



Journal Homepage: -[www.journalijar.com](http://www.journalijar.com)

## INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/16094  
DOI URL: <http://dx.doi.org/10.21474/IJAR01/16094>



### RESEARCH ARTICLE

#### PREVALENCE, PREDICTORS AND PROFILE OF MALNUTRITION IN CHILDREN WITH CONGENITAL HEART DEFECTS

Ummer Mohd, Prof. Khurshid Ahmad Wani, Amber Bashir, Anzeen Kanth and Prof. Muzaffar Jan  
Department of Paediatrics, GMC Srinagar.

#### Manuscript Info

##### Manuscript History

Received: 28 November 2022

Final Accepted: 30 December 2022

Published: January 2023

##### Keywords: -

Congenital Heart Diseases,  
Malnourishment, Paediatrics, Cyanotic  
Heart Diseases

#### Abstract

**Background:** Malnutrition in children with congenital heart defects [CHDs] has been linked to increased morbidity and mortality as indicated by frequent hospitalisation, poor surgical outcomes, persistent impairment of somatic growth and increased death. All types of malnutrition, and severe forms in particular, contribute to mortality in childhood. There is strong evidence that poor growth [ malnutrition] is associated with delayed mental development and poor school performance. The mechanisms for growth deficiency in CHD are multifactorial and include associated chromosomal anomalies/ genetic syndromes, inadequate nutritional intake due to feeding difficulties, and poor absorption of nutrients from digestive tract in chronic congestive heart failure. Also increased calories are required to sustain the increased myocardial, respiratory and neuro-humoral functions in CHD-related heart failure. Chronic CHF and chronic hypoxemia in CHD impair cellular metabolism and cell growth. Other mechanisms of growth deficiency in CHD have been reported. The WHO recommends the WHO/National Center for Health Statistics [NCHS] growth standards for nutrition surveys. The present study aims to describe the prevalence, profile and predictors of malnutrition using recommended case definitions. The findings of present study could be applied to current and future paediatric cardiac care practice.

**Aims And Objectives:** To investigate the prevalence, profile, predictors and epidemiological pattern of malnutrition in children with congenital heart defects.

**Materials And Methods:** This was a Case-control, observational study which was conducted in post graduate department of paediatrics GB Pant Children Hospital on all patients above 6 months of age admitted in the hospital as cases of congenital heart defects and controls will include the patients with minor ailments admitted for short stay. Study was conducted between December 2020-November 2022

**Statistical Analysis:** Data was entered in a Microsoft Excel spreadsheet. Categorical variables were summarized as frequency and percentage. Continuous variables were summarized as mean and SD.

**Results:** The study was conducted on two hundred and fifty cases of CHD. The detected cardiac anomalies were classified as acyanotic heart diseases (n=163; 65%) and cyanotic heart diseases (n=87; 35%). The prevalence of CHD-related malnutrition was 90% as compared to

21.2% in the control patients with 63.3% of cases having severe malnutrition. Among cases, the relative proportions of wasting, stunting and underweight were 40.8%, 28.8% and 20.8%, respectively. Wasting was proportionately higher (58.3%) in acyanotic CHD, while stunting was predominant (67.8%) in cyanotic CHD ( $p=0.0001$ ).

Copy Right, IJAR, 2023,. All rights reserved.

## Introduction:-

Children with congenital heart diseases may become easily malnourished due to multiple factors such as effects of the cardiac lesion, low energy intake, hyper metabolism, age at the time of corrective/palliative procedure, and other prenatal factors.<sup>1</sup> At birth, infants with CHD are usually of normal weight-for-gestational age. However, nutrition and growth problems often become evident very early in their lives.<sup>2</sup> Cardiac malformations are undoubtedly responsible for malnutrition, which may range from mild under-nutrition to severe failure to thrive. Malnutrition may then significantly undermine the outcome of corrective surgical operations and postoperative recovery. Mechanisms linking CHD to malnutrition may be related either to decreased energy intake and/or to increased energy requirements. Decreased energy intake can involve deficiencies of specific nutrients, or insufficient total caloric intake. Increased respiratory rate accompanying congestive heart failure may be responsible for increased energy requirements.<sup>1</sup> The incidence of malnutrition in infants with cardiovascular diseases (CVDs) depends on several factors, including:

- 1) Insufficient nutrient intake caused by anorexia due to side effects of medications, fatigue during feeding, swallowing problems (dysphagia) such as uncoordinated breast sucking, neurological dysfunction, laryngeal dysfunction, nausea and reflux, early satiety due to the reduced gastric volume caused by hepatomegaly or ascites, tachypnoea, observing fluid intake restrictions, NPO courses in hospitals for performing therapeutic processes, and recurrent respiratory infections<sup>3,4</sup>;
- 2) Increased energy requirement caused by severe metabolic stress responses in CHF, postoperative metabolic stress, relative increase in fat-free mass to fat mass ratio in malnourished patients, tachypnoea, tachycardia, cardiac hypertrophy, polycythaemia for chronic hypoxia compensation, increased activity of sympathetic system, infections, fever, and sepsis<sup>3,4</sup>
- 3) Malabsorption of nutrients due to nausea, decreased splanchnic blood flow, poor gastric emptying, intestinal motility changes, intestinal wall edema (due to right-sided heart failure) resulting in malabsorption, considerable excretion of nutrients such as protein-losing enteropathy and steatorrhea, excretion of trace elements such as calcium and potassium, intestinal mucosal atrophy causing malabsorption, and lack of insulin-like growth factor (IGF1)<sup>3,4</sup>.

**Table A:- Mechanisms of Malnutrition in Children with CHD.**

1. Type of cardiac lesion (cyanotic versus acyanotic malformations)
2. Low energy intake
Direct: Loss of appetite
Indirect: Anoxia and peripheral acidosis
Malabsorption
Relative increased nutrient requirements
3. Hypermetabolism
Multiple infective episodes
Increased oxygen consumption
Increased basal body temperature
Low body fat stores
4. Age at time of operation
5. Prenatal factors
6. Associated genetic factors

Typically, these children need 50% more calorie intake than their healthy peers.

In developed countries, development in paediatric cardiac care, early prenatal and postnatal diagnosis, and supportive and timely corrective interventions for cardiac lesions have almost eliminated the impact of CHD on nutritional status.

The present study aims to describe the prevalence, profile and predictors of malnutrition using recommended case definitions.

The findings of present study could be applied to current and future paediatric cardiac care practice.

**Aims And Objectives:-**

To investigate the prevalence, profile, predictors and epidemiological pattern of malnutrition in children with congenital heart defects.

**Materials And Methods:-****Study Design:**

Case-control, observational study

**Study Setting:**

The study was conducted in the post graduate department of paediatrics, GB Pant Children Hospital, associated with Government Medical College Srinagar, on all patients above 6 months of age admitted to the hospital as cases of congenital heart defects; and controls included the patients with minor ailments admitted for short stay.

**Study Duration:**

December 2020-November 2022

**Participants:**

Children between 6 months to 18 years of age.

**Inclusion Criteria****Cases:**

Age group 6 months to 18 years  
Children with CHD.

**Controls:**

Age and Sex matched patients with minor ailments with no history of any chronic disease like TB, chronic diarrhoea, prolonged fever etc. admitted for short stay.

Short stay = Patients with a range of low to moderate risk symptoms who with optimal diagnostic and clinical management, discharged in less than 24 hours.

**Exclusion Criteria**

1. Infants less than 6 months of age.
2. Children with corrective Congenital Heart Defects.
3. Additional morbidities like genetic or chromosomal anomalies.

**Statistical Analysis:**

Data was entered in a Microsoft Excel spreadsheet. Categorical variables were summarized as frequency and percentage. Continuous variables were summarized as mean and SD.

**Financial Issue.**

No financial issues

**Ethical Issues:**

Ethical clearance taken from the institutional ethical committee and consent taken from the parents/ legal guardians of the participants.

**Results:-**

The study was conducted on two hundred and fifty cases of CHD with equal number of controls taken from the patients that reported to our department. The number of males and females in case subjects was 135 and 115

respectively and for control subjects was 130 and 120 respectively. The average age for the case subjects was 42 months and for control subjects was 44 months, with an age range of 06-216 months. The maximum number of cases fell in the age group of 06- 60 months. The detected cardiac anomalies were classified as acyanotic heart diseases (n=163; 65%) and cyanotic heart diseases (n=87; 35%), with details given in table 1.

**Table 1:-**

Type of cardiac defects	Number (%)
<b>Acyanotic group(n=163)</b>	
Ventricular septal defect (VSD)	79 (31.6%)
Patent ductus arteriosus (PDA)	34(13.6%)
Atrial septal defect (ASD)	20 (8%)
VSD+ASD	11(4.4%)
VSD+PDA	9(3.6%)
Mitral stenosis (MS)+ASD	4 (1.6%)
Partial anomalous pulmonary venous return (PAPVR)	3(1.2%)
Pulmonary stenosis (PS) without VSD	3(1.2%)
<b>Cyanotic group(n=87)</b>	
Tetralogy of Fallot (TOF)	37 (14.8%)
Transposition of the great arteries (TGA)+VSD without PS	10(4%)
Transposition of the great arteries (TGA)+ASD+VSD+PDA without PS	8(3.2%)
Single atrium	7 (2.8%)
Single ventricle with PS	7 (2.8%)
Single ventricle without PS	6 (2.4%)
Tricuspid atresia+ASD	5 (2%)
Persistent truncus arteriosus+VSD	4 (1.6%)
Double outlet right ventricle+VSD	3 (1.2%)

The socioeconomic status of the study subjects was categorised using the Modified Kuppuswamy Scale. The maximum number of cases and control subjects belonged to the upper lower class. There was no significant difference between the two groups, as depicted in table (2).

**Table 2:- Socio-Economic Status.**

MODIFIED KUPPUSWAMY SCALE CLASS	CASES (%)	CONTROLS (%)
I	0	0
II	5(2)	6(2.4)
III	82(32.8)	76(30.4)

IV	147(58.8)	155(62)
V	16(6.4)	13(5.2)

In this study, the main study groups (cases and controls) were compared for height, weight, BMI, gestational age, birth order, and birth weight. The study reported statistically significant difference (P-value <0.05) in respects to weight, height and body mass index between the two groups. However, the difference was not statistically significant between the two groups in respects to gestational age, birth order and birth weight. The prevalence of CHD-related malnutrition was 90% as compared to 21.2% in the control patients with 63.3% of cases having severe malnutrition. Among cases, the relative proportions of wasting, stunting and underweight were 40.8%, 28.8% and 20.8%, respectively. Contrary to the usual distribution of growth deficiency in the general paediatric population as per the WHO<sup>8</sup> reports, wasting was the most prevalent type of malnutrition in our study, instead of underweight and stunting, and the prevalence of wasting in children with CHD was five times higher than the WHO national estimate for wasting in Nigeria.

Wasting was proportionately higher (58.3%) in acyanotic CHD, while stunting was predominant (67.8%) in cyanotic CHD (p=0.0001). The detailed description of various assessed parameters is depicted in following tables.

**Table 3:-** The baseline characteristics of the study population.

Parameter	Cases	Controls	P Value
Weight (kgs)	13.72±13	17.48±8.8	<0.001
Height (cms)	89.07±31.3	101.57±23.8	<0.001
BMI	14.72±3.3	15.96±2.5	<0.001
Gestational Age (wks)	39.5±0.86	39.4±0.88	0.573
Birth Order	3±1.29	3±1.6	0.656
Birth Weight (kgs)	3.1±0.77	3.3±0.59	0.319

**Table4:-** Distribution of malnutrition in study population.

Type of Nutrition	Cases	Control	P-value
Underweight (WAZ)	51(20.4%)	36(14.4%)	0.395
Wasting (WHZ)	102(40.8%)	7(2.8%)	0.0001
Stunting (HAZ)	72(28.8%)	10(4.0%)	0.0001

Table (4) presents distribution of malnutrition in study population. 225 of the 250 children with CHD were malnourished with a WAZ score of  $\leq -2$ , giving an overall prevalence of malnutrition of 90.0% in cases compared with 21.2% InControl's (p=0.0001).

**Table 5:-** Distribution of malnutrition amongst the cases.

Type of Nutrition	Acyanotic Group	Cyanotic Group	P-value
Underweight (WAZ)	41(25.1%)	7(8%)	0.067
Wasting (WHZ)	95(58.3%)	7(8%)	0.0001
Stunting (HAZ)	17(10.4%)	59(67.8%)	0.0001

Table (5) presents the prevalence of malnutrition. Wasting was proportionately higher (58.3%) in acyanotic CHD (p=0.0001), while stunting was predominant (67.8%) in cyanotic CHD (p=0.0001).

**Table 6:-** Multivariate logistic regression analysis of predictors of malnutrition in children with CHD.

Variable	OR (95% CI)	p Value
Haemoglobin level $\leq 10.0$ g/dl	6.51 (4.01 to 8.00)	<0.001
Congestive heart failure	4.20 (2.30 to 6.64)	<0.001
Modified Ross score $\geq 7$	4.34 (2.00 to 4.64)	<0.001
Low arterial oxygen saturation	4.15 (2.14 to 8.16)	<0.001
Type of CHD	2.53 (1.50 to 10.20)	<0.001

Duration of symptoms of CHD	3.33 (2.30 to 4.56)	<0.001
Poor dietary fat intake	2.12 (4.12 to 5.98)	<0.001
Age less than 5 years	3.23 (1.30 to 8.56)	<0.001
Sex	2.98 (2.30 to 4.56)	0.340
Birth weight	1.96 (2.30 to 10.56)	0.060
Birth order	1.34 (2.30 to 4.12)	0.340
Social class	3.98 (1.30 to 5.56)	0.450

Table (6) presents the multivariate logistic regression analysis of predictors of malnutrition in children with CHD. Malnutrition correlated significantly ( $p<0.001$ ) with low haemoglobin (anaemia), age under 5 years, heart failure, low arterial oxygen saturation, poor dietary fat intake and duration of symptoms of CHD, but not with social class, birth weight, sex or birth order.

### Discussion:-

In the present study the main study groups (cases and controls) were compared regarding height, weight, BMI, Gestational age, birth order, and birth weight. The study reported statistically significant difference ( $P$ -value  $<0.05$ ) with respect to weight, height and Body mass index between the two groups. However, the difference was not statistically significant between the two groups with respect to gestational age, birth order and birth weight. The prevalence of CHD-related malnutrition was 90% as compared to 21.2% in the control patients with 63.3% of cases having severe malnutrition. Among cases, the relative proportions of wasting, stunting and underweight were 40.8%, 28.8% and 20.8%, respectively. Wasting was proportionately higher (58.3%) in acyanotic CHD, while stunting was predominant (67.8%) in cyanotic CHD ( $p=0.0001$ ). The predictors of malnutrition in the present study included CHF, type of CHD, duration of symptoms, age under 5 years and poor dietary fat intake. Malnutrition correlated significantly ( $p<0.001$ ) with low haemoglobin (anaemia), age under 5 years, heart failure, low arterial oxygen saturation, poor dietary fat intake and duration of symptoms of CHD, but not with social class, birth weight, sex or birth order. These predictors are similar to those in previous reports and are generally modifiable by early corrective interventions, growth monitoring and nutrition supplementation. Previous studies that compared growth impairment in CHD before and after corrective interventions including surgery have demonstrated satisfactory recovery in somatic growth, although most such studies focused on infants.

The strengths of the present study include its case–control design that allows temporal relationships and associations between CHD and malnutrition to be examined. Also, anthropometric measurements and categorisation of malnutrition are based on standard reference growth standards and the study population is well characterised. Furthermore, the broad age range of these children with unoperated CHD allows long-term growth impairment (wasting and stunting) to be evaluated.

### References:-

1. Forchielli ML, McColl R, Walker WA, et al. Children with congenital heart disease: a nutrition challenge. *Nutr Rev* 1994 ; 52 : 348 – 53
2. Krovetz W. Weight gain in children with patent ductus arteriosus. *Dis Chest* 1963;44:274-83
3. De Staebel O. Malnutrition in Belgian children with congenital heart disease on admission to hospital. *J Clin Nurs*. 2000;9(5):784–91. doi: 10.1046/j.1365-2702.2000.00409.x.
4. Chen CW, Li CY, Wang JK. Growth and development of children with congenital heart disease. *J Adv Nurs*. 2004;47(3):260–9. doi: 10.1111/j.1365-2648.2004.03090.x. [PubMed: 15238120].
5. Gomez F. Mortality in second and third degree malnutrition. *J Trop Pediatr Afr Child Health* 1956 ; 2 : 77 .
6. Man WDC. Nutritional status of children admitted to hospital with different diseases and its relationship to outcome in the Gambia, West Africa. *Trop Med Int Health* 1998 ; 3 : 678 –86 .
7. Pelletier DL, Frongillo EA Jr, Habicht JP. Epidemiologic evidence for a potentiating effect of malnutrition on child mortality. *Am J Public Health* 1993 ; 83 : 1130 – 3

8. Campbell M, Reynolds G. The physical and mental development of children with congenital heart disease. Arch Dis Child 1949 ; 24 : 294 – 302 .
9. Menon G, Poskitt EME. Why does Congenital heart disease cause failure to thrive? Arch Dis Child 1985; 60: 11 34-9
10. Thommessen M, Heiberg A, Kase BF. Feeding problems in children with congenital heart disease: the impact on energy intake and growth outcome. Eur J Clin Nutr1992;46:457-64
11. Krauss AN, Auld P. Metabolic rate of neonates with congenital heart disease. Arch Dis Child 1975;50: 539-41
12. Less MH, Bristow JD. Relative hypermetabolism in infants with congenital heart disease and undernutrition. Pediatrics1965;36:183-91
13. KrovetzW. Weight gain in children with patent ductusarteriosus. Dis Chest 1963;44:274-83
14. Pittman JG, Cohen P. The pathogenesis of cardiac cachexia. N Engl J Med 1964;271:453
15. Butte NF. Energy requirements during infancy. In: Tsang and Nichols, eds. Nutrition in Infancy. Philadelphia, PA: CV Mosby, 1988:86-99
16. Stranway A, Fowler R, Cunningham K, Hamilton JR. Diet and growth in congenital heart disease. Pediatrics1976;57:75-86
17. Sondheimer JM, Hamilton JR. Intestinal function in infants with severe congenital heart disease. J Pediatr1987;92:572-8
18. Huse DM, Feldt RH, et al. Infants with congenital heart disease. Am J Dis Child 1975;129:65
19. Yahav J, Avigad S, Frand M. Assessment of intestinal and cardiorespiratory function in children with congenital heart disease on high-caloric formulas. JPediatr Gastroenterol Nutr1985;4:77&85
20. Jackson M, Poskitt EME. The effects of high-energy feeding on energy balance and growth in infants with congenital heart disease and failure to thrive. Br J Nutr1991;65:131-43
21. Mehzi A, Drash A. Growth disturbance in congenital heart disease. J Pediatr1962;61:418-29
22. Friedli B, Rouge JC, Faidutti B, Hahn C. Correction chirurgicale complete de cardiopathies congenitales chez le nourisson. HelvPaediatr Acta 1977;32:443-9
23. Okoromah CA, Ekure EN, Lesi FE, Okunowo WO, Tijani BO, Okeiyi JC. Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study. Arch Dis Child. 2011 Apr;96(4):354-60.