Original Article

Estimation of Serum Iron Status in Patients with Cyanotic Congenital Heart Disease – A Cross-Sectional Study

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ABSTRACT

Background: Cyanotic congenital heart disease (CCHD) is associated with chronic hypoxia, leading to compensatory erythrocytosis and paradoxically elevated hematological indices, which can mask underlying iron deficiency anemia (IDA). **Objective:** To assess the serum iron levels and prevalence of IDA in patients with CCHD. **Methods:** This cross-sectional study was conducted from July 1, 2021, to December 31, 2022, at All India Institute of Medical Sciences (AIIMS) Rishikesh for 18 months, enrolling 127 children aged 6 months to 18 years with unrepaired CCHD. Serum iron, ferritin, total blood count, and erythrocyte indices were assessed. Nutritional assessment, anthropometric measures, and socioeconomic data were collected. Different erythrocytic indices were compared between iron-deficient and non-iron-deficient groups. **Results:** A total of 127 children, with a mean age of 5.6±4.7 years, were included in the study. The most frequent CCHD was Tetralogy of Fallot (55.1%). Based on serum iron and ferritin levels, 25 participants (19.7%; 95% CI: 12.7%–26.7%) had iron deficiency. Comparison of hematological indices showed no significant difference in mean hemoglobin (16.7 g/dL in both groups; p = 0.99) and hematocrit (52.6 vs 53.2; p = 0.86) between iron-deficient and non-iron-deficient groups. Similarly, mean corpuscular volume (77.3 vs 80.5 fL), mean corpuscular hemoglobin (25 vs 24.4 pg), and mean corpuscular hemoglobin concentration (28.4 vs 28.9 g/dL) differences were also not statistically significant. **Conclusion:** The study revealed that 19.7% of children with CCHD have iron deficiency. Neither the clinical predictors of iron deficiency nor the erythrocytic parameters varied between the two groups, underscoring the importance of routine iron status evaluation.

Key words: Congenital heart disease, CCHD, iron deficiency anemia, serum ferritin, erythrocyte indices.

Congenital heart disease (CHD) is the most frequently occurring congenital anomaly globally, accounting for approximately 28% of all congenital birth defects. The global birth prevalence of CHD is estimated to range between 8 and 12 per 1,000 live births [1, 2]. CHDs are traditionally classified into acyanotic and cyanotic lesions based on the presence or absence of systemic hypoxemia, and cyanotic congenital heart diseases (CCHD) comprise 20% of all CHD cases [1]. CCHD encompasses a diverse range of defects, including Tetralogy of Fallot (TOF), transposition of the great arteries (TGA), tricuspid atresia, and truncus arteriosus. Of these, TOF is the most common, accounting for more than half of cyanotic lesions in many population studies [3]. Children with CCHD often remain unrepaired for prolonged periods in low and middle-income countries due to economic and infrastructural barriers.

These children are subjected to a chronic hypoxic state, which exerts complex systemic effects, particularly on hematologic physiology and nutritional status. Chronic hypoxia in CCHD stimulates the renal production of erythropoietin, leading to secondary erythrocytosis. The compensatory increase in red cell mass aims to enhance oxygen-carrying capacity but is dependent on adequate iron availability. The resultant high demand for iron can exhaust iron stores, especially in the presence of poor dietary intake or malabsorption, leading to iron deficiency. However, this deficiency is often masked due to paradoxically elevated hemoglobin (Hb), hematocrit (Hct), and other erythrocyte indices, which are misleadingly interpreted as markers of adequate or excessive iron status [6, 7]. This creates a significant diagnostic blind spot, where children at risk of iron deficiency anemia (IDA) remain undiagnosed [4].

Iron deficiency is the most common nutritional deficiency worldwide and is particularly prevalent in developing countries. In India, the prevalence of anemia among children

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aged 6 months to 5 years is reported to be as high as 56%, with iron deficiency being the leading cause [5]. In CCHD, iron deficiency can aggravate clinical symptoms and increase the risk of complications such as hyperviscosity syndrome, cyanotic spells, metabolic acidosis, and even cerebrovascular accidents. However, iron deficiency often remains underrecognized in children with CCHD due to paradoxically elevated Hb, Hct, mean corpuscular volume (MCV), and other erythrocyte indices used in traditional screening of anemia. Serum ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin saturation are more accurate indicators but are not routinely measured in many settings [6, 7].

The prevalence of iron deficiency in children with CCHD varies widely in existing literature from 18% to 72%, depending on the population studied and the diagnostic criteria employed [6-10]. However, there is limited data from Indian tertiary care centers evaluating iron status using standardized serum biomarkers in children with unrepaired CCHD [6, 7]. Therefore, we planned this study to estimate the serum iron status in patients with CCHD. Establishing the prevalence of iron deficiency and identifying its predictors can help develop targeted therapies for nutritional intervention and optimize clinical outcomes.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of Pediatrics, Division of Pediatric Cardiology, at the All India Institute of Medical Sciences (AIIMS), Rishikesh. The study was conducted over 18 months, from July 1, 2021, to December 31, 2022, following approval from the Institutional Ethics Committee. Patient confidentiality was strictly maintained throughout the study. All identifiable information was anonymized. Participants were recruited from pediatric cardiology outpatient clinics, as well as from inpatient units including the Pediatric Cardiology Ward, Cardiothoracic and Vascular Surgery (CTVS) Ward, and Pediatric Intensive Care Unit (PICU).

Children aged 6 months to 18 years, with a confirmed diagnosis of unrepaired CCHD or those who had undergone palliative procedures like Blalock-Taussig and Bidirectional Glenn shunts, were eligible for inclusion. Informed written consent was obtained from the parents before enrollment. Children on current iron supplementation, receiving long-term therapy with proton pump inhibitors (PPIs), H2-receptor antagonists, antacids, or with a history of surgical repair for CCHD, partial exchange transfusion, blood transfusion, or repeated phlebotomy within the last 3 months, were excluded.

The sample size was calculated based on the prevalence of iron deficiency anemia in CCHD, estimated at 9.3% as reported by Itiola *et al.* [8]. Using a 95% confidence level (Z = 1.96), an absolute precision of 10%, and a two-sided alpha error of 0.05, the minimum sample size was calculated using the formula ($N = Z^2P(1-P)/d^2$), and a total of 127 participants were included in the final analysis.

CCHD was confirmed using echocardiography, ECG, and chest X-ray. Routine investigations, including complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), and iron profile (serum iron, serum ferritin), were sent. Anthropometric assessment (weight, height/length, mid-upper arm circumference) was done, using the World Health Organization (WHO) growth charts, to classify malnutrition. A 24-hour dietary recall was used to estimate caloric and protein intake, and deficits were categorized as high (>20%) or low (<20%). Socioeconomic status was assessed using the Modified Kuppuswamy Scale, and the frequency of cyanotic spells was recorded. IDA was diagnosed per WHO criteria. Clinical history, physical examination, demographics, and baseline biochemical parameters were documented in a structured proforma. Predictors of iron deficiency were identified and analyzed for statistical significance

Statistical Analysis

Data were entered and analyzed using Microsoft Excel and IBM SPSS (Statistical Package for Social Sciences) software version 24.0. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range), depending on the distribution of data. Categorical variables were presented as proportions. The unpaired Student's t-test was used to compare means for normally distributed variables. A p-value <0.05 was considered statistically significant. All p-values were two-tailed.

RESULTS

A total of 135 children with CCHDs were screened for possible inclusion in the study. Eight children were excluded; out of them, six were receiving iron supplementation, and two had undergone surgical correction. The final study population comprised 127 children, including 75 (59.1%) males and 52 (40.9%) females. The mean age was 5.6 ± 4.7 years, with a median of 4 years (IQR: 2–9 years).

Table 1 - Demographic characteristics of the study population

Characteristic	Value
Age	
Mean age (years)	5.6 ± 4.7
Median age (years)	4 (IQR: 2–9)
Gender	
Male	75 (59.1%)
Female	52 (40.9%)
Socioeconomic Status *(Modified Kuppuswamy Scale)*	
Lower	10 (7.9%)
Lower middle	44 (34.7%)
Upper lower	70 (55.1%)
Upper middle	3 (2.4%)
Parental Education	
Uneducated	85 (66.9%)
Educated	42 (33.1%)
Weight-for-Age Z-scores (WAZ)	

Normal (Mean to -1 SD)	19 (15.0)
-2 to -3 SD	55 (43.3)
< -3 SD	53 (41.7)
Height-for-Age Z-scores (HAZ)	
Mean to +1 SD	1 (0.8)
Mean to -1 SD	4 (3.2)
-1 to -2 SD	8 (6.3)
-2 to -3 SD	37 (29.1)
< -3 SD	77 (60.6)
Nutritional Deficiency	
Calorie deficiency (%)	26.8 ± 11.2
Protein deficiency (%)	17.5 ± 8.3

According to the modified Kuppuswamy scale, most participants (n = 70, 55.1%) were in the upper-lower class. Most parents (n = 85, 66.9%) were uneducated, while 42 (33.1%) had some form of education. 43.3% of children had moderate undernutrition, and 41.7% were severely undernourished. Caloric and protein deficiencies were present in all participants. The demographic characteristics are described in Table 1. TOF was the most prevalent diagnosis (70, 55.1%), followed by ventricular septal defect (VSD) with severe pulmonary stenosis (PS) or atresia (17, 13.3%), Double outlet right ventricle (DORV) with VSD and PS (12, 9.4%), complex CCHD (12, 9.4%), TGA with VSD and PS (8, 6.2%), Tricuspid atresia (7, 5.5%), and complete AV canal defect with severe pulmonary stenosis (1, 0.7%).

The mean serum iron level was 9.9±3.1 ng/mL, and the mean serum ferritin was 22.4±10.9 ng/mL. Iron deficiency, defined by WHO age-specific serum ferritin cut-offs, was present in 25 children (19.7%, 95% CI: 12.7%–26.7%), and anemia was present in 11 children (8.7%). The comparison of hematological parameters between iron-deficient (n=25) and non-deficient (n=102) patients revealed no statistically significant changes. Mean hemoglobin levels in both groups were the same (16.7 g/dL), and hematocrit values were similar (52.6% vs. 53.2%; p=0.86). Red cell indices, such as MCV (77.3 fL vs. 80.5 fL; p=0.26), MCH (25.0 pg vs. 24.4 pg; p=0.49), and MCHC (28.4 g/dL vs. 28.9 g/dL; p=0.48), did not differ significantly between groups (Table 2).

Table 2 - Hematological indices in iron-deficient vs. non-deficient children

Parameter	Iron Deficient (n=25)	Non-Deficient (n=102)	p-value
Hemoglobin (g/dL)	16.7 ± 3.9	16.7 ± 5.4	0.99
Hematocrit (%)	52.6 ± 14.5	53.2 ± 14.5	0.86
MCV (fL)	77.3 ± 12.8	80.5 ± 12.6	0.26
MCH (pg)	25.0 ± 3.4	24.4 ± 3.7	0.49
MCHC (g/dL)	28.4 ± 4.1	28.9 ± 4.1	0.48

Analysis of potential predictors for iron deficiency revealed no significant associations across age, gender, socioeconomic status (SES), nutritional status, or dietary deficits (Table 3). Among children aged ≤5 years, 18.1% were iron deficient compared to 21.8% of those >5 years (p=0.59). Iron deficiency was observed in 21.3% of males and 17.3% of females (p=0.57). The prevalence of iron deficiency was similar in undernourished (WAZ < -2SD) and the normal WAZ group (21.3% vs. 10.5%; p=0.27). Stunting (HAZ < -2SD) showed similar trends (18.4% of affected children vs. 30.8% in normal HAZ group; p=0.29).

Table 3 - Association of Demographic and Nutritional Predictors with Iron Deficiency

Predictor	Iron Deficient (%)	Non-Deficient (%)	p-value
Age ≤5 years	13 (18.1)	59 (81.9)	0.59
Male gender	16 (21.3)	59 (78.7)	0.57
Low SES	12 (22.2)	42 (77.8)	0.53
Undernutrition (WAZ)	23 (21.3)	85 (78.7)	0.27
Stunting (HAZ)	21 (18.4)	93 (81.6)	0.29
High Calorie Deficit	15 (18.1)	68 (81.9)	0.53
High Protein Deficit	6 (15.0)	34 (85.0)	0.37

DISCUSSION

In our study, the prevalence of iron deficiency was 19.7% (25/127) and of IDA was 8.7% (11/127). IDA did not vary significantly with age, gender, socioeconomic status, and level of education in this study. No significant difference was found in various hematological parameters and red cell indices between iron-deficient and non-deficient patients. The most common CCHD in this study was TOF (n=70, 55.1%), followed by VSD and PS/PA (17, 13.3%). Other authors reported similar findings. TOF was diagnosed in 48.8% and 74.51% of children with CCHD in studies by Ossei *et al.*, and Mukherjee *et al.*, respectively [6, 11]. The prevalence of TOF was 39.3% in a study by Zhou *et al.* and 52% in a study by Itiola *et al.* [8, 12]. The next common CCHDs found in our study were VSD with severe PS/PA, DORV, and TGA. Other authors also reported similar findings [6, 8, 11, 12].

In our study, 19.7% (25/127) of CCHD patients had iron deficiency, and 8.7% (11/127) had IDA. Studies assessing iron deficiency among children with CCHD are limited and have shown varying results. A few studies have reported a high prevalence of iron deficiency in children with CCHD. In a case-control study by Ossei *et al.*, 47.5% of CCHD patients had IDA [11]. Similarly, Mukherjee *et al.* found that 47.06% (24/51) CCHD patients were iron deficient [6]. A study conducted by West *et al.* found that more than one-third of patients with CCHD had iron deficiency [13]. According to Olcay et al., the prevalence rate was 52% [14]. A previous study from India by Gaiha *et al.* found a prevalence of 18.18%, however, they included adolescents and young adults in their study [15].

On the contrary, Itiola *et al.* reported a lower prevalence of iron deficiency than other studies. In their study, the prevalence rate of iron deficiency was only 9.3% in children with CCHD, and none of them had IDA [8]. In a study from Kenya, Lang'o et al. reported a lower prevalence of iron deficiency (16.9%)

[10]. These variations in the reported prevalences of iron deficiency and IDA may be because of differences in study populations, such as sample size, age group, and nutritional status of the children.

In this study, we defined iron deficiency according to WHO age-specific serum ferritin cut-offs. The mean serum iron level was 9.9±3.1 ng/mL, and the mean serum ferritin was 22.4±10.9 ng/mL. The American Academy of Pediatrics also recommended the measurement of ferritin and CRP, or reticulocyte Hb concentration (Ret-He), in addition to Hb levels to make a diagnosis of IDA [16]. Cheng *et al.* found that Ret-He, accompanied by Hb levels, can be a good diagnostic marker in evaluating iron deficiency and IDA in children with CHD [17]. They suggested cut-off values of Ret-He <28.8 pg with Hb ≥16.5 g/dL to diagnose iron deficiency without anemia, and Ret-He <28.15 pg or Ret-He of 28.15-28.8 pg with Hb <16.5 g/dL to diagnose IDA [17]. Another large pediatric study has also confirmed the use of Ret-He as a reliable diagnostic marker of IDA in children [18].

We also compared various red cell indices in the iron-deficient and non-iron-deficient groups and found no significant differences between iron-deficient and non-deficient children. Many studies have utilized other hematological parameters and red cell indices to assess iron deficiency in children with CCHD; however, different studies have shown conflicting results. Previous studies suggested a cut-off value of <80 fL for MCV to diagnose iron deficiency in children with CHD [11, 14]. However, MCV values can be affected by secondary erythropoiesis. Previous studies have shown lower values of MCV, MCH, and MCHC in children with CHD in different iron status groups [6, 17, 19]. In our study, MCV, MCH, and MCHC values in the two groups did not vary significantly.

Among the predictors of iron deficiency, age, gender, socioeconomic status (SES), and anthropometric measurements did not show a significant difference between the two groups. Children with CHD have a higher risk of malnutrition. In this study, 21 stunted children were found to have iron deficiency. In the case-control study by Mir *et al.*, 31% of children were underweight, while 48% were stunted. They also found that 31% of children had moderate malnutrition, and 68.9% had severe malnutrition [19]. Other researchers also reported a high prevalence of stunting in children with CCHD [20-22]. Similar results have been reported in this study, and the probable cause for this was the fact that CCHD is a chronic, debilitating heart condition taking a toll on the child's nutritional status and growth. Chronic hypoxia has been shown to disrupt the linear growth in such children. Secondly, synthesis of insulin-like growth factor-binding protein (IGFBP-1), which facilitates growth, is affected by iron deficiency [23].

Patients with CCHD are at increased risk of developing IDA secondary to increased iron demands and depletion of anatomic reserves due to chronic hypoxia and secondary erythropoiesis. Malabsorption due to diarrhea and iatrogenic blood loss due to therapeutic interventions may add to the woe. Increased blood viscosity due to secondary erythropoiesis and increased erythrocyte mass can impair tissue oxygen delivery in these children. Worsening hyperviscosity increases the risk of thromboembolic events, cyanotic spells, metabolic acidosis, and mortality [6, 24, 25].

The study was conducted at a government-backed hospital setup, catering mainly to the poor population, therefore, the sample size might not be representative of the actual disease burden in the general population. Further large-scale research is necessary to assess the actual disease burden in society. Many other conditions, like recent and chronic infections or renal diseases, etc. can affect the bone marrow and iron metabolism. Serum ferritin, being an acute-phase reactant, can be falsely elevated in such conditions.

CONCLUSION

This study investigated the prevalence of iron deficiency in children with CCHD and explored various predictors of iron deficiency. The study revealed that 19.7% of children with CCHD have iron deficiency. Neither the clinical predictors of iron deficiency, such as age, gender, socio-economic status, nor the red cell indices varied between the two groups. This underscores the importance of routine iron status evaluation in children with congenital heart diseases.

REFERENCES

1. Saxena A, Mehta A, Sharma M, *et al*. Birth prevalence of congenital heart disease: A cross-sectional observational study from North India. Ann Pediatr Cardiol. 2016 Sep-Dec; 9(3):205-9. doi: 10.4103/0974-2069.189122.
2. Wu W, He J, Shao X. Incidence and mortality trend of congenital heart disease at the global, regional, and national level, 1990-2017. Medicine (Baltimore). 2020; 99(23):e20593. doi: 10.1097/MD.0000000000020593.
3. Bhat NK, Dhar M, Kumar R, *et al*. Prevalence and pattern of congenital heart disease in Uttarakhand, India. Indian J Pediatr. 2013; 80(4):281-5. doi: 10.1007/s12098-012-0738-4.
4. Kumsa H, Woldesenbet R, Mulugeta F, *et al*. Anemia in Children with Congenital Heart Disease: A Finding from Low-Resource Setting Hospitals. Int J Pediatr. 2024; 2024:8095150. doi: 10.1155/2024/8095150.
5. Bharati S, Pal M, Bharati P. Prevalence of anaemia among 6- to 59-month-old children in India: the latest picture through the NFHS-4. J Biosoc Sci. 2020; 52(1):97-107. doi: 10.1017/S0021932019000294.
6. Mukherjee S, Sharma M, Devgan A, *et al*. Iron deficiency anemia in children with cyanotic congenital heart disease and effect on cyanotic spells. Med J Armed Forces India. 2018; 74(3):235-240. doi: 10.1016/j.mjafi.2017.07.003.
7. Onur CB, Sipahi T, Tavil B, *et al*. Diagnosing iron deficiency in cyanotic heart disease. Indian J Pediatr. 2003; 70(1):29-31. doi: 10.1007/BF02722740.
8. Itiola AY, Animasahun BA, Njokanma OF. Serum Iron Status of Children with Cyanotic Congenital Heart Disease in Lagos, Nigeria. Sultan Qaboos Univ Med J. 2019; 19(4):e345-e351. doi: 10.18295/squmj.2019.19.04.010.
9. Said YH, Assenga E, Munubhi E, *et al*. Prevalence of iron deficiency and iron deficiency anaemia among children with congenital heart defects at tertiary hospitals in Dar es Salaam,

Tanzania: a cross-sectional study. Pan Afr Med J. 2022; 43:175. doi: 10.11604/pamj.2022.43.175.30944.
10. Lang'o MO, Githanga JN, Yuko-Jowi CA. Prevalence of iron deficiency in children with cyanotic heart disease seen at Kenyatta National Hospital and Mater Hospital Nairobi. East Afr Med J. 2009; 86(12 Suppl):S47-51. doi: 10.4314/eamj.v86i12.62901.
11. Ossei I, Buabeng KO, Ossei PPS, *et al*. Iron-deficiency anaemia in children with congenital heart diseases at a teaching hospital in Ghana. Heliyon. 2020; 6(2):e03408. doi: 10.1016/j.heliyon.2020.e03408.
12. Zhou D, Deng LJ, Ling YF, *et al*. Preoperative Hemoglobin Level, Oxygen Saturation, and Postoperative Outcomes in Children with Cyanotic Congenital Heart Disease: A Propensity-Score Matching Analysis. Front Pediatr. 2022; 9:762241. doi: 10.3389/fped.2021.762241.
13. West DW, Scheel JN, Stover R, *et al*. Iron deficiency in children with cyanotic congenital heart disease. J Pediatr. 1990; 117(2 Pt 1):266-8. doi: 10.1016/s0022-3476(05)80544-x.
14. Olcay L, Ozer S, Gürgey A, *et al*. Parameters of iron deficiency in children with cyanotic congenital heart disease. Pediatr Cardiol. 1996 May-Jun; 17(3):150-4. doi: 10.1007/BF02505204.
15. Gaiha M, Sethi HP, Sudha R, *et al*. A clinico-hematological study of iron deficiency anemia and its correlation with hyperviscosity symptoms in cyanotic congenital heart disease. Indian Heart J. 1993; 45(1):53-5.
16. Baker RD, Greer FR; Committee on Nutrition American Academy of Pediatrics. Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children (0-3 years of age). Pediatrics. 2010; 126(5):1040-50. doi: 10.1542/peds.2010-2576.
17. Cheng W, Yanuarso PB, Wahidiyat PA, *et al*. The role of reticulocyte hemoglobin equivalent on the evaluation of iron deficiency and iron deficiency anemia in pediatric cyanotic heart disease: a diagnostic study in Indonesia. BMC Pediatr. 2024 Aug 23; 24(1):541. doi: 10.1186/s12887-024-05000-w.
18. Poventud-Fuentes I, Chong TH, Dowlin M, *et al*. Reticulocyte hemoglobin equivalent as a marker to assess iron deficiency: A large pediatric tertiary care hospital study. Int J Lab Hematol. 2024; 46(1):148-155. doi: 10.1111/ijlh.14188.
19. Mir AB, Shah JA, Mushtaq S, *et al*. Prevalence, profile of malnutrition and iron deficiency anemia in children with cyanotic congenital heart defects: a case control observational study in a tertiary care hospital in North India. Int J Contemp Pediatr. 2019; 6:1303–5. 10.18203/2349-3291.ijcp20192033.
20. Okoromah CA, Ekure EN, Lesi FE, *et al*. Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study. Arch Dis Child. 2011; 96(4):354-60. doi: 10.1136/adc.2009.176644.
21. Hassan BA, Albanna EA, Morsy SM, *et al*. Nutritional Status in Children with Un-Operated Congenital Heart Disease: An Egyptian Center Experience. Front Pediatr. 2015; 3:53. doi: 10.3389/fped.2015.00053.
22. Chinawa AT, Chinawa JM, Duru CO, *et al*. Assessment of Nutritional Status of Children With Congenital Heart Disease: A Comparative Study. Front Nutr. 2021; 8:644030. doi: 10.3389/fnut.2021.644030.
23. Mohammed SH, Larijani B, Esmaillzadeh A. Concurrent anemia and stunting in young children: prevalence, dietary and non-dietary associated factors. Nutr J. 2019; 18(1):10. doi: 10.1186/s12937-019-0436-4.
24. González R, Urbano J, Solana MJ, *et al*. Microcirculatory Differences in Children With Congenital Heart Disease According to Cyanosis and Age. Front Pediatr. 2019; 7:264. doi: 10.3389/fped.2019.00264.
25. Shebl SS, El-Shehaby WAN, Said YS, *et al*. Thrombo-hemorrhagic liability in children with congenital heart diseases. Hematol Oncol Stem Cell Ther. 2018; 11(3):123-128. doi: 10.1016/j.hemonc.2017.07.001.

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