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Comparative Study of the Nutritional Status of Children Aged 1-6 Years with Unoperated Congenital Heart Diseases in a Tertiary Hospital in Benin-City

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Abstract

Background: Children with congenital heart disease (CHD) are at increased risk of malnutrition due to elevated metabolic demands, feeding difficulties and recurrent infections. Malnutrition adversely affects morbidity, surgical outcomes and survival, yet data from Nigeria and West Africa remain limited.

Objective: To assess the nutritional status of children with CHD and examine its association with socio-demographic characteristics, anthropometric indices and types of cardiac lesions

Methods: A hospital- based comparative cross- sectional study was conducted among 100 children with echocardiographically confirmed CHD (cases) and 60 apparently healthy age- and sex matched controls. Socio-demographic data were collected using a structured questionnaire. Anthropometric measurements were obtained using World Health Organisation growth standards.

Results: Most participants were toddlers (55 % of the cases and 60 % of the controls), and over half of the CHD cases were diagnosed during infancy. Acyanotic CHD predominated (70%), with ventricular septal defect being the most common lesion. Although the majority of children with CHD were well nourished, 24% were acutely malnourished. Wasting and underweight were significantly associated with CHD occurrence ($p < 0.001$), whereas stunting showed no significant association. No significant relationship was observed between CHD category and gender, birth order, family size or nutritional status.

Conclusion: Children with unoperated CHD have a significantly higher burden of wasting and underweight compared with healthy peers. Integrating routine nutritional screening and targeted nutritional intervention into cardiac care pathways may improve growth outcomes in resource-limited settings.

Keywords: Acyanotic Congenital Heart Disease, Anthropometry, Underweight, Wasting.

Introduction

Congenital heart disease (CHD) is defined as a gross structural abnormality of the heart or intrathoracic great vessels present at birth that is actually or potentially of functional significance.[1] CHD is the most common birth defect and accounts for a third of all major congenital abnormalities in the world.[2] The cardiovascular anomalies are frequently the result of defective morphogenesis that occurs during the embryological development. The malformations may be limited to the cardiovascular system (non-syndromic) or occur in association with anomalies of other systems as part of defined syndromes (syndromic).[3] They are a diverse group of disorders with varying aetiology, symptomatology, severity and outcome.[4]

CHD is among the causes of morbidity and mortality in children. Globally, the prevalence of congenital heart diseases is estimated to be 10-12 per 1000 live births and represents approximately 1.35 million live births each year. Reported figures vary worldwide, and a meta-analysis by van der Linde found that the highest CHD birth prevalence was in Asia, 9.3 per 1000 live births and the lowest in Africa, 1.9 per 1000 live births.[4] In Nigeria, Sadoh *et al.*, Ekure *et al.*, Chinawa *et al.*, and Otaigbe *et al.* documented prevalence of 14.4, 6.6, 2.2, 3.5 per 1000 in Benin, Lagos, Enugu, Port-Harcourt, Nigeria, respectively.[5 - 8]

Children with CHD are particularly vulnerable to impaired growth and malnutrition due to the combined effects of increase metabolic demand, reduced cardiac output, chronic hypoxaemia, recurrent infections and feeding difficulties.[9,10] Malnutrition in this population may manifest as stunting, wasting, underweight or deficiencies in macro- and micronutrients, all of which can exacerbate morbidity, increase susceptibility to infections and reduce survival.[11,12] Studies have consistently demonstrated that the severity of cardiac lesion, presence of cyanosis and congestive heart failure are strongly associated with the degree of nutritional compromise in affected children.[13,14]

In children with CHD, malnutrition can lead to delayed somatic growth, poor surgical outcomes, prolonged hospital stays and impaired functional status.[15] Nutritional deficits are further compounded by increased energy expenditure due to tachycardia and heightened respiratory effort as well as reduced caloric intake from early satiety, difficulty feeding or fatigue.[16,17] Micronutrient deficiencies, particularly of iron, zinc and vitamins A and D are commonly reported and can exacerbate anaemia, delay wound healing and impair immune responses.[18,19]

Comparative studies examining nutritional status in children with CHD and age- matched healthy peers are essential to quantify the burden of malnutrition, identify at-risk subgroups and inform targeted interventions.[20] Previous research in low- and middle- income countries including Nigeria has shown that children with CHD often have significantly lower weight-for-age, height-for-age and body mass index compared to apparently healthy controls.[20,21] Factors influencing these outcomes include type and severity of cardiac lesions, socioeconomic status, age at diagnosis, access to corrective surgery and adherence to dietary recommendations.[22,23] Early identification of growth deficits and timely nutritional intervention are critical in children with CHD particularly in the 1-6 year age group, a period of rapid growth and development.[24] Understanding the patterns and determinants of malnutrition in this population is essential for both preventive and therapeutic strategies, including individualised feeding plans, micronutrient supplementation and preoperative nutritional optimization.[25]

This study utilised a comparative assessment of nutritional status in children aged 1-6 years with unoperated CHD and apparently healthy peers. The findings from this research are expected to provide evidence for targeted nutritional interventions, inform policy on the care of children with CHD and ultimately contribute to improved growth, functional outcomes and quality of life in this vulnerable population. The study aimed to assess and compare the nutritional status of children with unoperated congenital heart diseases to their healthy counterparts and identify clinical and sociodemographic factors associated with nutritional status in children with CHD.

Methods

Study location

This study was conducted at the Paediatric Cardiology Clinic of the University of Benin Teaching Hospital and the Children Emergency Ward/Immunization Clinics.

Study design

This was a descriptive, cross-sectional study. The controls were frequency-matched to cases by age category (toddler, preschool, school age) and sex. Individual pairing was not performed.

Ethical considerations

Ethical approval was obtained from the hospital's Research Ethics Committee (NHREC-UBTH-HREC/24/12/2022B).

Informed written consent was obtained from the parents/caregivers before enrolment into the study.

Study population

Children aged 1 to 6 years with unrepaired congenital heart defects (this includes both acyanotic and cyanotic congenital heart diseases) such as atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), Tetralogy of Fallot (TOF), Transposition of Great Arteries (TGA) with a 2D-Echocardiographic diagnosis were included into the study with their age- and sex-matched controls.

Exclusion criteria

Subjects and controls with genetic disorders, other chronic illnesses, and those who were delivered preterm were excluded.

Sample size determination

The minimum sample size was calculated using the formula for comparison of two proportions in case–control studies. Using a previous Nigerian study which reported a prevalence of underweight of 45% among children with CHD and 20% among controls,[26] with a power of 80% and alpha of 0.05, the minimum required sample size was 54 participants per group. To increase the study power and compensate for possible incomplete data, 100 cases aged 1-6 years were recruited. Sixty age- and sex-matched controls were enrolled, giving a total sample size of 160 participants. They were recruited consecutively from the Paediatric Cardiology Clinic and Children Emergency Ward of the University of Benin Teaching Hospital, Benin City, Edo State, Nigeria. Post-hoc power analysis using the observed prevalence of malnutrition in this study (24% vs 0%) showed a study power greater than 90%.

Although a 1:1 ratio is statistically efficient, increasing the number of cases improves the precision of estimates within the exposed group. The 100:60 (1.67:1) case–control ratio resulted from consecutive recruitment of all eligible CHD cases during the study period, while the controls were recruited from children attending the immunisation clinic for routine wellness visits. These children were apparently healthy, not acutely ill at the time of recruitment, and had no known chronic medical conditions.

Data collection methods

Two resident doctors in paediatrics in the hospital served as research assistants and were trained in the relevant aspects of the methodology. Data were collected using a structured data inquiry sheet developed by the primary investigator specifically for this study. The data included demographic characteristics, including age, gender, age at diagnosis, social status of the parents and other data, including cardiac diagnosis at echocardiography, anthropometric measurements: weight, height/length, MUAC, occipitofrontal circumference**.** Nutritional indices were assessed using WHO growth standards (Z scores) of the anthropometric measurements, and malnutrition was classified as severe or moderate and normal nutrition. [27]

Definition of composite nutritional status variables

Nutritional status was classified as "malnourished" if a child had any of the following:

(i) weight-for-height Z-score < −2 (wasting),
(ii) weight-for-age Z-score < −2 (underweight), or
(iii) height-for-age Z-score < −2 (stunting).

Children without any of these abnormalities were classified as "well-nourished.

Anthropometry

The body weight was measured using a weighing scale (model 769, Seca, Hamburg, Germany). The scale was calibrated on each clinic day using a known weight. The scale was placed on the solid floor before use and adjusted to zero. The participants were instructed to stand gently with light clothing and barefoot. The body weight was recorded to the nearest 0.1kg. Height was measured using a calibrated stadiometer (model 769, Seca, Hamburg, Germany) with an adjustable headpiece according to standard procedures. The occipitofrontal and mid-arm circumference were measured in centimetres, using a flexible but non-stretchable measuring tape, using reference anatomical landmarks and standard protocols.

Data processing and analysis

The data were entered into the Excel spreadsheet, and IBM Statistical Package for Social Sciences (SPSS) version 26.0 was used to analyse the data. Quantitative variables with a normal distribution were summarised using the mean and standard deviation, while skewed variables were summarised using the median and interquartile ranges. For categorical variables, percentages and frequencies were generated. Comparative analysis was done using the Chi-Square and Fisher's Exact tests were used to compare percentages of categorical variables. Mann–Whitney U test was used for the comparison of non-parametric variables. Binary logistic regression was performed to identify predictors of malnutrition. A p-value of <0.05 was considered statistically significant.

Results

Sociodemographic characteristics

Table I compares the sociodemographic characteristics of children with CHD (cases) and the controls. There was no statistically significant difference in the mean age between the cases and controls. The mean age of the cases was 34.06 ± 19.78 months, while that of controls was 32.17 ± 16.60 months ($t = 0.65$, $p = 0.516$). Similarly, the distribution of age categories (toddler, preschool, school age) did not differ significantly between cases and controls ($\chi^2 = 0.91$, $p = 0.634$). More than half of both groups were toddlers (55.0% of cases and 60.0% of controls), further demonstrating age comparability.

Although a higher proportion of the controls were males (56.7%) compared to the cases (40.0%), the difference was not statistically significant ($\chi^2 = 3.41$, $p = 0.065$). There was also no statistically significant difference between the cases and the controls in terms of birth weight distributions ($\chi^2 = 5.63$, $p = 0.060$). Low birth weight was insignificantly more frequent among the cases (16.0%) than the controls (3.3%). Other variables such as age at diagnosis, birth order, and family size were reported only for the cases and therefore were not compared statistically. Among the cases, the clinical diagnosis was frequently made during infancy (53.0%), and the majority belonged to medium-sized families (4-6 members) (54.0%).

Anthropometric parameters and nutritional status

Children with CHD had significantly lower mean weight ($p = 0.002$), MUAC ($p < 0.001$), and head circumference ($p = 0.018$) compared with controls, while mean height did not differ significantly ($p = 0.213$) (Table IIa). There was a statistically significant difference in weight-for-height distribution between CHD cases and controls ($\chi^2 = 14.84$, $p < 0.001$), with wasting present in 34% of cases compared to 6.7% of controls. Similarly, weight-for-age differed significantly ($\chi^2 = 16.67$, $p < 0.001$); 36% of cases were underweight compared to 6.6% of controls. However, the height-for-age distribution was comparable ($\chi^2 = 1.41$, $p = 0.494$) (Table IIb).

Table I: Comparison of sociodemographic characteristics between cases and controls

Variable	Cases (n = 100)	Controls (n = 60)	Statistics	p-value
Age (months) (Mean ± SD)	34.06 ± 19.78	32.17 ± 16.60	t = 0.65	0.516
Age Category				
Toddler	55 (55.0)	36 (60.0)	$\chi^2 = 0.91$	0.634
Preschool	26 (26.0)	16 (26.7)		
School age	19 (19.0)	8 (13.3)		
Sex				
Male	40 (40.0)	34 (56.7)	$\chi^2 = 3.41$	0.065
Female	60 (60.0)	26 (43.3)		
Birth Weight Category				
Low	16 (16.0)	2 (3.3)	$\chi^2 = 5.63$	0.06
Normal	78 (78.0)	54 (90.0)		
High	6 (6.0)	4 (6.7)		
Age at diagnosis				
Birth	10 (10.0)			
Neonatal period	12 (12.0)			
Infancy	53 (53.0)			
Toddler	20 (20.0)			
Preschool age	5 (5.0)			
Birth order				
First	31 (31.0)			
Second	28 (28.0)			
Third	19 (19.0)			
Fourth	15 (15.0)			
Fifth	4 (4.0)			
Sixth	3 (3.0)			
Family size				
Medium	54 (54.0)			
Large	46 (46.0)			

Proportion of each type of congenital heart lesion among the cases

Seventy per cent (70.0%) of the children had acyanotic congenital heart disease, while thirty per cent (30.0%) had cyanotic congenital heart disease. Taken together as they occur (combined), Tetralogy of Fallot (TOF), ventricular septal defect (VSD), complete atrioventricular septal defect (complete AVSD), and atrioventricular septal defect (ASD) accounted for the greater proportions (28%, 23%, 12%, and 10%, respectively) of the cases (Table III).

Comparison of Nutritional Status between Cases and Controls

Acute malnutrition (severe or moderate) was present in 24.0% of children with congenital heart disease, whereas none of the controls was malnourished ($p < 0.001$). All the children in the control group (100%) were well nourished compared to 76.0% of cases ($p < 0.001$) (Table IV).

Table IIa: Comparison of anthropometric measurements between cases and controls

Variable	Cases (n=100) Mean ± SD	Controls (n=60) Mean ± SD	t-value	p-value
Weight (kg)	11.56 ± 4.04	14.09 ± 5.52	-3.21	**0.002**
Height (cm)	87.87 ± 14.42	90.28 ± 13.14	-1.25	0.213
MUAC (cm)	13.64 ± 1.80	19.36 ± 2.65	-14.52	**<0.001**
Head Circumference (cm)	47.50 ± 3.70	49.10 ± 3.20	-2.41	**0.018**

Table IIb: Comparison of nutritional status indicators between cases and controls

Weight for height	**Frequency (%)**	**Frequency (%)**	**χ^2**	**p**
Severe wasting	19 (19.0)	0 (0	14.84	**< 0.001**
Wasted	15 (15.0)	4 (6.7)		
Normal	58 (58.0)	49 (81.6)		
Overweight	7 (7.0)	3 (5.0)		
Obese	1 (1.0)	4 (6.7)		
Weight for age	**Frequency (%)**	**Frequency (%)**	χ^2	**p**
Severe underweight	24 (24.0)	2 (3.3)	16.67	**< 0.001**
Underweight	12 (12.0)	2 (3.3)		
Normal	64 (64.0	56 (93.4)		
Height for age	**Frequency (%)**	**Frequency (%)**	χ^2	**p**
Severe stunting	17 (17.0)	5 (8.3)	1.41	**0.494**
Stunted	16 (16.0)	6 (10.0)		
Normal	67 (67.0)	49 (81.7)		

Association between anthropometric measures and CHD occurrence.

All (100%) the cases who had severe wasting also had CHD (χ^2 = 17.272; p < 0.001). Likewise, the greater proportion (92.3%) of the cases who had severe underweight also had CHD (χ^2 = 17.378; p < 0.001). The greater proportion (77.3%) of the cases who had severe stunting also had CHD. However, the association between height for age and CHD was not statistically significant (χ^2 = 4.143, p = 0.126) (Table V).

Association of nutritional status with CHD status among total participants.

The greater proportion (100%) of children who did not have CHD were well-nourished. Association between nutritional status and CHD status was found to be statistically significant (χ^2 = 16.941, p < 0.001) (Table VI).

Association of nutritional status with socio-demographic characteristics and category of CHD.

The greater proportion (100.0%) of school-age children with CHD were well-nourished. The association of age category of children who have CHD with nutritional status was statistically significant (χ^2 = 14.278, p < 0.001). The greater proportion (82.5%) of male children who had CHD were well-nourished. However, the association of gender of children who had CHD with nutritional status was not statistically significant (χ^2 = 1.544, p = 0.241). The greater proportion (78.2%) of children who had CHD diagnosed during infancy or later were well-nourished, but the association of age of diagnosis with nutritional status was insignificant (χ^2 = 0.945, p = 0.398). The greater proportion (85.4%) of first or second children who had CHD were well-nourished, whereas there was no statistical significance in the

association of birth order with nutritional status among children with CHD (χ^2 = 3.342, p = 0.095). The greater proportion (82.6%) of children with CHD from large families was well-nourished but without statistical significance (χ^2 = 2.040, p = 0.168). The greater proportion (80.0%) of children who had a cyanotic CHD were well-nourished but without statistical significance (χ^2 = 0.376, p = 0.617) (Table VII).

Table III: Proportion of each type of congenital heart lesion among the cases

Variable	Frequency (n = 100)	Percentage
Category of heart lesion		
Acyanotic	70	70
Cyanotic	30	30
Type of heart lesion		
TOF	28	28
VSD	23	23
Complete AVSD	12	12
ASD	10	10
ASD, VSD	7	7
VSD, TR	5	5
PDA	4	4
ASD, PDA	3	3
Pulmonary. stenosis	2	2
TGA	2	2
Truncus arteriosus	2	2
Single atrium,	1	1
Univentricle	1	1

Key: Provide keys to the abbreviations

Table VIII shows the multivariable logistic regression analysis of factors associated with malnutrition among children with congenital heart disease after adjusting for potential confounders. Toddlers were about three times more likely to be malnourished compared to older children (Adjusted OR = 3.12; 95% CI: 1.28–7.62; p = 0.012). Children with a history of low birth weight had approximately 2.4 times higher odds of being malnourished compared to those with normal birth weight (Adjusted OR = 2.41; 95% CI: 1.01–5.77; p = 0.048). However, the type of congenital heart disease (cyanotic versus acyanotic) was not significantly associated with malnutrition after adjustment (Adjusted OR = 1.21; 95% CI: 0.52–2.83; p = 0.657). Overall, toddler age and low birth weight emerged as independent predictors of malnutrition among children with congenital heart disease in this study.

Discussion

This study demonstrated that most of both cases and controls were toddlers. The predominance of females among the cases contrasts with several Nigerian studies that reported a slight male preponderance in CHD, [28, 29] although others have documented no consistent sex difference. [30] This variation may reflect differences in healthcare-seeking behaviour, referral patterns or survival bias. More than half of the children with CHD in this study were diagnosed during infancy, aligning with findings from Lagos and Enugu, where infancy remains the peak period for CHD diagnosis due to increased clinical suspicion during routine immunisation visits and recurrent illness. [31,32]

The high proportion of normal birth weight among cases is similar to observations by Sadoh *et al.* and Animasahun *et al.*, suggesting that intrauterine growth restriction is a universal feature of CHD in Nigerian children and that postnatal factors may play a larger role in growth faltering. [28,31]

Table IV: Comparison of nutritional status between cases and controls

Nutritional Status	Cases (n = 100) n (%)	Controls (n = 60) n (%)	Test	p-value
Severe acute malnutrition	11 (11.0)	0 (0.0)	Fisher's Exact	**<0.001**
Moderate acute malnutrition	13 (13.0)	0 (0.0)		
Well nourished	76 (76.0)	60 (100.0)		

Table V: Association between anthropometric measures with CHD occurrence

Variable	CHD status		χ^2 or Fisher's exact statistic	p-value
	Present	Absent		
Weight for height				
Severe wasting	19 (100%)	0 (0)	17.272	< 0.001
Wasted	15 (78.9%)	4 (21.1%)		
Normal	58 (54.2%)	49 (45.8%)		
Overweight/Obese	8 (53.3%)	7 (46.7%)		
Weight for age				
Severe underweight	24 (92.3%)	2 (7.7%)	17.378	< 0.001
Underweight	12 (85.7%)	2 (14.3%)		
Normal	64 (53.3%)	56 (46.7%)		
Height for age				
Severe stunting	17 (77.3%)	5 (22.7%)	4.143	0.126
Stunted	16 (72.7%)	6 (27.3%)		
Normal	67 (57.8%)	49 (42.2%)		

Table VI: Association of nutritional status with CHD status among total participants

CHD status	Nutritional status		χ^2 or Fisher's exact statistic	P-value
	Malnourished	Malnourished		
Present	24 (24.0%)	76 (76.0%)	16.941	< 0.001
Absent	0 (0)	60 (100.0%)		

Children with CHD in this study had significantly poorer anthropometric indices compared with controls. The finding that no school-aged child with CHD was malnourished is noteworthy and may reflect survivor bias. In resource-limited settings, children with severe CHD and significant malnutrition may have higher early mortality, resulting in a relatively healthier cohort surviving into school age. This phenomenon has been described in similar low-income settings and should be considered when interpreting age-specific nutritional patterns.

Although the majority of the cases were classified as well-nourished, a substantial proportion exhibited wasting, underweight and stunting. Similar patterns have been reported in

Nigeria and Ghana, where children with CHD consistently demonstrate lower weight-for-age and weight-for-height z-scores compared with apparently healthy peers. [29,30,32]

Table VII: Association of nutritional status with socio-demographic characteristics and CHD category

Variables	Nutritional status		χ^2 or Fisher's Exact Test	P-value
	Malnourished	Well-nourished		
Age category				
Toddler	21 (38.2%)	34 (61.8%)	14.278	**< 0.001***
Preschool	3 (11.5%)	23 (88.5%)		
School age	0 (0)	19 (100%)		
Gender				
Male	7 (17.5%)	33 (82.5%)	1.544	0.241
Female	17 (28.3%)	43 (71.7%)		
Age at diagnosis				
Before infancy	7 (31.8%)	15 (68.2%)	0.945	0.398
Infancy or later	17 (21.8%)	61 (78.2%)		
Birth order				
First or second	18 (30.5%)	41 (69.5%)	3.342	0.095
Third or lower	6 (14.6%)	35 (85.4%)		
Family size				
Medium	16 (29.6%)	38 (70.4%)	2.04	0.168
Large	8 (17.4%)	38 (82.6%)		
Category of CHD				
Acyanotic	18 (25.7%)	52 (74.3%)	0.376	0.617
Cyanotic	6 (20.0%)	24 (80.0%)		

Table VIII: Multivariable logistic regression analysis of predictors of malnutrition among children with CHD

Variable	Adjusted OR	95% CI	p-value
Toddler age	3.12	1.28–7.62	0.012
Cyanotic CHD	1.21	0.52–2.83	0.657
Low birth weight	2.41	1.01–5.77	0.048

The higher prevalence of wasting and underweight among cases supports existing evidence that increased metabolic demands, feeding difficulties, recurrent infections and reduced caloric intake contribute significantly to acute and chronic malnutrition in children with CHD.[33] The relatively lower prevalence of stunting and the lack of association between height-for-age and CHD in study may indicate that linear growth retardation is less sensitive to short- term nutritional insults and may require prolonged exposure to adverse conditions before becoming apparent, as previously suggested by Nigerian and Senegalese studies.[30,34]

Acyanotic lesions accounted for 70% of CHD cases, which is consistent with numerous Nigerian and West African studies reporting acyanotic disease as the predominant category of CHD. [28,29,35] Ventricular septal defect (VSD) emerged as the most common lesion, followed

by Tetralogy of Fallot (TOF) and atrial septal defect (ASD). This pattern mirrors findings from tertiary centres in Benin City, Ibadan, Lagos and Accra, where VSD consistently represents the most frequent CHD subtype.[28,30,32] The relatively high proportion of TOF among cyanotic lesions aligns with previous reports indicating TOF as the most common cyanotic CHD in West Africa, likely reflecting better survival and referral of TOF compared with more complex cyanotic defects. [29,34]

Although most children with CHD were classified as well-nourished, nearly one-quarter were acutely malnourished, a finding comparable to reports from Cameroon and other parts of Nigeria, where malnutrition rates among children with CHD ranged from 20% to 45% depending on severity and age at diagnosis. [28,34] The absence of malnutrition among the controls underscores the strong association between CHD and poor nutritional outcomes, reinforcing the role of CHD as a significant contributor to childhood malnutrition in low-resource settings. This study found an association between nutritional status and CHD status among the study participants. All children without CHD were well nourished whereas a substantial proportion of children with CHD were malnourished. This finding highlights the strong relationship between the presence of CHD and poor nutritional status in affected children. The high prevalence of malnutrition among children with CHD observed in this study is consistent with findings by Chinawa *et al.*[20] in Enugu and Okoromah *et al.* [26] in Lagos, who reported underweight and stunting being common findings. Similarly, Olatunya *et al.* [36] in Ado Ekiti, reported a high burden of malnutrition among children with incorrected CHD, attributing this to increased metabolic demands and recurrent illnesses. The observed association can be explained by pathophysiological mechanisms. Children with CHD often experience increased energy expenditure due to chronic hypoxia, tachypnea and heart failure. Additionally, poor feeding, early satiety, recurrent respiratory infections and frequent hospitalisations further compromise nutritional intake.

The present study found an association between CHD and both wasting and underweight but not with stunting. Similar associations have been reported by Okoromah *et al.* and Animasahun *et al.* in Lagos, where children with CHD were significantly more likely to be wasted or underweight than the controls. [26,31] These findings suggest that CHD exerts a stronger influence on acute nutritional status than on long-term linear growth, particularly in settings where diagnosis and intervention occur relatively early. The lack of significant association between CHD (acyanotic versus cyanotic) and gender, birth order, family size and nutritional status in this study is consistent with the findings from Nigeria and Ghana. [30,33] Although cyanotic lesions are often presumed to confer greater nutritional risk, several studies have shown that malnutrition is influenced more by heart failure severity, recurrent infections and feeding practices than by the lesion type alone. [33,37]

In the present study, toddler age was significantly associated with malnutrition, as toddlers were observed to be three times more likely to be malnourished compared to older children. This finding may be explained by the increased nutritional demands during the toddling period, which coincides with rapid growth and development. Children with CHD often have increased metabolic demands and reduced appetite, making them more vulnerable to malnutrition during this stage of life. Similar findings have been reported in several studies conducted in developing countries where younger children with CHD were more prone to undernutrition due to delayed diagnosis, recurrent infections and inadequate nutritional support.[26,38,39] A study by Vaidyanathan *et al.* also noted that younger age groups were particularly vulnerable to growth failure in the presence of CHD.[40] Low birth weight also

emerged as a significant independent predictor of malnutrition in children with CHD. Children with a history of low birth weight had approximately 2.4 times higher odds of being malnourished compared with those who had normal birth weight. Low birth weight is known to predispose children to growth faltering due to reduced nutrient stores at birth and impaired postnatal growth potential. In children with CHD, this vulnerability may further be compounded by chronic hypoxia, increased energy expenditure and recurrent illness.

Previous studies in Nigeria and other African countries have reported similar associations between low birth weight and poor nutritional outcomes among children with CHD. [11,26,40] These findings highlight the importance of early nutritional monitoring and intervention in infants born with low birth weight who also have CHD. However, the type of CHD (cyanotic versus acyanotic) was not significantly associated with malnutrition after adjusting for other factors. Although cyanotic CHD is traditionally associated with more severe nutritional compromise due to chronic hypoxaemia and increased metabolic demands, several studies have reported no significant difference in the prevalence of malnutrition between cyanotic and acyanotic CHD after controlling for other variables. [34,38,40] This suggests that factors such as feeding difficulties, recurrent infections, delayed surgical corrections and socioeconomic conditions may play a more important role in determining nutritional status than the anatomical type of heart defect alone.

Limitations

The cross-sectional design limits causal inference between CHD and malnutrition. Being hospital-based, the study may over-represent more severe CHD cases while missing milder community-dwelling cases, thereby limiting generalisability. Additionally, feeding history and detailed dietary intake were not quantitatively assessed in the study.

Conclusion

This study demonstrated that congenital heart diseases remain an important contributor to childhood undernutrition in the study population. Increased metabolic demand, feeding difficulties and recurrent infections likely play a more prominent role in malnutrition among children with CHD than lesion category alone. Regular nutritional assessment using standardised anthropometric indices should be incorporated into routine clinical management of all children with CHD, especially during infancy and early childhood. Children with CHD identified with wasting or underweight should receive prompt nutritional support, including individualised feeding plans, caregiver counselling and, where necessary, therapeutic feeding programs to prevent progression to severe malnutrition.

Authors' Contributions: EL conceived the study, while EL, NB and EV designed the study. NB did the literature review. EL, NB and EV did data analysis and interpretation. EL drafted the manuscript. All the authors revised the draft for sound intellectual content and approved the final version of the manuscript.

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