Iran J Pediatr. 2020 February; 30(1):e98542. doi: 10.5812/ijp.98542.

Published online 2020 February 10. **Research Article**

Nutritional Status in Non-Syndromic Cyanotic Congenital Heart Diseases Patients: A Single Tertiary Center Study in Iran

Mohammad Reza Mirzaaghayan [1, 2], Azin Ghamari [3], Amir Salimi [3] and Ehsan Aghaei Moghadam [2, 4, *]

[1] Department of Surgery, Tehran University of Medical Sciences, Tehran, Iran
[2] Childern's Medical Center, Pediatric Center of Excellence, Tehran, Iran
[3] Growth and Development Research Center, Tehran University of Medical Sciences, Tehran, Iran
[4] Department of Pediatrics, Tehran University of Medical Sciences, Tehran, Iran

[*] *Corresponding author*: Division of Pediatric Cardiology, Children's Medical Center, No. 62, Dr. Gharib St, Tehran, Iran. Email: ehsan_aghaei1358@yahoo.com

Received 2019 October 01; **Revised** 2019 December 24; **Accepted** 2020 January 15.

Abstract

Background: Patients with congenital heart disease (CHD) are susceptible to mild malnutrition up to failure to thrive. The prevalence of malnutrition among patients with CHD is higher in developing countries. Many factors are considered to affect the nutritional status such as cyanosis.
Objectives: This study aims to assess the prevalence and severity of malnutrition in patients with CHD prior to the surgical corrective or palliative repair.
Methods: This study was performed by reviewing inpatient medical records and cardiac surgery databases of children with cyanotic CHD who underwent palliative or corrective surgery between March 2011 and March 2017. Patients' age and height at the time of surgery, birth weight, duration of intensive care unit (ICU) stay and 30-day mortality were extracted and weight for age z score (WAZ), weight for length z score (WLZ) and length for age z score (LAZ) were calculated. Z scores above -1 were considered as normal, between -1 and -2 as mild, between -2 and -3 as moderate and below -3 as severe malnutrition.
Results: In a total number of 639, the average age, weight and height were 16.688 ± 24.859 months, 7.509 ± 5.629 kilograms and 73.759 ± 95.869 centimeters, respectively. The average length of ICU stay was 8.36 ± 6.254 days and the 30-day mortality was 36 (5.7%). The average WAZ, LAZ, WLZ and BMI Z was -1.5 ± 1.69, -0.9 ± 2.38, -1.2 ± 3.97 and -1.4 ± 2.11, respectively. Regarding WAZ, LAZ, WLZ and BMI Z, 62.2%, 46.1%, 72.3% and 70.9% of patients had mild to severe forms of malnutrition, since the rate of malnutrition based on WAZ ($P = 0.001$), LAZ ($P < 0.0001$), WLZ ($P < 0.0001$) and BMI Z ($P = 0.007$), was significantly higher than normal subjects.
Conclusions: The prevalence of mild to severe forms of malnutrition, based on different growth indices, for both cyanotics and acyanotics are higher than those reported in the other previous studies. These findings reflect the fact that cultural, genetic and racial differences of Iranians compared to others, cause malnutrition to be more frequent in Iranian CHD children. This calls for higher requirement of nutritional support for these patients.

Keywords: Congenital Heart Diseases, Malnutrition, Growth, Cyanosis, Non-Syndromic CHD

1. Background

Congenital heart diseases (CHD) include a variety of malformations defined by Mitchell et al. (1) as the structural abnormalities of the heart and great vessels which exist at birth and have functional response at some point of life. CHD is among congenital anomalies that impose remarkable burden on families and health care system. It includes about 28% of all congenital anomalies worldwide (2). The annual global prevalence of CHD is estimated to be 1.35 million in 150 million live births (3). CHD is more common in Asia, with prevalence of 9.3 per 1,000 live births, mostly due to high consanguinity, especially among Iranians (4).

Patients with congenital heart disease are susceptible to mild malnutrition up to failure to thrive (5). Malnutrition has its highest rate and severity in developing countries (6, 7); although it has a significant prevalence even in developed countries (8). Several factors have been identified to contribute to the occurrence of malnutrition, such as increased mean total daily energy expenditure, decreased energy intake, increased oxygen consumption, disturbed absorption due to bowel congestion, inability of the tissues to use the nutrients and disturbed cellular metabolism due to under oxygenation (9). Children with complex heart lesions need beyond 120% of the common energy intake to have acceptable growth pattern (10, 11).

This situation is more critical for children, because besides the increased energy demand caused by CHD, they require more nutrient and energy for growth and neurodevelopment. Children affected by malnutrition are at a higher risk of infection and mortality even after surgery (10). Nutritional challenges often appear at birth or soon after birth and growth impairment is more pronounced during neonatal period, especially for those with hemodynamic-significant defects. Malnutrition causes both short-term complications, including longer hospital and Intensive care unit (ICU) stay, postoperative infection, long-term complications such as impaired neurodevelopment and mortality (11-13). Nutritional assessment and management in these children could result in remarkable improvement, including better results and less complications of the surgery, better growth and neurodevelopment and less imposed burden on the affected families and health care system; however, the first step in this regard, is to identify and evaluate the prevalence and severity of malnutrition and growth impairment on these patients.

2. Objectives

This study aimed to assess the prevalence and severity of malnutrition among patients with CHD prior to the surgical corrective or palliative repair.

3. Methods

3.1. Study Design

This study was performed as a retrospective study, evaluating the medical records of children with cyanotic congenital heart diseases who had undergone palliative or corrective surgery between March 2011 and March 2017 at Children's Medical Center of Excellence, Tehran, Iran. All patients with cyanotic congenital heart disease, diagnosed by echocardiography had moderate/high risk cardiac defects (Box 1). Children with complex gastrointestinal anomalies affecting the nutritional state, syndromic patients and children with birth weight below 1300 grams were excluded from the study. This study is approved by our institutional board review.

Box 1. The Severity of Cardiac Defects Among the Participants

Cardiac Defects
1. low nutrition risk
Coarctation of aorta
Patent ductus arteriosus (if early surgery)
Atrial septal defect (minor lesion)
Cor triatriatum
Transposition of great arteries
Total anomalous pulmonary venous drainage
VSD (small lesion)
Pulmonary stenosis
2. Medium nutrition risk
Pulmonary atresia
Tetralogy of fallot
Atrial septal defect (severe lesion)
3. High nutrition risk
Ventricular septal defect (moderate to large)
Arterioventricular septal defect
Hypoplasiic left heart syndrome
Truncus arteriosus
Aortopulmonary window
Patent ductus arteriosus (if large or delayed surgery)
Tricuspid atresia
Ebstein anomaly
Partial anomalous pulmonary venous drainage

3.2. Data Collection and Statistical Analysis

All inpatient medical records and cardiac surgery databases were reviewed. patients' and height at the time of surgery, birth weight, duration of intensive care unit (ICU) stay and 30-day mortality were extracted and weight for age z score (WAZ), weight for length z score (WLZ) and length for age z score (LAZ) were calculated using World Health Organization (WHO) Anthro (version 3.2.2 January 2011) software. Z scores above -1 were considered as normal, between -1 and -2 as mild, between -2 and -3 as moderate and below -3 as severe malnutrition (14). WAZ represents immediate, WLZ acute and LAZ chronic malnutrition. BMI z scores were also categorized in the same way as severely thin, moderately thin, mildly thin and normal (15). The amount and severity (normal, mild, moderate and severe) of malnutrition were compared between males and females. All data were analyzed by SPSS version 22. Qualitative variables are expressed by number (percent) and qualitative variables by mean $\pm$ standard deviation. P value less than 0.05 was considered significant.

4. Results

4.1. Study Population

Totally 639 patients were recruited. 359 (56.2%) were male and 280 (43.8%) female without any significant difference (P value = 0.415). The average age, weight and height were 16.688 $\pm$ 24.859 months, 7.509 $\pm$ 5.629 kilograms and 73.759 ± 95.869 centimeters, respectively. The average length of ICU stay was 8.36 ± 6.254 days. The 30-day mortality was 5.7% (36 cases). The mean birth weight of the subjects was 2986.462 + 443.57 grams.

4.2. Malnutrition Status

The average WAZ, LAZ, WLZ, and BMI Z was -1.5 ± 1.69, -0.9 ± 2.38, -1.2 ± 3.97, and -1.4 ± 2.11, respectively. Regarding WAZ, LAZ, WLZ and BMI Z, 62.2%, 46.1%, 72.3%, and 70.9% of patients had mild to severe forms of malnutrition, as the rate of malnutrition based on WAZ ($P = 0.001$), LAZ ($P < 0.0001$), WLZ ($P < 0.0001$) and BMI Z ($P = 0.007$), was significantly higher than normal subjects. (Table 1 and Figures 1 and 2). The severity of malnutrition did not differ between males and females, based on WAZ ($P = 0.130$), LAZ ($P = 0.661$), and WLZ ($P = 0.125$). The mortality rate was not associated with lower WAZ ($P = 0.866$), LAZ ($P = 0.907$), and BMI Z ($P = 0.357$); but was associated with lower WLZ ($P = 0.023$), birth weight ($P = 0.014$), weight ($P = 0.038$), and height ($P = 0.026$). There was no significant association between length of the ICU stay and severity of malnutrition, based on WAZ ($P = 0.203$), LAZ ($P = 0.295$), WLZ ($P = 0.734$), and BMI Z ($P = 0.777$). Lower birth weight was associated with more severe malnutrition based on all growth indices (P value < 0.0001).

5. Discussion

Malnutrition is a common problem for CHD patients, regardless of the presence of cyanosis and type of cardiac anomaly. In USA, 33% of children with CHD have acute and 64% have chronic malnutrition (11). Determination of the growth disturbance for cyanotic and acyanotic defects has been changed considerably over the past 25 years due to surgical interventions in early childhood, especially for patients with cyanotic lesions. Several factors are associated with the type and severity of the malnutrition, including presence of cyanosis, congestive heart failure, and pulmonary hypertension. Villasis-Keever et al. (16) found that presence of cyanosis, lack of nutritional supplementation and great number of family numbers are among the risk factors of malnutrition. Delayed surgical repair put patients with CHD in greater risk for developing malnutrition (17). In developing countries, the surgical corrections are often delayed, due to high demands for the surgery in a tertiary center, on the other hand, some procedures are preferred to be performed later until adequate weight gain; although this point of view is reconsidered recently (18). Okoromah et al. (9) indicated that the predictors of malnutrition among patients with CHD includes type of CHD, presence of CHF, duration of the symptoms, less than 5 years of age, anemia, lower oxygen saturation and poor dietary condition. All patients in the current study had cyanotic CHD, which causes low oxygen saturation and had a mean age of 16.88 months. As most of cyanotic CHD lesions need to be operated as soon as possible due to becoming symptomatic, 16.8-month duration of having symptoms could cause considerable impact on growth pattern. Cyanotic defects lead to disturbed height and weight growth patterns (19). Linde et al. (20) have indicated that acute and chronic malnutrition is more common among cyanotic patients than acyanotic ones. In the current study, 72.3% and 46.1% of cyanotic patients had acute and chronic malnutrition.

Surgical stress and activation of the inflammatory cascade postoperatively, lead to postoperative endocrine dysfunction, and increased energy requirement which bring about further malnutrition exacerbation (21). Infants and newborns are susceptible to hyper catabolic state postoperatively, due to lower protein and energy reserves, although postoperative hyper catabolism is not a common phenomenon in all patients with CHD (22, 23). Previous studies suggested association between poor wound healing, myocardial dysfunction, and vascular endothelial dysfunction and malnutrition prior to the surgery (24). These findings highlighten the importance of the nutritional assessment and support for these patients.

Lower WLZ, called "wasting", represents acute malnutrition and lower LAZ, called "stunting", represents chronic malnutrition (25), lower WAZ also indicates immediate malnutrition. The prevalence of underweight, stunting and wasting in the study of Hassan et al. (26) in 2015 in Egypt reported to be 14.3%, 61.9% and 23.8%; but the prevalence of the aforementioned variables in the study of Okoromah et al. (9) in 2011 in Nigeria were 20.5%, 28.8% and 41.1%, and the relative proportions of the underweight, stunting and wasting in the study of the Ratanachu-Ek (27) in 2011 in Thailand were 28%, 16% and 22%, respectively. Arodiwe et al. (28) found that 66% of the patients had severe malnutrition, and they also found that stunting was more common than wasting. The prevalence of underweight, stunting and wasting in the current study is 70.9%, 46.1%, and 72.3%, respectively, which is remarkably higher than those of other countries. The prevalence of the immediate malnutrition, wasting and stunting in the study of Aghaei-Moghadam et al. (29) in 2019 in Iran among acyanotic patients were 68.7%, 66.4%, and 48.6%, respectively. The prevalence of mild to severe forms of malnutrition, based on different growth indices, for both cyanotics and acyanotics are higher than those reported in the other previous studies. These findings reflect the fact that cultural, genetic and racial differences of Iranians compared to those of others, cause malnutrition to be more frequent in Ira-

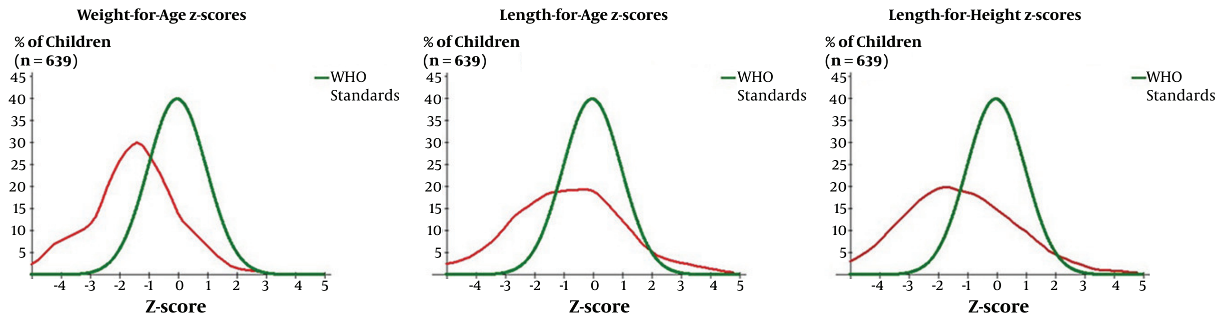

Figure 1. The Gaussian distribution of malnutrition in the present study, compared to WHO standards

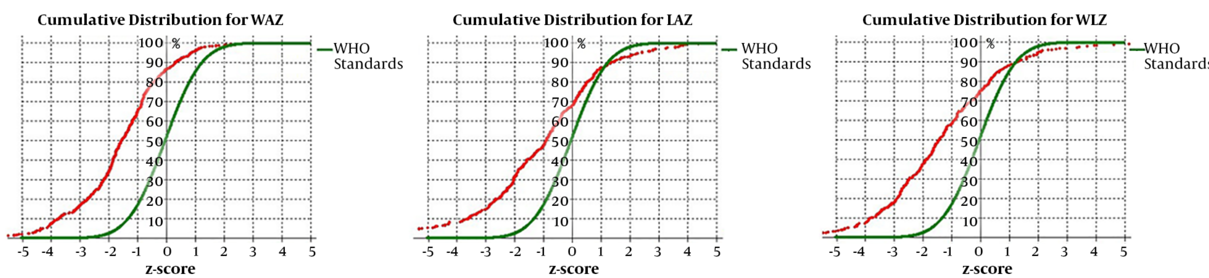

Figure 2. The cumulative curves of malnutrition in the present study, compared to the WHO standards

Table 1. The Prevalence of Different Stages of the Malnutrition Among Our Patients[a]

	Normal (> -1)	Mild (-2 to > -1)	Moderate (-3 to > -2)	Severe (< -3)
WAZ	238 (37.8)	188 (29.9)	105 (16.7)	98 (15.6)
LAZ	241 (53.9)	80 (17.9)	64 (14.3)	62 (13.9)
WLZ	174 (27.7)	303 (48.2)	78 (12.4)	74 (11.8)
BMI-Z	183 (29.1)	279 (44.4)	83 (13.2)	84 (13.4)

[a]Values are expressed as No. (%).

nian children diagnosed with CHD. This calls for higher requirement of nutritional support for these patients.

Previous studies have shown that malnutrition is associated with increased mortality and morbidity; it causes frequent hospitalization, poor surgical results and persistent impaired somatic growth (12). Furthermore, protracted and recurrent hospitalization worsen the nutritional status (30). Growth failure in early childhood is associated with delayed mental development, poor school performance and reduced intellectual abilities (13). This study investigated the 30-day mortality rate and length of ICU stay as the short-term postoperative complications. The 30-day mortality rate was 5.7%, which was related to the lower birth weight, weight, height and WLZ. Several studies have demonstrated that malnourished CHD patients have a longer hospital stay length (25, 31). Ross et al. (11) in 2017 found that lower WAZ and LAZ have a significant association with postoperative mortality, cardiac arrest, infection, longer mechanical ventilation and ICU stay. They also demonstrated that the nutritional support for those with growth indices below -2 results in lower mortality rate (11). In line with the previous study, Mitting et al. (25) in 2015 and Eskedal et al. (32) in 2008 found that lower WAZ is associated with higher mortality and increased duration of mechanical ventilation. The mortality rate was associated with WLZ (acute malnutrition), but it was not associated with other growth indices. The length of ICU stay in this study was 8.36 ± 6.254 days, with no association with WAZ, LAZ or WLZ.

Infants with single cardiac defect usually are born with normal weight, but the growth impairment appears thereafter (33), but the scenario is different for patients with more complex heart lesions. In a multi centric study investigating 1245 infants, known cases of univentricular CHD, in North America these patients had a lower birth weight and small for gestational age cases were more among them, compared to the normal subjects. This study concluded that the growth failure process begins during the fetal period which continues after the birth (34). The findings of the present study are in line with the previous study. Lower birth weight was associated with lower growth indices, which represents the impaired weight gain has initiated from fetal period and also that the growth and weight gain of the infants with lower birth weight need to be evaluated by a pediatric endocrinologist or dietician.

Footnotes

Authors' Contribution: Mohammad Reza Mirzaaghayan and Ehsan Aghaei Moghadam designed the project and wrote the final report. Amir Salimi and Azin Ghamari collected the data and analyzed it.

Conflict of Interests: The authors declare no conflict of interests.

Ethical Approval: This study is approved by our Institutional Board Review.

Funding/Support: None.

References

1. Mitchell SC, Korones SB, Berendes HW. Congenital heart disease in 56,109 births. Incidence and natural history. *Circulation*. 1971;**43**(3):323–32. doi: 10.1161/01.cir.43.3.323. [PubMed: 5102136].
2. Dolk H, Loane M, Garne E; European Surveillance of Congenital Anomalies Working Group. Congenital heart defects in Europe: Prevalence and perinatal mortality, 2000 to 2005. *Circulation*. 2011;**123**(8):841–9. doi: 10.1161/CIRCULATIONAHA.110.958405. [PubMed: 21321151].
3. Hoffman JI. Incidence of congenital heart disease: I. Postnatal incidence. *Pediatr Cardiol*. 1995;**16**(3):103–13. doi: 10.1007/BF00801907. [PubMed: 7617503].
4. Naderi S. Congenital abnormalities in newborns of consanguineous and nonconsanguineous parents. *Obstet Gynecol*. 1979;**53**(2):195–9. [PubMed: 570260].
5. Day M. Growth and nutritional intake of infants with congenital heart disease. *Pediatr Ann*. 1989;**7**:35–57.
6. Thompson Chagoyan OC, Reyes Tsubaki N, Rabiela Barrios OL, Buendia Hernandez A, Miranda Chavez I, Carrasco Quintero R. [The nutritional status of the child with congenital cardiopathy]. *Arch Inst Cardiol Mex*. 1998;**68**(2):119–23. Spanish. [PubMed: 9810354].
7. Varan B, Tokel K, Yilmaz G. Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension. *Arch Dis Child*. 1999;**81**(1):49–52. doi: 10.1136/adc.81.1.49. [PubMed: 10373135]. [PubMed Central: PMC1717989].
8. Cameron JW, Rosenthal A, Olson AD. Malnutrition in hospitalized children with congenital heart disease. *Arch Pediatr Adolesc Med*. 1995;**149**(10):1098–102. doi: 10.1001/archpedi.1995.02170230052007. [PubMed: 7550812].
9. 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;**96**(4):354–60. doi: 10.1136/adc.2009.176644. [PubMed: 21266339]. [PubMed Central: PMC3056291].
10. Medoff-Cooper B, Ravishankar C. Nutrition and growth in congenital heart disease: A challenge in children. *Curr Opin Cardiol*. 2013;**28**(2):122–9. doi: 10.1097/HCO.0b013e32835dd005. [PubMed: 23370229].
11. Ross F, Latham G, Joffe D, Richards M, Geiduschek J, Eisses M, et al. Preoperative malnutrition is associated with increased mortality and adverse outcomes after paediatric cardiac surgery. *Cardiol Young*. 2017;**27**(9):1716–25. doi: 10.1017/S1047951117001068. [PubMed: 28625194]. [PubMed Central: PMC5908464].
12. Forchielli ML, McColl R, Walker WA, Lo C. Children with congenital heart disease: A nutrition challenge. *Nutr Rev*. 1994;**52**(10):348–53. doi: 10.1111/j.1753-4887.1994.tb01359.x. [PubMed: 7816352].
13. Pelletier DL, Frongillo EA, Jr, Habicht JP. Epidemiologic evidence for a potentiating effect of malnutrition on child mortality. *Am J Public Health*. 1993;**83**(8):1130–3. doi: 10.2105/ajph.83.8.1130. [PubMed: 8342721]. [PubMed Central: PMC1695164].
14. Webb P, Bhatia RA. Manual: Measuring and interpreting malnutrition and mortality. *Nutr Serv WFP Rome*. 2005;**17**.
15. Carter RG, Feigelman S. The second year. In: Kliegman R, Stanton B, Geme J, editors. *Nelson textbook of pediatrics*. Elsevier Health Sciences: New York; 2020. p. 137–43.
16. Villasis-Keever MA, Aquiles Pineda-Cruz R, Halley-Castillo E, Alva-Espinosa C. [Frequency and risk factors associated with malnutrition in children with congenital cardiopathy]. *Salud Publica Mex*. 2001;**43**(4):313–23. Spanish. [PubMed: 11547592].
17. Roman B. Nourishing little hearts: Nutritional implications for congenital heart defects. *Pract Gastroenterol*. 2011;**8**:11–32.
18. Mirzaaghayan MR, Moghadam EA, Dehestani A, Ghamari A. Early surgical closure of the ventricular septal defects; to be done or not to be done? A question to be answered. *Iran J Pediatr*. 2018;**28**(6).
19. Mehrizi A, Drash A. 50 years ago in the journal of pediatrics: Growth disturbance in congenital heart disease. *J Pediatr*. 1962;**61**:418–29. doi: 10.1016/j.jpeds.2012.03.017. [PubMed: 14472142].
20. Linde LM, Dunn OJ, Schireson R, Rasof B. Growth in children with congenital heart disease. *J Pediatr*. 1967;**70**(3):413–9. doi: 10.1016/s0022-3476(67)80139-2.
21. Milne EMG, Elliott MJ, Pearson DT, Holden MP, Ørskov H, Alberti KGMM. The effect on intermediary metabolism of open-heart surgery with deep hypothermia and circulatory arrest in infants of less than 10 kilograms body weight. A preliminary study. *Perfusion*. 2016;**1**(1):29–40. doi: 10.1177/026765918600100104.
22. Finnerty CC, Mabvuure NT, Ali A, Kozar RA, Herndon DN. The surgically induced stress response. *JPEN J Parenter Enteral Nutr*. 2013;**37**(5 Suppl):21S–9S. doi: 10.1177/0148607113496117. [PubMed: 24009246]. [PubMed Central: PMC3920901].
23. Mehta NM, Costello JM, Bechard LJ, Johnson VM, Zurakowski D, McGowan FX, et al. Resting energy expenditure after Fontan surgery in children with single-ventricle heart defects. *JPEN J Parenter Enteral Nutr*. 2012;**36**(6):685–92. doi: 10.1177/0148607112445581. [PubMed: 22539159]. [PubMed Central: PMC3713488].
24. Radman M, Mack R, Barnoya J, Castaneda A, Rosales M, Azakie A, et al. The effect of preoperative nutritional status on postoperative outcomes in children undergoing surgery for congenital heart defects in San Francisco (UCSF) and Guatemala City (UNICAR). *J Thorac Cardiovasc Surg*. 2014;**147**(1):442–50. doi: 10.1016/j.jtcvs.2013.03.023. [PubMed: 23583172]. [PubMed Central: PMC3787941].
25. Mitting R, Marino L, Macrae D, Shastri N, Meyer R, Pathan N. Nutritional status and clinical outcome in postterm neonates undergoing surgery for congenital heart disease. *Pediatr Crit Care Med*. 2015;**16**(5):448–52. doi: 10.1097/PCC.0000000000000402. [PubMed: 25828781].

26. 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. *Front Pediatr*. 2015;**3**:53. doi: 10.3389/fped.2015.00053. [PubMed: 26125014]. [PubMed Central: PMC4467172].
27. Ratanachu-Ek S, Pongdara A. Nutritional status of pediatric patients with congenital heart disease: pre- and post cardiac surgery. *J Med Assoc Thai*. 2011;**94 Suppl 3**:S133–7. [PubMed: 22043766].
28. Arodiwe I, Chinawa J, Ujunwa F, Adiele D, Ukoha M, Obidike E. Nutritional status of congenital heart disease (CHD) patients: Burden and determinant of malnutrition at university of Nigeria teaching hospital Ituku - Ozalla, Enugu. *Pak J Med Sci*. 2015;**31**(5):1140–5. doi: 10.12669/pjms.315.6837. [PubMed: 26649002]. [PubMed Central: PMC4641271].
29. Aghaei-Moghadam E, Mirzaaghayan MR, Zeinaloo AA, Malakan Rad E, Sayadpour K, Alavi AH, et al. Nutritional status in Iranian nonsyndromic patients with acyanotic congenital heart disease undergoing surgical repair. *J Compr Pediatr*. 2019;**10**(2). e86639. doi: 10.5812/compreped.86639.
30. Ross RD, Bollinger RO, Pinsky WW. Grading the severity of congestive heart failure in infants. *Pediatr Cardiol*. 1992;**13**(2):72–5. doi: 10.1007/BF00798207. [PubMed: 1614922].
31. Grippa RB, Silva PS, Barbosa E, Bresolin NL, Mehta NM, Moreno YM. Nutritional status as a predictor of duration of mechanical ventilation in critically ill children. *Nutrition*. 2017;**33**:91–5. doi: 10.1016/j.nut.2016.05.002. [PubMed: 27364223].
32. Eskedal LT, Hagemo PS, Seem E, Eskild A, Cvancarova M, Seiler S, et al. Impaired weight gain predicts risk of late death after surgery for congenital heart defects. *Arch Dis Child*. 2008;**93**(6):495–501. doi: 10.1136/adc.2007.126219. [PubMed: 18230653].
33. Naeye RL. Anatomic features of growth failure in congenital heart disease. *Pediatrics*. 1967;**39**(3):433–40. [PubMed: 6018974].
34. Williams RV, Ravishankar C, Zak V, Evans F, Atz AM, Border WL, et al. Birth weight and prematurity in infants with single ventricle physiology: Pediatric heart network infant single ventricle trial screened population. *Congenit Heart Dis*. 2010;**5**(2):96–103. doi: 10.1111/j.1747-0803.2009.00369.x. [PubMed: 20412481]. [PubMed Central: PMC2895559].