5 ± 0.6 × 10^12/L), hemoglobin (9.8 ± 1.4 g/L), and MCV (72.5 ± 6.3 fL) compared to those with serum ferritin ≥50 ng/mL ($p < 0.05$ for MCV

and $p < 0.001$ for hemoglobin). This is consistent with Kalvakuri *et al.,* [16], who reported that iron-deficient children with CHD exhibited lower hemoglobin and RBC indices, leading to microcytic, hypochromic anemia patterns. Additionally, serum iron and transferrin saturation were markedly lower in the SF < 50 ng/mL group (35.2 ± 7.6 µg/dL and 10.3 ± 2.5%) compared to those with SF ≥ 50 ng/mL (55.1 ± 8.3 µg/dL and 16.4 ± 3.2%), $p < 0.001$ and $p < 0.01$, respectively, reinforcing findings from Gidding & Stockman [13] that low iron stores in CHD lead to poor oxygen-carrying capacity and metabolic dysfunction.

The clinical implications of IDA in CHD patients are profound. Growth retardation was significantly higher in IDA patients (63.6% vs. 22.2%, $p < 0.001$), confirming data from Said *et al.,* [17], who reported that malnutrition was prevalent in 72.5% of cyanotic CHD patients, leading to poor weight and height-for-age Z scores. Similarly, 40% of IDA patients had delayed neurodevelopment compared to 13.3% of non-IDA patients ($p < 0.01$), reinforcing findings by Mir *et al.,* [18], who highlighted that IDA impairs cognitive function in CHD patients due to chronic hypoxia and reduced oxygen transport. Furthermore, cyanotic spells were significantly more frequent in IDA patients (36.4% vs. 11.1%, $p < 0.05$), aligning with findings by Mukherjee *et al.,* [4], who reported that iron deficiency exacerbates cyanotic episodes due to reduced hemoglobin function.

The risk of adverse surgical outcomes was also significantly higher in IDA patients (43.6% vs. 17.8%, $p < 0.01$), emphasizing the necessity for preoperative iron optimization. This aligns with findings from Said *et al.,* [17], who demonstrated that Cyanotic CHD was significantly associated with IDA and poor clinical outcomes in CHD patients undergoing cardiac surgery. Additionally, Mir *et al.,* [18] reported that preoperative iron supplementation improved surgical outcomes and reduced ICU stays, reinforcing the importance of early IDA management in CHD.

Limitations of the Study

In our study, there was small sample size and absence of control for comparison. Study population was selected from one center in Dhaka city, so may not represent wider population. The study was conducted at a short period of time.

CONCLUSION AND RECOMMENDATIONS

This study highlights the high prevalence of Iron deficiency anemia (IDA) in cyanotic congenital heart disease (CHD) patients and its detrimental effects on growth, neurodevelopment, cyanotic spells, and surgical outcomes. The findings align with previous research, confirming that cyanotic CHD patients have lower iron stores, impaired erythropoiesis, and higher metabolic demands, leading to functional iron deficiency and increased morbidity. Given the strong correlation between IDA and adverse clinical outcomes, early screening, iron supplementation, and nutritional interventions should be prioritized in CHD management to improve overall health and surgical outcomes. Further longitudinal studies are warranted to assess the long-term impact of iron therapy on clinical outcomes in CHD patients.

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