an idea health journal
ISSN (online): 2797-0604

Volume 6 No 1, Page 174-188

Malnutrition and its Association with Heart Failure Severity in Children with Congenital Heart Disease: A Cross-Sectional Study in Indonesia

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Article Info:
Received: 10 February, 2026
Revised: 10 March, 2026
Accepted: 24 March, 2026

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How to Cite:
Lainsamputty F, Widiantoro FX, Sumarmi, Pandelaki KM, & Maramis JR. Malnutrition and its Association with Heart Failure Severity in Children with Congenital Heart Disease: A Cross-Sectional Study in Indonesia. An Idea Health Journal. 2026;6(1):174-188. https://doi.org/10.53690/ihj.v6i01.651

Abstract

Background: Congenital heart disease (CHD) is a major cause of pediatric morbidity and mortality. Malnutrition often coexists with CHD and may be associated with poorer heart failure (HF) outcomes. The study aimed to assess the prevalence of malnutrition (underweight, wasting, and stunting) and its association with HF severity in children with CHD.

Methods: This was a single-center cross-sectional study at a tertiary hospital in Indonesia. The study included 41 pediatric CHD patients, recruited using a total sampling technique. Anthropometric data were assessed based on World Health Organization standards. HF severity was evaluated using the Modified Ross Score. The Mann-Whitney, Kruskal-Wallis, Spearman correlation, and multiple linear regression were applied.

Result: Among participants, 26.8% were underweight, 22.0% were wasted, and 36.6% were stunted. The mean HF severity score was 3.07 (SD = 1.40). Most were categorized as having mild HF (63.4%). Residence ($Z = 1.967$; $p = 0.049$), underweight ($Z = -2.394$; $p = 0.017$), stunting ($Z = -2.497$; $p = 0.013$), and wasting ($Z = -2.236$; $p = 0.025$) were significantly associated with higher HF severity scores. WAZ score significantly correlated with HF severity ($r = -0.348$; $p = 0.026$). Stunting ($\beta = -0.334$; 95% CI: -1.787 to -0.138; $p = 0.023$) and wasting ($\beta = -0.316$; 95% CI: -2.018 to -0.098; $p = 0.032$) were significant determinants of HF severity.

Conclusion: Stunting and wasting were independently associated with greater HF severity. These findings suggest the potential clinical importance of early nutritional screening and multidisciplinary interventions to mitigate HF progression. Given the single-center design and small sample size, further multicenter studies are needed to confirm these findings and strengthen causal inference.

Keywords: Congenital Heart Disease, Heart Failure, Malnutrition

BACKGROUND

Congenital heart disease (CHD) affects about 1% of live births, and remains a leading cause of childhood morbidity and mortality despite advances in care (1,2). Each year, CHD affects over one million newborns globally and contributes substantially to childhood deaths (3,4). Survivors often experience growth faltering and malnutrition, while untreated or severe CHD may progress to chronic heart failure (HF) with impaired growth, such as underweight and stunting (5). Thus, beyond its hemodynamic consequences, CHD represents a major nutritional challenge worldwide.

Malnutrition, including underweight (low weight for age), wasting (low weight for height), and stunting (low height for age), is highly prevalent in children with CHD. Rates are far higher than those in otherwise healthy children (6–8). Even in high-income settings, up to half of pediatric CHD patients show acute or chronic undernutrition (9–11). In low and middle-income countries (LMICs; countries with limited economic resources), rates can exceed 70% (12,13). These nutritional deficits are not only anthropometric (body measurement) concerns. They are also associated with frequent infections, longer hospitalizations, and poorer outcomes (10,14).

Evidence indicates a bidirectional link between malnutrition and HF severity in CHD, hemodynamic stress and feeding difficulties precipitate growth failure, while undernutrition reduces muscle and cardiac reserve, worsening HF symptoms (13,15). Biological mechanisms include increased metabolic demands due to hypoxemia and cardiac overload combined with limited intake from fatigue, tachypnea, or feeding intolerance (16,17). Over time, this imbalance accelerates weight loss and growth retardation (10).

To better conceptualize the relationship between malnutrition and HF severity in children with CHD, it is useful to integrate three complementary frameworks: The cardiac cachexia/catabolic-stress model, CHD-specific growth-failure pathways, and a bidirectional nutrition-HF reinforcing cycle. The cardiac cachexia model describes a chronic hypercatabolic state in which inflammatory and neurohormonal dysregulation, reduced intake, and metabolic inefficiency promote progressive loss of edema-free lean mass (with or without fat loss), reducing physiologic reserve and functional capacity (18–20). In pediatric HF, where CHD is a major cause, malnutrition is similarly understood as emerging from the combined effects of decreased dietary intake, decreased absorption, and an altered metabolic state, supporting the relevance of a catabolic-stress framing when HF is persistent or more severe (21). In parallel, growth-failure pathways emphasize that CHD physiology (e.g., cyanosis, pulmonary hypertension, and low cardiac output) can disrupt normal growth trajectories by increasing energy requirements and impairing endocrine and musculoskeletal development, while simultaneously limiting effective nutrient delivery through feeding intolerance and gastrointestinal mechanisms (22,23). Evidence also indicates that feeding and swallowing difficulties are common in pediatric CHD and may reduce feeding efficiency and caloric intake, reinforcing growth faltering in clinically stressed children (24). These mechanisms converge into a nutrition-HF cycle in which worsening HF increases metabolic demand and reduces feeding tolerance, whereas malnutrition, particularly protein-energy deficiency, may impair myocardial structure and function and further diminish reserve, thereby exacerbating HF symptom burden and contributing to worse clinical outcomes (20–22,25).

The challenge of malnutrition in pediatric CHD is especially pronounced in developing countries such as Indonesia. Pediatric undernutrition (insufficient intake of nutrients for growth and health) is a pervasive public health issue in Indonesia, contributing to high childhood morbidity and mortality. In this challenging context, children with CHD in Indonesia show alarmingly high rates of growth failure (including underweight, stunting, and wasting as previously defined). A recent hospital-based study in Yogyakarta reported that about half of children with CHD were underweight (49%) and nearly as many were stunted (48%), with one-third meeting criteria for wasting (5). Notably, these figures mirror findings from other LMIC (low- and middle-income country) settings, reflecting the compounded effect

of CHD on an already vulnerable nutritional status. Resource constraints and health system factors likely play a role: many children experience delayed diagnosis of CHD and limited access to early corrective surgery, prolonging their exposure to HF and inadequate nutrition (13). In addition, socioeconomic factors such as poverty and food insecurity (limited ability to obtain enough safe, nutritious food) further exacerbate malnutrition in this group. Taken together, in Indonesia and similar contexts, the intersection of CHD with prevalent childhood malnutrition poses a serious clinical and public health challenge.

Despite growing recognition of the coexistence of malnutrition and HF in children with CHD, important gaps remain in the current literature. Most previous studies have primarily focused on the prevalence of malnutrition or postoperative outcomes, with limited attention to how different dimensions of nutritional status (underweight, wasting, and stunting) are associated with HF severity. Moreover, evidence from LMICs, including Indonesia, remains scarce, despite these settings bearing a disproportionately high burden of both CHD and childhood malnutrition. To address this gap, the present study is guided by a conceptual linkage model that hypothesizes a bidirectional relationship between nutritional status and HF severity. In this model, CHD-related hemodynamic abnormalities contribute to increased metabolic demands, feeding difficulties, and impaired nutrient absorption, leading to various forms of malnutrition. In turn, malnutrition, particularly protein-energy deficiency, may impair myocardial structure, reduce cardiac reserve, and exacerbate HF symptoms. This reciprocal interaction is further reinforced through a nutrition-HF cycle, in which worsening HF accelerates nutritional decline, while poor nutritional status further aggravates disease severity. By explicitly examining this relationship, this study aimed to assess the prevalence of underweight, wasting, and stunting, and examine their association with HF severity in children with CHD.

METHODS

Study Design

This study employed a cross-sectional design. Forty-one pediatric patients with congenital heart disease (CHD) were recruited using a total sampling approach at a tertiary hospital in North Sulawesi, Indonesia, between December 2023 and May 2024.

Sample/Participants

Sample using total sampling, a formal a priori sample size calculation was not conducted due to the limited number of eligible cases during the study period; thus, the sample size was based on feasibility. Previous institutional data reported approximately 27 to 43 pediatric CHD cases over similar timeframes (26,27). Eligible participants were children aged 0 to 15 years diagnosed with CHD. Inclusion required caregiver consent and agreement to anthropometric assessments. Exclusion criteria included acute infections, syndromic conditions affecting growth (e.g., Down syndrome), or incomplete medical records.

Instruments

Collected demographic and clinical data comprised age, gender, residence, income, diagnosis, CHD type, and family history of heart disease. Anthropometric measurements included birth and current weight, height, mid-upper arm circumference (MUAC), and waist circumference. Nutritional assessment was restricted to anthropometric indicators based on World Health Organization (WHO) growth standards; dietary intake, feeding practices, and biochemical markers were not evaluated. Malnutrition in children was defined as underweight (low weight-for-age), wasting (low weight-for-height), or stunting (low height-for-age). Z-scores were calculated using WHO Anthro and AnthroPlus software and expressed as weight-for-age Z-score (WAZ), categorized as normal or underweight (< -2

SD); height-for-age Z-score (HAZ), categorized as normal or stunted (< -2 SD); and weight-for-height/BMI-for-age Z-score (WHZ/BAZ), categorized as normal or wasted (< -2 SD).

Heart failure (HF) severity was assessed using the Modified Ross Score, a validated, reliable, and non-invasive tool for evaluating HF symptoms, monitoring disease progression, and assessing treatment outcomes in infants and young children (28). This tool has been previously applied in pediatric populations in Indonesia (29). Participants were classified into four HF severity categories: 0–2 (no HF), 3–6 (mild), 7–9 (moderate), and 10–12 (severe) (30,31).

Following consent, demographic and socioeconomic data were collected via structured interviews with caregivers using a validated questionnaire. Clinical information, including diagnosis and CHD type, was extracted from patients' medical records. Trained nurses conducted anthropometric measurements—weight, height, mid-upper arm circumference (MUAC), and waist circumference—each measured twice and averaged to enhance accuracy. Data were entered into the WHO Anthro and AnthroPlus software to calculate Z-scores for WAZ, HAZ, and WHZ/BAZ, which were used to determine nutritional status categories of underweight, stunting, and wasting, respectively. A pediatric cardiologist assessed HF severity by observing and scoring respiratory effort, feeding patterns, growth parameters, and systemic perfusion indicators. Assessments were conducted in a private clinical setting to ensure accuracy and objectivity.

Data forms were reviewed immediately after completion to ensure completeness and accuracy. All information was coded and securely stored to maintain participant confidentiality. Ethical clearance was granted by the Institutional Review Board of Prof. Dr. R. D. Kandou General Hospital, Manado (approval number: 262/EC/KEPK-KANDOU/XII/2023). Eligible children with CHD were enrolled after their parents or legal guardians received information about the study objectives and provided written informed consent. Confidentiality and anonymity were strictly maintained throughout the study.

Data Analysis

Data analysis was conducted using SPSS version 27.0 (IBM Corp.). Descriptive statistics summarized demographic, clinical, and anthropometric characteristics, as well as HF severity, using means, standard deviations, frequencies, and percentages. Due to non-normal variable distributions determined by the Shapiro-Wilk test, the Mann-Whitney U test and Kruskal-Wallis H test were used for group comparisons, and Spearman's rank correlation assessed associations. Multiple linear regression examined associations between low birth weight, wasting, stunting, and the total Modified Ross Score (HF severity). The dependent variable was treated as a continuous numeric score, with low birth weight, stunting, and wasting entered as binary predictors. Regression assumptions—including normality of residuals, linearity, homoscedasticity, and absence of influential outliers—were verified and met. Multicollinearity was assessed via tolerance (>0.10) and variance inflation factor (<10). The Durbin-Watson statistic (1.679) indicated no autocorrelation. Statistical significance was set at $p < 0.05$.

RESULT AND DISCUSSION

RESULT

Table 1 presents the demographic and clinical characteristics of the participants. The mean age was 2.79 years (SD = 2.86), with a range from 0.02 to 9.00 years. Females comprised 61.0% of the sample, and 61.0% of participants resided in rural areas. Most families (73.2%) reported a monthly household income below 3 million rupiahs. Regarding clinical diagnoses, 29.3% of children had a ventricular septal defect (VSD), 31.7% had an atrial septal defect (ASD), and 39.0% had other congenital heart defects (CHD). Acyanotic defects were more common (68.3%) than cyanotic defects (31.7%). Furthermore, 46.3% of participants reported a family history of heart disease.

Table 1. Demographic and Clinical Characteristics (n = 41)

Variables	n (%)	Mean (SD)	Min – Max
Age (years)		2.79 (2.86)	0.02 – 9.00
Gender			
Female	25 (61.0)		
Male	16 (39.0)		
Place of residence			
Urban	16 (39.0)		
Rural	25 (61.0)		
Family income			
<Rp 3 million	30 (73.2)		
≥Rp 3 million	11 (26.8)		
Diagnosis			
VSD	12 (29.3)		
ASD	13 (31.7)		
Others	16 (39.0)		
Type of CHD			
Cyanotic	13 (31.7)		
Acyanotic	28 (68.3)		
Family history of heart disease			
No	22 (53.7)		
Yes	19 (46.3)		

Note: ASD – Atrial Septal Defect; CHD – Congenital Heart Disease; VSD – Ventricular Septal Defect.

Table 2 presents the participants' anthropometric data. The mean birth weight was 2.83 kg (SD = 0.53), with 80.5% classified as having normal birth weight. The mean current weight was 11.21 kg (SD = 6.55). According to the weight-for-age z-score (WAZ), 26.8% of participants were underweight, with a mean WAZ of -1.25 (SD = 1.34). The mean birth height was 48.32 cm (SD = 3.17), and the mean current height was 83.93 cm (SD = 26.75). The mean height-for-age z-score (HAZ) was -1.12 (SD = 1.62), with 36.6% of participants categorized as stunted. The weight-for-height z-score (WHZ) and BMI-for-age z-score (BAZ) had means of -0.78 (SD = 1.44), indicating that 22.0% of children were classified as wasted. The mean birth mid-upper arm circumference (MUAC) was 10.78 cm (SD = 1.86). The mean birth waist circumference was 32.56 cm (SD = 3.07), and the current waist circumference was 47.34 cm (SD = 10.83).

Table 2. Participants Anthropometric Data (n = 41)

Variables	n (%)	Mean (SD)	Min – Max
Birth weight (kg)		2.83 (0.53)	1.80 – 4.40
Low	8 (19.5)		
Normal	33 (80.5)		
Current weight (kg)		11.21 (6.55)	2.60 – 25.00
Weight-for-age Z-Score		-1.25 (1.34)	-3.74 – 1.94
Underweight	11 (26.8)		
Normal	30 (73.2)		
Birth height (cm)		48.32 (3.17)	41.00 – 60.00
Current height (cm)		83.93 (26.75)	47.00 – 136.00
Height-for-age Z-Score		-1.12 (1.62)	-3.40 – 1.86
Stunting	15 (36.6)		
Normal	26 (63.4)		
Weight-for-height/BMI-for-age Z-Score		-0.78 (1.44)	-3.04 – 1.99
Wasting	9 (22.0)		
Normal	32 (78.0)		

Variables	n (%)	Mean (SD)	Min – Max
Birth mid-upper arm circumference (cm)		10.78 (1.86)	6.00 – 14.00
Current mid-upper arm circumference (cm)		15.05 (3.55)	6.00 – 23.00
Birth waist circumference (cm)		32.56 (3.07)	27.00 – 45.00
Current waist circumference (cm)		47.34 (10.83)	31.00 – 85.00

Note: BMI – Body Mass Index; cm – Centimeter; kg – Kilogram.

Table 3 shows the severity of HF among the participants based on the Modified Ross Score. The mean score was 3.07 (SD = 1.40), ranging from 1.00 to 8.00. The majority of participants (63.4%) were classified as having mild HF symptoms (score 3-6).

Table 3. HF Severity Based on Modified Ross Score (n=41)

Variables	n (%)	Mean (SD)	Min – Max
Modified Ross score		3.07 (1.40)	1.00 – 8.00
No HF (0-2)	14 (34.1)		
Mild HF (3-6)	26 (63.4)		
Moderate HF (7-9)	1 (2.4)		
Severe HF (10-12)	0 (0.0)		

Note: HF-Heart Failure

Table 4 displays the results of bivariate analyses assessing differences in HF severity across categorical variables. Place of residence was significantly associated with HF severity ($Z = 1.967$, $p = 0.049$), with participants from rural areas showing higher severity scores (3.40 ± 1.53). Underweight status was also significantly associated with HF severity ($Z = -2.394$, $p = 0.017$), as underweight participants exhibited higher severity scores (4.09 ± 1.76). Similarly, stunting status demonstrated a significant association with HF severity ($Z = -2.497$, $p = 0.013$), with stunted children presenting more severe HF (3.67 ± 1.18). Wasting status was likewise significantly associated with HF severity ($Z = -2.236$, $p = 0.025$), with wasted children showing elevated severity scores (3.89 ± 1.27).

Table 4. Differences between categorical variables and HF severity (n = 41)

Variables	Mean (SD)	Statistics	*p*-value
Gender		1.288	0.217[a]
Female	2.92 (1.47)		
Male	3.31 (1.30)		
Place of residence		1.967	0.049[a]*
Urban	2.56 (1.03)		
Rural	3.40 (1.53)		
Family income		-1.540	0.124[a]
<Rp 3 million	3.30 (1.44)		
≥Rp 3 million	2.45 (1.13)		
Diagnosis		0.738	0.691[b]
VSD	2.83 (1.27)		
ASD	3.08 (1.71)		
Others	3.25 (1.29)		
Type of CHD		0.174	0.862[a]
Cyanotic	2.92 (1.04)		
Acyanotic	3.14 (1.56)		
Family history of heart disease		0.217	0.828[a]
No	2.95 (1.17)		
Yes	3.21 (1.65)		
Birth weight (kg)		-0.921	0.357[a]
Low	3.75 (2.19)		
Normal	2.91 (1.13)		

Variables	Mean (SD)	Statistics	*p*-value
Underweight status		-2.394	0.017[a]*
Underweight	4.09 (1.76)		
Normal	2.70 (1.06)		
Stunting status		-2.497	0.013[a]*
Stunting	3.67 (1.18)		
Normal	2.73 (1.43)		
Wasting status		-2.236	0.025[a]*
Wasting	3.89 (1.27)		
Normal	2.84 (1.37)		

*Note: *p < 0.05; **p < 0.01; ***p < 0.001; a – Mann-Whitney U test; b – Kruskal-Wallis H test; ASD – Atrial Septal Defect; CHD – Congenital Heart Disease; kg – Kilogram; VSD – Ventricular Septal Defect.*

Table 5 depicts the correlation analysis between numerical variables and HF severity among 41 participants. Age, birth weight, current weight, height measures, and circumferences did not show statistically significant correlations with HF severity ($p > 0.05$). However, weight-for-age Z-score (WAZ) demonstrated a significant negative correlation with HF severity ($r = -0.348$; $p = 0.026$).

Table 5. Correlation between numerical variables and HF severity (n = 41)

Variables	Modified Ross Score
Age (years)	$r = -0.043^a$ $p = 0.788$
Birth weight (kg)	$r = 0.028^a$ $p = 0.864$
Current weight (kg)	$r = -0.054^a$ $p = 0.737$
Weight-for-age Z-Score	$r = -0.348^{a*}$ $p = 0.026$
Birth height (cm)	$r = -0.293^a$ $p = 0.063$
Current height (cm)	$r = -0.054^a$ $p = 0.737$
Height-for-age Z-Score	$r = -0.250^a$ $p = 0.116$
Weight-for-height/BMI-for-age Z-Score	$r = -0.217^a$ $p = 0.173$
Birth mid-upper arm circumference (cm)	$r = 0.284^a$ $p = 0.072$
Current mid-upper arm circumference (cm)	$r = 0.175^a$ $p = 0.273$
Birth waist circumference (cm)	$r = -0.118^a$ $p = 0.463$
Current waist circumference (cm)	$r = -0.003^a$ $p = 0.986$

*Note: *p < 0.05; **p < 0.01; ***p < 0.001; a – Spearman's rank correlation; BMI – Body Mass Index; cm – Centimeter; kg – Kilogram.*

The determinant analysis for HF severity included three variables: Underweight status, stunting status, and wasting status. The overall model was statistically significant ($F = 4.370, p = 0.010$), accounting for 26.2% of the variance in HF severity, with an adjusted R-squared of 20.2%. Among these predictors, only stunting status ($\beta = -0.334$; 95% CI: -1.787 to -0.138; $p = 0.023$) and wasting status ($\beta = -0.316$; 95% CI: -2.018 to -0.098; $p = 0.032$) were significant determinants of HF severity.

Table 6. Determinants of HF Severity (n = 41)

Variables	B	ß	*t*	*p*	95% Confidence Interval (CI)		VIF
					Lower Bound	Upper Bound	
Underweight status	-0.790	-0.226	-1.597	0.119	-1.792	0.212	1.001
Stunting status	-0.962	-0.334	-2.366	0.023*	-1.787	-0.138	1.001
Wasting status	-1.058	-0.316	-2.233	0.032*	-2.018	-0.098	1.003
F	4.370						
p	0.010*						
R^2	0.262						
Adjusted R^2	0.202						

*Note: *p < 0.05; **p < 0.01; ***p < 0.001; B – Unstandardized Regression Coefficient; β – Standardized Beta Coefficient; VIF – Variance Inflation Factor.*

DISCUSSION

The high prevalence of underweight status among children with congenital heart disease (CHD) reflects chronic undernutrition and the physiological burden imposed by cardiac anomalies. Approximately one-quarter of participants in this study were underweight, aligning with a global estimate of 27% (8) and exceeding rates observed in healthy children. LMICs report even higher prevalence, ranging from 50% to 68% (5,32). This condition results from an energy imbalance caused by increased metabolic demands and reduced intake due to fatigue and feeding difficulties (33,34). Additional factors such as malabsorption, recurrent infections, and fluid restriction further exacerbate the condition (35). Persistent underweight status may delay surgical intervention and worsen clinical outcomes, underscoring the importance of early detection and nutritional support.

Approximately one-fifth of children in this study exhibited wasting, which is 3 times the global prevalence of 7% (8). Higher rates are frequently reported in LMICs, such as Uganda (31.5%) and Ethiopia (39–50%) (36), while even high-income countries report prevalence rates of 33%-52% (9). Wasting indicates the acute effects of heart failure (HF)-related metabolic stress and energy imbalance, emphasizing the critical need for early nutritional intervention in this population.

Stunting was observed in one-third of participants, higher than global CHD estimates (24.4%) and the general child population (23.2%) (37). Nearly half of Indonesian children with CHD are stunted, consistent with findings from Uganda (12). Even in some high-resource contexts, significant stunting has been observed in up to two-thirds of children with severe CHD who exhibit growth faltering in height despite optimal surgical care (38). This high prevalence of stunting reflects the cumulative impact of chronic illness, repeated illness episodes, prolonged HF, and inadequate nutrition over time, which impede linear growth. Such findings reinforce that stunting is a pervasive comorbidity of pediatric heart disease that can have lasting developmental consequences (32).

In Southeast Asia, malnutrition patterns in CHD mirror those in Africa, though prevalence varies. Indonesian and Thai cohorts report that nearly half to two-thirds of patients are malnourished (39). Regional prevalence often exceeds global averages. Importantly, malnutrition was consistently associated with greater HF severity, while improved nutritional status or rehabilitation after surgery is associated with better outcomes (39). This highlights the need for integrated nutritional support across Southeast Asia.

Our findings also show HF severity was predominantly mild, similar to reports outside tertiary centers (30). More advanced HF is often reported in referral units, reflecting differences in case mix. Variation in HF severity across clinical settings shows the need to consider referral patterns, healthcare access, and timing of diagnosis when interpreting such data. In addition, inconsistencies in pediatric HF classification systems have led to efforts to standardize diagnostic criteria. The American Heart Association (AHA) has recently stressed the need for uniform definitions to improve clinical

management and research comparability (40). Early recognition and risk stratification remain critical in preventing HF progression among children with CHD.

Rural residence was associated with increased heart failure severity in this study. This finding aligns with prior evidence indicating that limited access to specialized care contributes to delayed diagnosis and poorer clinical outcomes (41,42). Data from Indonesia and China confirm that children from rural areas frequently present with heart failure at diagnosis (43,44). This urban-rural disparity reflects underlying health inequities and underscores the necessity for enhanced referral systems and outreach programs in rural settings (45,46).

Underweight status correlated with higher HF severity, supporting prior studies where undernourished children had worse outcomes (5,13). This relationship is biologically plausible because malnutrition reduces cardiac reserve and exacerbates HF (8). These findings highlight that underweight status in a child with CHD is a red flag for clinicians. It may signal more severe cardiac insufficiency and potentially worse outcomes, requiring prompt nutritional support (47).

We found that stunting also correlates with HF severity. Stunted children in our study had significantly higher HF scores than non-stunted children. This indicates more severe HF symptoms among those with long-standing growth failure (48). Chronic malnutrition may impair somatic and myocardial growth, while persistent HF disrupts tissue perfusion and growth trajectories (5,39,49). This suggests a vicious cycle where stunting and HF reinforce each other. It underscores the need for integrated cardiac and nutritional interventions.

Wasting was another factor associated with greater HF severity, affecting about one-fifth of participants. This aligns with the concept of cardiac cachexia, where severe HF drives catabolism and muscle loss. Cardiac cachexia is a marker of poor prognosis in both children and adults with CHD (5,13,15). Pathophysiologically, elevated metabolic demands and cytokine-driven catabolism combined with poor intake accelerate wasting (16). These findings highlight the potential importance of nutritional support strategies. High-calorie feeding, tube supplementation, or timely surgical management may help in the clinical care of children with CHD. Addressing nutritional status may be associated with improved clinical outcomes and may help mitigate the interaction between HF severity and malnutrition (50).

Stunting remained a significant determinant of HF severity in this study. This association is biologically plausible: linear growth failure shows a prolonged imbalance between nutrition and the increased metabolic demands from congenital heart lesions, recurrent illness, and HF-related feeding and gastrointestinal issues. In a systematic review and meta-analysis, about one-quarter of children with CHD had stunting before intervention. This underscores that chronic growth restriction is common and a meaningful clinical signal, not an outlier (8). A large cohort study of children with CHD also found that congestive HF was independently associated with stunting, and stunting was linked to increased mortality. These findings suggest linear growth failure often clusters with more advanced or complicated heart disease (5). Contemporary pediatric CHD nutrition syntheses show that undernutrition in CHD stems from increased metabolic requirements, reduced intake, and impaired absorption linked to HF and abnormal hemodynamics. Severe protein-energy malnutrition can impair cardiac structure and function (e.g., myocardial atrophy and reduced ventricular mass), limiting physiologic reserve and worsening symptoms (22).

Wasting was also an independent determinant of HF severity in our analysis. Wasting captures acute energy-protein deficit and catabolic stress, and in children with CHD, it may both reflect and amplify a vicious cycle of diminished oral intake (feeding fatigue, early satiety), potential caloric restriction in the setting of HF management, higher resting energy expenditure, and impaired nutrient absorption. In a contemporary pediatric CHD-HF scientific statement, it is explicitly highlighted that children with CHD and HF are at risk for illness-related malnutrition because of increased metabolic

demands, decreased intake, and malabsorption, and they emphasize that optimizing nutrition is crucial to improving strength and reducing stress/fatigue related to cardiac insufficiency, supporting the clinical relevance of wasting when interpreting HF severity models (40). Consistent with a bidirectional nutrition-HF relationship, a meta-analysis synthesizing malnutrition risk factors in CHD noted that greater HF severity is associated with higher malnutrition risk and discusses plausible pathways, including venous congestion-related gastrointestinal dysfunction and impaired nutrient utilization during HF (34). Physiologic evidence from echocardiographic evaluation in children with severe acute malnutrition demonstrates reductions in left ventricular mass/mass index and systolic function parameters compared with nourished controls, lending biologic plausibility to the concept that acute wasting can translate into reduced cardiac reserve and thereby contribute to more severe HF manifestations (51).

These findings highlight the potential importance of early nutritional assessment and intervention in children with CHD. Routine screening for underweight, wasting, and stunting may help identify high-risk patients who could benefit from targeted nutritional support. Early interventions, including optimized caloric intake and feeding support, may be important parts of comprehensive HF management in pediatric populations. In LMIC settings, including Indonesia, several health system challenges may further complicate the management of children with CHD. Limited access to specialized pediatric cardiology services, delayed diagnosis, and restricted availability of advanced interventions such as corrective surgery may contribute to prolonged disease burden. Constraints in nutritional support services and caregiver education may also hinder optimal management of malnutrition in this population. These findings underscore the need for integrated approaches that address both cardiac and nutritional aspects of care, particularly in resource-limited settings.

This study has several limitations that should be considered when interpreting the findings. The study was conducted at a single center with a relatively small sample size (n = 41), which may limit statistical power, generalizability, and the possibility of reverse causality cannot be excluded. Although a multiple linear regression analysis was performed, the limited sample size restricted the number of variables that could be included in the model, increasing the risk of overfitting and unstable estimates. Consequently, important potential confounders such as age, CHD type, cyanotic status, socioeconomic factors, and micronutrient deficiencies or anemia were not adjusted for, and residual confounding cannot be excluded. The cross-sectional design precludes causal interpretation of the relationship between nutritional status and HF severity. Participants were not stratified by specific CHD types, which may have introduced clinical heterogeneity. HF severity was assessed using the Modified Ross Score, which, although widely used in pediatric settings, is based on clinical signs and symptoms and may be subject to interobserver variability. The absence of adjunctive objective measures such as echocardiography or biomarkers (e.g., BNP) may limit the precision of HF classification. Nutritional status was assessed solely using anthropometric indicators (underweight, wasting, and stunting), which, although widely used, may be less sensitive in children with CHD and may not fully capture the multidimensional nature of nutritional status, including body composition, micronutrient status, and metabolic alterations. Finally, important clinical and behavioral factors such as dietary intake, feeding practices, and inflammatory status were not assessed, which may influence both nutritional status and HF severity.

CONCLUSION

This study demonstrates that malnutrition remains prevalent among children with CHD. Stunting and wasting were identified as independent determinants associated with greater HF severity. While the findings suggest a possible bidirectional relationship between nutritional status and HF, further research

is needed to clarify whether chronic and acute undernutrition directly contribute to worsening clinical outcomes or result from more severe HF.

Building on these findings, recognizing stunting and wasting as clinical indicators of increased HF severity is essential for pediatric CHD care. Routine anthropometric screening should be integrated into standard clinical assessment, with emphasis on early detection of growth faltering. Targeted nutritional interventions, supported by multidisciplinary collaboration, may help mitigate disease progression. In LMIC settings, strengthening health systems through structured nutritional protocols and improving access to early diagnosis are critical priorities. Future multicenter and longitudinal studies are needed to validate these findings and explore causal mechanisms.

ACKNOWLEDGEMENTS

The authors express gratitude to all children with CHD and their caregivers for their trust and cooperation, which enabled this study. They also sincerely appreciate Srikanti Gracela Pangajow and Joel Marcelino Wewengkang for their valuable assistance in data collection and study implementation.

AUTHOR'S CONTRIBUTION STATEMENT

FL, FXW: Conceptualization, methodology, formal analysis, writing original draft, review & editing. S: Manuscript review, supervision. KMP, JRM: Formal analysis, review & editing.

CONFLICTS OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors acknowledge the use of ChatGPT to support language improvement, improve clarity, and improve the overall readability of the manuscript.

FUNDING

This research was supported by the Ministry of Higher Education, Science, and Technology, Republic of Indonesia, under the BIMA Grant Program 2023, number: 0557/E5.5/AL.04/2023.

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