

Nutritional Status Profile in Children with Acyanotic Congenital Heart Disease: Analysis of Relationship with Diagnosis and Defect Size

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Abstract

Congenital heart disease (CHD) occurs in approximately 9 per 1,000 live births and can impair growth due to chronic hemodynamic burden, feeding difficulties, and increased energy demands. This study aimed to describe the nutritional status of children with acyanotic CHD and assess its relationship with diagnosis and defect size. A cross-sectional analytical study was conducted involving 34 children with patent ductus arteriosus (PDA) or ventricular septal defect (VSD) scheduled for transcatheter closure. Nutritional status was determined using anthropometric measurements, while defect size was assessed by 2D echocardiography. Associations were analyzed using Spearman correlation and Kruskal-Wallis tests. Of the 34 subjects, 58.8% (n=20) had good nutritional status, 29.4% (n=10) were undernourished, and 11.8% (n=4) had severe malnutrition. Malnutrition was slightly more common in VSD (50.0%) than PDA (37.5%). Defect size ranged from 2.0–7.0 mm, with a median of 5.0 mm, and most defects were classified as small to moderate. Median defect sizes were 5.0 mm in both the good nutritional status and undernutrition groups and 4.5 mm in the severe malnutrition group, with no significant difference ($p=0.549$). Nutritional status was also not significantly correlated with absolute defect size ($p=0.128$; $p=0.473$). Although most children had good nutritional status, malnutrition remained prevalent (41.2%). These findings suggest that malnutrition in CHD is multifactorial and cannot be explained solely by the anatomical diameter of the defect.

Keywords: Nutritional Status, Congenital Heart Disease, Defect Size, Patent Ductus Arteriosus, Ventricular Septal Defect

1. Introduction

Congenital heart disease (CHD) is one of the most prevalent congenital abnormalities. A systematic review and meta-analysis involving 114 studies found that, since 1995, CHD has occurred in approximately 9.1 out of every 1,000 live births, equivalent to around 1.35 million affected newborns annually.¹ Acyanotic defects involving left-to-right shunts, such as patent ductus arteriosus (PDA) and ventricular septal defect (VSD), can lead to excessive pulmonary blood flow and volume overload. These physiological disturbances, combined with feeding difficulties and recurrent respiratory infections, may impair growth and contribute to malnutrition.^{2,3}

Malnutrition among individuals with congenital heart disease (CHD) develops through several interconnected mechanisms. A substantial left-to-right cardiac shunt increases pulmonary circulation and places an excessive volume burden on the heart, thereby raising basal metabolic activity and overall energy needs. At the same time, patients may consume insufficient calories because feeding can cause fatigue and rapid breathing, while recurrent respiratory infections further reduce intake. In patients who develop heart failure, systemic venous congestion may also interfere with the absorption of essential nutrients.⁴

Clinically, the degree of hemodynamic burden is often associated with the anatomical size of the defect. Based on pediatric

echocardiography guidelines, defect size is classified relative to the surrounding heart structures. In patients with VSD, a defect smaller than 50% of the aortic annulus diameter is considered small and restrictive. Conversely, a defect exceeding the diameter of the aortic annulus is classified as large and nonrestrictive, resulting in a substantial volume load on the heart.⁵ In PDA, a defect with a minimal diameter of less than 1.5 mm is generally considered hemodynamically insignificant, whereas a size above 3 mm carries a high risk of causing heart failure.⁶

Although theoretically a larger defect would produce a more massive shunt and have a worse impact on child growth, empirical data regarding the direct correlation between absolute defect size and nutritional status in the CHD population in developing countries is still limited.⁷ This study aimed to investigate the relationship between nutritional status, clinical diagnosis, and the absolute size of cardiac defects measured by transthoracic echocardiography in children with acyanotic congenital heart disease, specifically patent ductus arteriosus (PDA) and ventricular septal defect (VSD).

2. Method

This cross-sectional analytical observational study involved children aged 1 month to 18 years who had isolated patent ductus arteriosus (PDA) or ventricular septal defect (VSD) and were scheduled for transcatheter defect closure at a referral healthcare facility. Patients with complex congenital cardiac abnormalities, growth-related genetic conditions such as Down syndrome, or other chronic illnesses were not included in the study. Ethical approval was granted by the Health Research Ethics Committee of the Faculty of Medicine,

Universitas Sriwijaya, and Dr. Mohammad Hoesin General Hospital, Palembang (Approval No. 141/KEPKRSMHFKUNSRI/2016).

The researchers obtained the data from patients' medical records before they underwent the intervention procedure. Extracted variables included age (in months and years), gender, specific diagnosis (PDA or VSD), and the presence of comorbidities.

Defect size was measured using 2D transthoracic echocardiography (TTE) by an experienced pediatric cardiologist, according to standard guidelines.⁸ For VSD, the defect size was measured during the diastolic phase, while for PDA, the minimal ductal diameter was measured during the systolic phase. Measurement results were recorded in millimeters (mm).

Anthropometric data collected included body weight (in kilograms) and height (in centimeters). Nutritional status in the source dataset was recorded as good nutrition, undernutrition or severe malnutrition (g. For transparent and reproducible classification, the original anthropometric indicator used for each child must be reported before submission. Nutritional status should be assessed using WHO growth standards appropriate to the child's age: the WHO Child Growth Standards for children under five years of age and the WHO Growth Reference for those aged 5–19 years. Under-nutrition and severe malnutrition are conventionally defined by the relevant anthropometric z-score below –2 SD and below –3 SD, respectively.^{9,10}

Statistical analyses were conducted using Python version 3.11. Non-normally distributed continuous variables were summarized using medians and ranges. The association between nutritional status, treated as an ordinal variable, and defect size, treated as a continuous variable, was assessed using

Spearman's rank correlation. Differences in defect size across nutritional status groups were analyzed with the nonparametric Kruskal–Wallis test, followed by post hoc Mann–Whitney U tests. A p-value below 0.05 was considered statistically significant.

3. Result

A total of 34 children met the inclusion criteria and were included in the analysis. Females constituted the majority of participants, accounting for 55.9%, while the median age was 7 years, with an age range of 1–17 years. Based on the diagnosis, most subjects suffered from PDA (70.6%) and the rest from VSD (29.4%).

Analysis of the nutritional status profile showed that although more than half of the children (58.8%) had good nutritional status,

the proportion of children experiencing malnutrition remained significant, at 41.2% (consisting of 29.4% undernutrition and 11.8% severe malnutrition).

When stratified by congenital heart disease diagnosis, the prevalence of malnutrition differed slightly between the groups. Malnutrition, including undernutrition and severe malnutrition, was observed in 37.5% of children with PDA (9 of 24 patients) and 50.0% of those with VSD (5 of 10 patients).

Echocardiographic assessment showed that the cardiac defect sizes ranged from 2.0 to 7.0 mm, with a median size of 5.0 mm. Based on standard echocardiographic classification, this size range generally represents small to moderate defects that have sufficient hemodynamic significance to be considered for closure intervention.

Table 1. Subject Characteristics Based on Nutritional Status Category

Nutritional Status	n (%)	Age (years), median	Defect Size (mm), median	PDA, n (%)	VSD, n (%)
Good Nutrition	20 (58.8%)	7.0	5.0	15 (75.0%)	5 (25.0%)
Undernutrition	10 (29.4%)	8.0	5.0	6 (60.0%)	4 (40.0%)
Severe Malnutrition	4 (11.8%)	3.0	4.5	3 (75.0%)	1 (25.0%)

Table 2. Association Between Nutritional Status and Defect Size

Analysis	Participants, n	Statistic	p-value
Defect size across nutritional-status categories	34	H=1.200	0.549
Overall nutritional-status score vs defect size	34	rho=0.127	0.473
PDA: nutritional-status score vs defect size	24	rho=0.357	0.087
VSD: nutritional-status score vs defect size	10	rho=-0.527	0.117
Malnutrition proportion: PDA vs VSD	34	OR=0.600	0.704

Kruskal–Wallis test was used for defect-size comparison. Spearman correlation used an ordinal nutritional-status score (1=severe malnutrition, 2=undernutrition, 3=good nutrition). OR: odds ratio; PDA: patent ductus arteriosus; VSD: ventricular septal defect.

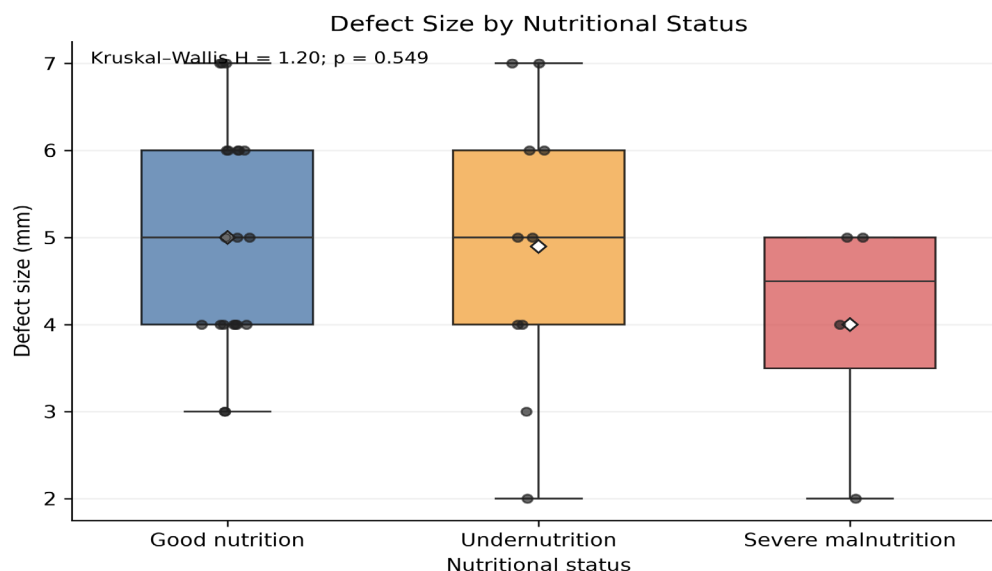


Figure 1. Distribution of defect size based on nutritional status

The distribution of defect size between nutritional status groups did not show a striking difference. The median defect size was 5.0 mm in both the normal nutritional status group (range: 3.0–7.0 mm) and the undernutrition group (range: 2.0–7.0 mm), whereas it was 4.5 mm among children with severe malnutrition (range: 2.0–5.0 mm). However, the Kruskal–Wallis test showed no statistically significant difference in defect size across the three nutritional status groups ($H = 1.200$; $p = 0.549$).

To evaluate the direct relationship between the severity of malnutrition and the anatomical size of the defect, a Spearman correlation test was performed. Analysis of all subjects ($n=34$) found no significant correlation between nutritional status and defect size (Spearman $\rho = 0.128$; $p = 0.473$). This indicates that the absolute defect size (in millimeters) cannot be used as a single predictor to determine the severity of a patient's nutritional status.

The median size of the defect was 5.0 mm in the good-nutrition and undernutrition groups and 4.5 mm in the severe-malnutrition

group. Table 2 summarizes all inferential analyses. Defect size did not differ significantly across nutritional-status categories (Kruskal–Wallis $H=1.200$; $p=0.549$). The overall correlation between nutritional status and absolute defect size was not statistically significant (Spearman $\rho=0.127$; $p=0.473$).

Figure 1 displays the distribution of defect size within each nutritional-status category. The distributions overlapped substantially, consistent with the non-significant Kruskal–Wallis result.

4. Discussion

This study offers valuable insight into the nutritional status of children with acyanotic congenital heart disease, specifically patent ductus arteriosus and ventricular septal defect, and examines its association with the anatomical size of the cardiac defect. The observed malnutrition rate of 41.2%, encompassing both undernutrition and severe malnutrition, is consistent with previous international studies, which have reported prevalence rates of 27%–65% among children

with congenital heart disease. This variation is influenced by the complexity of the cardiac defect and the socioeconomic characteristics of the population studied.^{11 12}

Among pediatric patients with left-to-right shunt cardiac lesions, malnutrition was observed in 50.0% of those with VSD and 37.5% of those with PDA. These descriptive findings should be interpreted cautiously because the diagnostic groups were small and unequal in size. This difference may be related to the specific hemodynamic dynamics of each anomaly. In VSD, the shunt occurs at the ventricular level (intracardiac), which can directly disrupt the efficiency of ventricular pumping mechanics early on.⁵ Meanwhile, in PDA, the shunt is extracardiac, and the left ventricular myocardium may have a better compensatory capacity (hypertrophy) in the early phase before eventually experiencing fatigue.

In this study, the absolute size of the cardiac defect was not significantly associated with the severity of nutritional impairment ($p = 0.128$; $p = 0.473$). However, this result should be interpreted with caution and does not indicate that defect size has no clinical importance. Previous studies have consistently demonstrated that malnutrition is prevalent among children with congenital heart disease, although its frequency and contributing factors differ considerably across populations. A systematic review by Diao et al. reported considerable preoperative underweight, stunting, and wasting in pediatric patients with congenital heart disease, while observational studies by Arodiwe et al. and Chinawa et al. identified factors including age at presentation, nutritional adequacy, and the presence of pulmonary hypertension. and overall clinical severity as important contributors to impaired growth. Therefore, the lack of a statistically

significant association observed in our study may reflect multifactorial nature of nutritional status and the limitation of using absolute defect diameter alone as a marker of haemodynamic burden. Unlike comprehensive haemodynamic measures, defect size expressed in millimeters does not account for age, body size, shunt magnitude, pulmonary vascular resistance, ventricular function, or pulmonary hypertension. Larger prospective studies using body-size-indexed echocardiographic parameters and standardized anthropometric z-scores are needed to clarify this relationship.^{2,12,13}

There are several pathophysiological and methodological explanations for this lack of correlation. First, the absolute size of the defect in millimeters does not accurately represent the true hemodynamic burden. The hemodynamic impact of a cardiac defect is largely determined by its size relative to the patient's body surface area (BSA) or cardiac dimensions.⁸ For example, a 5-mm defect in a 5-kg infant who is three months old may produce a substantial, nonrestrictive hemodynamic load, whereas the same-sized defect in a child 25-kg child aged 10 years may result in only a mild-to-moderate, restrictive burden.^{5,6} Because our study population has a very wide age range (1 to 17 years), using absolute defect size without indexation to BSA becomes a limitation in predicting clinical impact.

Second, the volume of the left-to-right shunt ($Q_p:Q_s$) is influenced not only by the diameter of the defect but also by the pressure gradient between the cardiac chambers and pulmonary vascular resistance (PVR).¹⁴ Thus, a patient with a large defect and elevated PVR due to reactive pulmonary vascular disease may have a smaller shunt volume than a patient with a moderate-sized defect and very low PVR.

Third, malnutrition in CHD is multifactorial. Besides hemodynamic factors, non-cardiac factors such as feeding practices, family socioeconomic status, frequency of respiratory tract infections (pneumonia), and genetic factors play a very dominant role in determining a child's growth trajectory.^{15, 16}

The clinical implications of these findings are highly relevant. Clinicians should not underestimate the increased susceptibility to malnutrition in pediatric patients with congenital heart disease because defect size appears "small" or "moderate" on echocardiography reports. Comprehensive nutritional evaluation, early nutritional intervention, and growth curve monitoring must be integrated standard care for all pediatric patients with congenital heart disease, irrespective of anatomical size of defect.

A major limitation of the present study was the limited number of participants included in the analysis, especially in VSD group and severe malnutrition group, which may limit the statistical power of the subgroup analysis. In addition, we did not measure more advanced functional echocardiographic parameters (such as Qp:Qs ratio, myocardial strain, or indexation of defect size to BSA) which might have a better correlation with nutritional status compared to absolute size.

5. Conclusion

Malnutrition remains a significant comorbidity in children with acyanotic CHD, with a prevalence reaching 41.2% in this study population. The proportion of malnutrition appears slightly higher in VSD patients compared to PDA patients. No significant correlation was found between absolute defect size based on echocardiography and the severity of nutritional status. This confirms that

malnutrition in CHD is a complex multifactorial process, and hemodynamic burden cannot be predicted solely from the anatomical diameter of the defect. Proactive nutritional interventions should be provided to all CHD patients regardless of their defect size. Future studies with larger cohorts and indexation of defect size to body surface area (BSA) are recommended to validate these findings.

References

1. van der Linde D, Konings EEM, Slager MA, Witsenburg M, Helbing WA, Takkenberg JJM, et al. [Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis](#). Journal of the American College of Cardiology. 2011;58(21):2241–7.
2. Diao J, Chen L, Wei J, Shu J, Li Y, Li J, et al. [Prevalence of malnutrition in children with congenital heart disease: a systematic review and meta-analysis](#). The Journal of Pediatrics. 2022;242:39–47.e4.
3. Hassan BA, Albanna EA, Morsy SM, Siam AG, Al Shafie MM, Elsaadany HF, et al. [Nutritional status in children with un-operated congenital heart disease: an Egyptian center experience](#). Frontiers in Pediatrics. 2015;3:53.
4. Chinawa, A. T., Chinawa, J. M., Duru, C. O., Chukwu, B. F., & Obumneme-Anyim, I. (2021). [Assessment of Nutritional Status of Children With Congenital Heart Disease: A Comparative Study](#). Frontiers in Nutrition. 8:644030:1-7.
5. NeoCardio Lab. [Ventricular Septal Defect Classification and Hemodynamic Assessment](#). 2023.
6. Merck Manuals. [Patent Ductus Arteriosus \(PDA\)](#) - Pediatrics. Merck Manuals Professional Version; 2023
7. Fitriany J, Maulina N, Mauliza, Wahyuni S. [Anthropometry and nutritional status of pediatric patients with congenital heart defects at a secondary hospital in North Aceh, Indonesia: a retrospective chart review](#).

- Progress in Pediatric Cardiology. 2025 Nov;80:101896.
8. Cohen MS, Eidem BW, Cetta F, et al. [Guidelines for Performing a Comprehensive Pediatric Transthoracic Echocardiography](#). Journal of the American Society of Echocardiography. 2024 Feb;37(2):119-170
9. World Health Organization. WHO [Child Growth Standards](#): methods and development. Geneva: World Health Organization; 2006.
10. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. [Development of a WHO growth reference for school-aged children and adolescents](#). Bulletin of the World Health Organization. 2007 ;85(9):660–7.
11. Okoromah CG, Ekure EN, Lesi FE, et al. [Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study](#). Archives of Disease in Childhood. 2011;96(4):354–60.
12. Arodiwe I, Ekure E, Eze J, et al. [Nutritional status of children with congenital heart disease \(CHD\) attending University of Nigeria Teaching Hospital Ituku - Ozalla, Enugu](#). Pakistan Journal of Medical. 2015;31(5):1140–5.
13. Chinawa AT, Chinawa JM, Duru CO, Chukwu BF, Obumneme-Anyim I. [Assessment of nutritional status of children with congenital heart disease: a comparative study](#). Frontiers in Nutrition. 2021;8:644030.
14. Cantinotti M, Di Salvo G, Voges I, et al. [Standardization in paediatric echocardiographic reporting and critical interpretation of measurements, functional parameters, and prediction scores: a clinical consensus statement](#). European Heart Journal - Cardiovascular Imaging. 2024;25(8):1029.
15. Blasquez A, Clouzeau H, Fayon M, et al. [Evaluation of nutritional status and feeding-related disorders in children with congenital heart disease](#). European Journal of Clinical Nutrition. 2016;175(2):233–41.
16. Daymont C, Neal A, Prosnitz A, et al. [Growth in children with congenital heart disease](#). Pediatrics. 2013;131(1):e236–42.