Serum Iron Profile in Children with Congenital Heart Disease Attending a Tertiary Care Hospital

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ARTICLE INFO

Received: 21 Feb 2026
Accepted: 26 Feb 2026
Published Online: 27 Feb 2026

DOI: 10.5281/zenodo.18806251

Volume: 9, Number: 1, Page: 2-6

e-ISSN: 2789-5912
ISSN: 2617-0817

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ABSTRACT

Background: Congenital heart disease is the most common congenital anomaly and has been associated frequently with malnutrition and hematologic complications, particularly iron deficiency and IDA. This study was done to assess the serum iron profile in children with CHD attending a tertiary care hospital. **Methods & Materials**: This cross-sectional study was conducted from July 2023 to June 2024 among 91 children aged 1 month to 12 years with echocardiographically confirmed CHD at the Institute of Child and Mother Health, Dhaka. Serum iron, ferritin, and total iron-binding capacity were measured, and iron deficiency and IDA were defined using the standard age-specific ferritin and haemoglobin criteria. Data analysis was done by using SPSS version 26, and a p-value less than 0.05 was considered statistically significant. **Results:** The mean age of participants was 12.7 months; 51% were male. Acyanotic CHD accounted for 78% of cases, with ventricular septal defect being the most common lesion (39.56%). Malnutrition was highly prevalent, with 51.6% of children wasted and 39.6% stunted. The mean serum iron, ferritin, and TIBC were 57.59 ± 34.49 µg/dl, 125.25 ± 141.34 ng/ml, and 322.15 ± 71.85 µg/dl, respectively. Iron deficiency was present in 53.5% of acyanotic and 65% of cyanotic CHD cases. Ferritin deficiency was also higher in cyanotic CHD (70%) compared to acyanotic CHD (56.3%). **Conclusion:** This study demonstrates a high prevalence of iron deficiency in children with congenital heart disease, as evidenced by low serum iron and ferritin levels in the majority of children and increased TIBC in many. While there was a trend towards a higher rate of deficiency in the cyanotic as compared with the acyanotic patients, this did not reach statistical significance.

Keywords: *Congenital Heart Disease (CHD), Iron Deficiency Anaemia, Serum Iron Profile.*

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INTRODUCTION

Congenital heart disease is the most common congenital anomaly in children and continues to be a major cause of morbidity and mortality among children in many parts of the world. Despite advances in diagnosis and treatment, it still remains a major global health concern and requires more emphasis on early detection and management strategies. It has been estimated that CHD accounts for a considerable proportion of deaths in children and so calls for timely intervention and improved supportive care [1,2]. CHD encompasses a wide range of structural cardiac malformations that are conventionally classified as either cyanotic or acyanotic. The most common acyanotic lesions include Ventricular Septal Defect (VSD), Atrial Septal Defect (ASD), and Patent Ductus Arteriosus (PDA), while Tetralogy of Fallot (TOF) is the most common cyanotic lesion [3,4]. Because of the chronic hypoxia, abnormal haemodynamics, and increased metabolic stress due to these conditions, the patients are at a significantly increased risk of nutritional deficiencies, with an especial predisposition to iron deficiency anaemia. Iron deficiency anaemia is one of the most common and yet under-recognized comorbidities among children suffering from CHD. Iron is an important mineral involved in oxygen transport, cellular respiration, and energy metabolism. Its deficiency can further aggravate tissue hypoxia, diminish exercise tolerance, and adversely impact overall cardiac performance. Both cyanotic and acyanotic CHD children are prone to diminished iron stores due to poor dietary intake, chronic illness, and increased erythropoietic demands [5,7]. The compensatory erythropoietin response to persistent hypoxia seen in cyanotic CHD promotes increased production of red blood cells and thus gradually depletes the iron reserves over time, often before the onset of clinically evident anaemia [6]. Many children may have a polycythaemic appearance, which masks the underlying iron deficiency and delays its diagnosis [8]. Assessment of iron status in CHD requires evaluation of serum iron profile, which consists of serum iron, serum ferritin, total iron-binding capacity (TIBC), and transferrin saturation. These biochemical parameters provide a more accurate reflection of iron stores and utilisation compared to red cell counts alone [9]. Red cell indices, including Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH), and Red Cell Distribution Width (RDW), are also useful early indicators of iron depletion and may help in diagnosing subclinical deficiency [8,10]. Iron deficiency in children with CHD has significant clinical implications. Low iron levels have been associated with increased hospitalisation, fatigue, and poor clinical outcomes in paediatric cardiac patients.[11] Frequent cyanotic spells and delayed recovery are more likely in children with cyanotic CHD and iron deficiency [12]. Similarly, preoperative anaemia is related to

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higher perioperative risk with a prolonged postoperative recovery [5]. Accordingly, identification and correction of iron deficiency are important for improving growth, neurodevelopmental status, and overall clinical outcomes in CHD children. Despite these observations, iron deficiency usually goes undiagnosed in children due to nonspecific clinical presentations and reliance on incomplete screening methods [6,8]. Routine testing of the serum iron profile can thus become instrumental in the early identification and intervention of iron deficiency anaemia in this population. Early recognition and timely management improve cardiac efficiency as well as overall physical and developmental outcomes. The current study proposes assessing the serum iron profile in children with CHD presenting to a tertiary care hospital.

METHODS & MATERIALS

This cross-sectional study was conducted among 91 children with CHD attending the Outpatient and Inpatient Departments of Paediatrics at ICMH, Dhaka, Bangladesh, from July 2023 to June 2024. Participants were selected through purposive sampling. Children aged 1 month to 12 years with echocardiographically confirmed CHD were included. Written informed consent was taken from their legal guardians before participation. Children with hemolytic disorders, previous cardiac surgery, recent iron supplementation, or systemic illnesses resulting in anemia were excluded. Data collection was done through a pre-tested semistructured questionnaire by interviewing the caregivers, physical examination, and review of medical records of the children. Information included demographic details, type of CHD, nutritional status, anemia, and feeding practices. Laboratory investigations included complete blood count and serum iron profile (iron, ferritin, and total iron-binding capacity). Iron deficiency was defined as serum ferritin <12 ng/mL (<5 years), <15 ng/mL (≥5 years), or <30 ng/mL with infection; iron deficiency anemia as Hb <11 g/dL with corresponding biochemical findings. Data were analyzed using SPSS version 26 and $p < 0.05$ was considered statistically significant. Ethical approval was obtained from the ICMH Ethical Review Committee.

RESULTS

Table I shows that the participants were categorized into three age groups: 1–6 months (32.97%), 7–12 months (30.77%), and >12 months (36.26%). The mean age of the population was 12.7 months, ranging from 1 to 32 months.

Table I
Age distribution of the study population (*n* = 91).

Age in months	Number	Percentage (%)
1–6	30	32.97
7–12	28	30.77
>12	33	36.26
Total	91	100
Mean	12.7	1–32 months

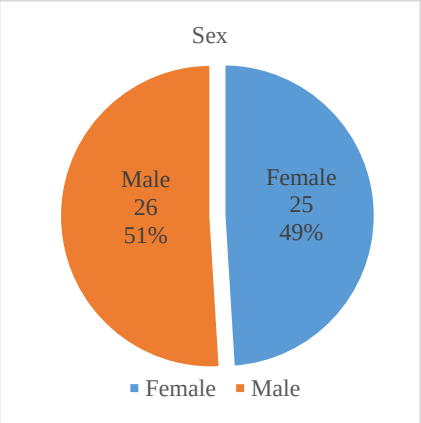

Figure 1 Sex distribution of the study population (*n*=91).

Figure 1 shows that 51% of the study population is male and 49% female child.

Table II summarizes the clinical characteristics of the study population. Among the participants, 39 had anemia, 18 had cyanosis, and 17 had heart failure. The mean height/length was 72.96 ± 13.13 cm, and mean weight was 8.98 ± 2.60 kg. Nutritional assessment showed mean WAZ, HAZ, and WHZ scores of -2.19 ± 1.48, -1.52 ± 1.04, and -1.69 ± 1.73, respectively, with 47 children wasted and 36 stunted. Features of acute illness were present in 27 children. Exclusive breastfeeding was reported in 48 children, and 33 received iron-containing appropriate complementary feeding.

Table II
Clinical characteristics of the study population (n = 91).

Clinical Feature	Number / Value
Anemia	39
Cyanosis	18
Heart Failure	17
Mean Height/Length (cm)	72.96 ± 13.13
Mean Weight (kg)	8.98 ± 2.60
Mean WAZ score	-2.19 ± 1.48
Mean HAZ score	-1.52 ± 1.04
Mean WHZ score	-1.69 ± 1.73
Child Wasted	47
Child Stunted	36
Features of acute illness (fever, cough, respiratory distress)	27
EBF (Exclusive Breastfeeding)	48
Iron-containing Appropriate Complementary Feeding	33

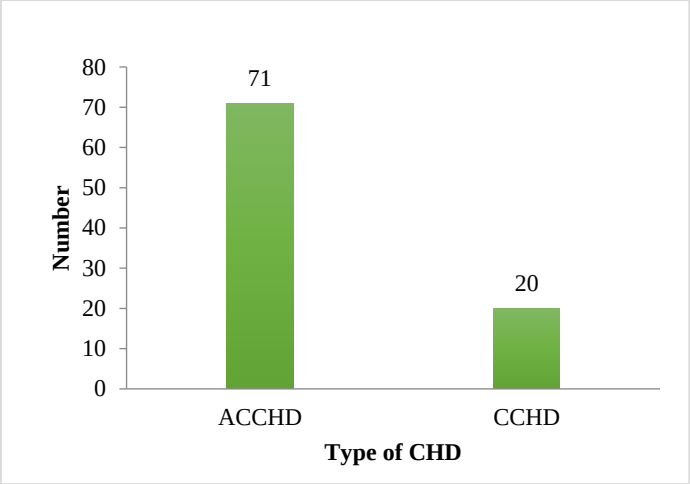

Figure 2 Types of CHD of the study population (*n* = 91).

Figure 2 shows that the number of ACHD is 71 and the number of CCHD is 20, respectively. *Table III* shows the types of congenital heart disease in the study population. VSD was the most common (36; 39.56%), followed by ASD (21; 23.07%) and TOF (13; 14.28%). Other types included PDA (7; 7.69%), VSD & ASD (5; 5.49%), VSD & PS (3; 3.35%), PDA & ASD (2; 2.19%), PAPVC (2; 2.19%), Truncus Arteriosus (1; 1.09%), and TGA (1; 1.09%).

Table III
Specific types of CHD of the study population (n = 91).

Specific Type of CHD	Number	Percentage (%)
VSD	36	39.56
ASD	21	23.07
TOF	13	14.28
PDA	7	7.69
VSD & ASD	5	5.49
VSD & PS	3	3.35
PDA & ASD	2	2.19
PAPVC	2	2.19
Truncus Arteriosus	1	1.09
TGA	1	1.09

Table IV shows the mean and range of iron parameters in the study population. Serum iron was 57.59 ± 34.49 µg/dl (range: 10–180), serum ferritin was 125.25 ± 141.34 ng/ml (range: 12–509), and TIBC was 322.15 ± 71.85 µg/dl (range: 180–528).

Table IV
Mean serum Iron, serum ferritin and TIBC (*n* = 91).

Parameter	Mean ± SD	Range (min–max)
Serum Iron	57.59 ± 34.49 µg/dl	10–180
Serum Ferritin	125.25 ± 141.34 ng/ml	12–509
TIBC	322.15 ± 71.85 µg/dl	180–528

Table V shows that among acyanotic CHD patients, 33 (46.5%) had normal serum iron and 38 (53.5%) had iron deficiency. Among cyanotic CHD patients, 7 (35.0%) had normal serum iron and 13 (65.0%) had iron deficiency, with a total of 91 patients and a p value of 0.36.

Table V
Serum Iron level among congenital heart disease patients (*n* = 91).

Serum Iron Status (µg/dl)	Acyanotic CHD n (%)	Cyanotic CHD n (%)	Total	p value
Normal	33 (46.5)	7 (35.0)	40	0.36
Deficiency	38 (53.5)	13 (65.0)	51	
Total	71 (100)	20 (100)	91	-

Table VI demonstrates that normal serum ferritin level for acyanotic congenital heart disease is 31 (43.7%) and 06 (30.0%) for cyanotic group. Deficiency of serum ferritin for acyanotic congenital heart disease is 40 (56.3%) and 14 (70.0%) for the cyanotic group among 54 congenital heart disease patients.

Table VI
Serum ferritin level among congenital heart disease patients (*n* = 91).

Serum Ferritin (ng/ml)	Acyanotic CHD n (%)	Cyanotic CHD n (%)	Total	p value
Normal	31 (43.7)	6 (30.0)	37	0.27
Deficiency	40 (56.3)	14 (70.0)	54	
Total	71 (100)	20 (100)	91	-

Table VII shows that normal TIBC levels were present in 40.8% of acyanotic and 40% of cyanotic CHD cases. High TIBC was the most common finding in both groups—52.1% in acyanotic and 50% in cyanotic patients. Low TIBC was seen in a small proportion of children (7.1% vs. 10%). Overall, the distribution of TIBC levels was similar between the two groups, with no significant difference (p = 0.9).

Table VII
Serum TIBC level among congenital heart disease patients (*n* = 91).

Serum TIBC (µg/dl)	Acyanotic CHD n (%)	Cyanotic CHD n (%)	Total	p value
Normal	29 (40.8)	8 (40.0)	37	
High	37 (52.1)	10 (50.0)	47	0.9
Low	5 (7.1)	2 (10.0)	7	
Total	71 (100)	20 (100)	91	-

DISCUSSION

The mean age of children with congenital heart disease (CHD) in this study was 12.7 months, ranging from 1 to 32 months. About one-third were aged 1–6 months, 30.77% were 7–12 months, and 36.26% were older than 12 months. Previous studies on children with cyanotic CHD reported a mean age of around 8 months, with most under 12 months [12]. Compared to these findings, the present cohort was older and had a higher proportion of children above 12 months, suggesting possible delayed referral or late presentation, which could be related to regional healthcare access, awareness, or early detection challenges. The sex distribution was nearly equal, with 51% male and 49% female, which is consistent with reports showing minimal sex bias in CHD prevalence [13]. Clinically, 42.9% of children had anaemia, 19.8% had cyanosis, and 18.7% had heart failure. The mean height/length was 72.96 ± 13.13 cm, and the mean weight was 8.98 ± 2.60 kg. Nutritional assessment revealed mean WAZ –2.19 ± 1.48, HAZ –1.52 ± 1.04, and WHZ –1.69 ± 1.73, with 51.6% of children wasted and 39.6% stunted. A meta-analysis of children with CHD reported pooled prevalences of wasting (~24.8%) and stunting (~24.4%) [2]. The higher rates observed in this cohort indicate a substantial nutritional burden. It may be influenced by socioeconomic factors, delayed intervention, increased metabolic demands or more severe cardiac lesions. Features of acute illness were present in a significant proportion of children, and exclusive breastfeeding was reported in less than half of the participants, with only a minority receiving iron-containing complementary feeding. These are highlighted here that suboptimal nutritional practices, which could contribute to growth retardation, poor immunity and hematologic deficits, emphasising the need for focused nutritional interventions in pediatric CHD care. Acyanotic lesions predominated, while cyanotic lesions accounted for a smaller proportion. Ventricular septal defect (VSD) was the most common lesion (39.56%). It is followed by atrial septal defect (ASD, 23.07%) and tetralogy of Fallot (TOF, 14.28%). On the other hand, global analyses also report that acyanotic lesions predominate, with VSD and ASD being the most frequent [14]. This distribution is consistent with the worldwide epidemiology of CHD, emphasising that isolated septal defects remain the most prevalent congenital cardiac abnormalities, which may have implications for early surgical intervention and iron deficiency risk. Biochemical assessment revealed a mean serum iron of 57.59 ± 34.49 µg/dl, ferritin 125.25 ± 141.34 ng/ml, and TIBC 322.15 ± 71.85 µg/dl. Lower mean serum iron and ferritin in children with CHD were reported in other studies [15]. Serum iron deficiency was observed in over half of children with acyanotic lesions and nearly two-thirds of those with cyanotic lesions, reflecting the higher prevalence of iron deficiency in cyanotic CHD [16]. Ferritin deficiency was also substantial, with 56.3% in acyanotic and 70% in cyanotic lesions, suggesting marked depletion of iron stores. Elevated TIBC was observed in roughly half of the children in both lesion types, reflecting a compensatory response to iron deficiency [17]. These results indicate that children with congenital heart disease attending a tertiary care hospital exhibit significant alterations in their serum iron profile.

LIMITATIONS

The study was limited by its purposive sampling, small sample size, absence of a control group, and lack of inflammatory marker assessment, which may affect accurate interpretation of iron status.

CONCLUSION

In conclusion, this study shows that children with congenital heart disease have a high prevalence of iron deficiency, with more than half exhibiting low serum iron and ferritin levels and many showing elevated TIBC. Although cyanotic patients had slightly higher rates of deficiency than acyanotic patients, the differences were not statistically significant.

RECOMMENDATIONS

Future research might involve larger, multicenter studies with control groups and include assessment of inflammatory markers alongside iron parameters. Studies could also evaluate long-term effects of iron supplementation on growth and clinical outcomes in children with CHD.

FUNDING

No funding sources

CONFLICT OF INTEREST

None declared

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