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ORIGINAL ARTICLE

Nutritional Management of Children with Congenital Heart Diseases in Zagazig University Children Hospital

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ABSTRACT

Background: Malnutrition and growth failure is highly prevalent among infants with congenital heart disease. The aim was the assessment of the nutritional status of children with congenital heart diseases (CHD) and evaluate the impact of nutritional counseling and support for those children.

Methods: This study included 42 children of CHD with under nutrition, where 19 had cyanotic CHD and 23 had acynotic CHD (of the total 13 had heart failure and 16 had pulmonary hypertension). They were subjected to medical history, clinical examination, cardiological assessment including cardiac symptoms, echocardiographic assessment, nutritional assessment through (nutritional history, calculation of subjective global assay, full anthropometric measures including weight, height, mid-arm circumference, triceps skinfold measurement, head circumference) and nutritional plan was applied to those patients. Follow-up visits were done every 2 weeks and patients were reassessed after 2 months.

Results: (54.8%) of our cases had a moderate degree of malnutrition while (45.2%) of cases had severe degree. There was a significant improvement in all anthropometric measurements post to nutritional support and there was a significant increase in weight all over the study follow-up intervals. There was no improvement in length.

Conclusions: Malnutrition is common in pediatric patients with CHD. It leads to serious complications. Proper nutritional assessment and intervention help improve the general conditions.

Keywords: *Nutrition; Children; Congenital Heart Disease.*

INTRODUCTION

The incidence of congenital heart disease is like the population of the world. It is about 8 per 1,000 live births [1]. The coincidence of CHD in Egypt is 1-2% [2,3]. Malnutrition complicated by failure to thrive is common among children with CHD irrespective of the type of cardiac defect and presence or absence of cyanosis [4]. The causes of malnutrition in cardiac patients are decreasing energy intake, increased catabolism due to infection and tachycardia and increase energy requirements. The degree of malnutrition may range from mild to severe growth failure [5]. About 64% of patients have failed to thrive in developed countries [6]. In developing regions, it may reach around 90% [5,7,8]. The presence of congestive heart failure (CHF), cyanosis or pulmonary hypertension may affect the type and degree of malnutrition [9,10]. Proper nutritional intervention is mandatory for cardiac patients due to frequent hospitalization which indicates increased morbidity and mortality. Proper nutritional care of patients improves surgical outcomes, decreases admission in hospital, improves somatic growth and decreases death, especially if begins early [11,12]. In most cases, the improvement of nutritional status of cases may be a challenge due to the complex interplay between medical, surgical, and social factors [13].

METHODS

This is an interventional study that was conducted at Outpatient Nutrition and Inpatient Cardiology Units of the Pediatric Department at Zagazig University. The duration of the study was 11 months. It began in March 2019 and ended in February 2020.

Before the start of the study, permission was obtained from the Institutional Review Board “IRB”

and Ethical Committee in the Faculty of Medicine, Zagazig University. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans, also informed consent from the patient's parents included in the study was obtained.
The study included 42 Cardiac patients with undernutrition. Among them 19 had cyanotic CHD and 23 had acyanotic congenital heart disease, although of the total 13 had heart failure and 16 had pulmonary hypertension.
They were selected according to the following criteria: children with malnutrition-associated congenital heart diseases including both cyanotic and non-cyanotic, children > 4 months and both male and female were included. While children with associated comorbidities such as chromosomal anomalies and chronic diseases other than CHD, children who underwent surgical intervention, children < 4 months, and children with other heart diseases like cardiomyopathy were excluded. All selected patients were subjected to the followings:
Clinical evaluation: Full detailed medical history was taken, a full general examination was done to all patients including the general appearance, activity, presence of edema, ascites, cachexia, skin changes, dry mucous membranes, cardiological assessment: including cardiac symptoms e.g., tachypnea, retractions, grunting and Cardiac examination by careful palpation of pulses and measurement of blood pressure, evaluation of heart rate, sounds and murmurs and echocardiographic assessment).
Nutritional assessment: (Full nutritional history regarding weight change, appetite, taste change, nausea, vomiting, bowel habits, chewing, swallowing habits, and shortness of breathing on feeding).
Full Anthropometric Measures: (Weight, length, mid-arm circumference, triceps skinfold, head circumference).
Nutritional plan: The Forty-two Patients were subjected to the following:
(Counseling was done about type, amount of feeding and the required energy expenditure was estimated according to the age plus extra calories for the cardiac condition which is about 30% of total caloric requirements). Protein was calculated according to age requirements. Smaller volumes and more frequent feeds orally were offered. Unfinished feeds were given via nasogastric tube. Change formula from ordinary one to high caloric formula for giving the required calories in a small volume. We used a high-energy formula that gives 1 kcal /ml to children whose age is below 1 year. While formula of 1.5 kcal/ml was given to children above 1 year. If they did not afford the high caloric formula we used instead of it nutrient-dense e.g., adding cereals to milk (Cerelac rice or wheat, each teaspoon gives 20 kcal). It is shown in table (1).
Follow up: Regular follow up visits every 2 weeks, with each visit the following were done: weight measurement, counseling, and reassessment.
Finally, patients were reassessed after 2 months with full anthropometric measures and all laboratory investigations were repeated.
Statistical analysis" We entered data on computer and used "Microsoft Office Excel Software" program (2010). We transferred data to a software program of the statistical package of social science. Its version is 21 (SPSS) for analyzing data by statistics. Summarizing data was done. For quantitative data we estimated mean, median, and calculate standard deviation and percentiles. While qualitative data we detect frequency and percentage. We used Kruskal-Walli's test then Mann-Whitney test (pairwise comparisons) for comparison between groups of quantitative variables while using Chi-square or Fisher's exact test for comparison qualitative data. If values of p were less than 0.05, we considered the comparison between groups were statistically significant.

RESULTS

The present study included 42 children with acyanotic and cyanotic CHD. Table (2) shows Age ranged from 4-18 months (8.58±2.58) and sex was distributed evenly 50% 50%, weight was 2.86±0.25 Kg, the majority were breastfeeder 47.6%, combined 38.1%, artificial 14.3% and developmental retardation regard millstone in 28.6%.
Table (3) shows the distribution of CHD among the studied group. In the acyanotic group, the most common congenital anomaly was ASD (33.3%) followed by VSD (21.5%). While in the cyanotic group the most common anomaly presented in cases was fallot tetralogy (28.7%) followed by dextrorotation (16.8%).
Change of body measurements (weight, length, MAC, triceps skinfold and head circumferences) among the studied children after nutritional intervention were significantly increased except length as shown in table (4).
Table (5) shows the change in mineral and CBC levels among studied children after 2 months of nutritional intervention, where there was a

significant increase of Ca, Mg, Ph, HCT and HB and a significant decrease in platelet count but no significant change regarding Na, K or WBCs.

Figure (1) shows the change in Mid-arm circumference pre and post nutritional support intervention.

Table (1): Selected Dietary Reference Values (DRV's) for infants and children requiring oral/enteral nutrition was used according to cape town metropole pediatric working group: clinical guidelines CHD, (2009b) as the following:

Mean± SD **Median (range)**		8.58±2.58 6.0 (4-18)	
Weight at birth/ Kg **Mean± SD** **Median (range)**		2.86±0.25 2.95 (2.4-3.3)	
		N	**%**
Sex	**Male** **Female**	21 21	50.0 50.0
Feeding	**Artificial** **Breast** **Combined**	6 20 16	14.3 47.6 38.1
Development problems	**No** **Yes**	30 12	71.4 28.6
Duration of developmental problems / months	**Mean± SD** **Median (range)**	4.83±3.89 2.0 (1-12)	

Table (2): Basic data of studied children

Age	**Weight (Kg)**	**KJ/Kg/day**	**Kcal/kg/day**	**Protein g/kg/day**
Male **0-3 months**	5.1	420-480	100-115	2.1
4-6	7.2	400	95	1.6
7-9	8.9	400	95	1.5
10-12	9.6	400	95	1.5
1-3 years	12.9	400	95	1.1
4-6	19.0	3800	90	1.1
7-10		8240/day	1970/day	28.3g/day
11-14		9270/day	2220/day	42.1g/day
15-18		11510/day	2755/day	55.2g/day
Females				
0-3months	4.8	420-480	100-115	2.1
4-6	6.8	400	95	1.6
7-9	8.1	400	95	1.5
10-12	9.1	400	95	1.5
1-3years	12.3	400	95	1,1
4-6	17.2	380	90	1.1
7-10		7280/day	1740/day	28.3g/day
11-14		7920/day	1845/day	42.1g/day
15-8		8830/day	2110/day	45.4g/day

Table (3): Distribution of CHD among the studied groups

Type of CHD	N	%
Acyanotic	**23**	**54.8**
ASD	-14-	33.3
• ASD	8	19.0
• ASD + VSD	5	11.9
• ASD + PS	1	2.4
VSD	-9-	21.5
• VSD	5	11.9
• VSD + PFO	2	4.8
• VSD + PS	2	4.8
Cyanotic	**19**	**45.2**
Fallot	-12-	28.7
• Fallot	4	9.6
• Fallot + ASD	6	14.3
• Fallot + PFO	1	2.4
• Fallot + Dextro rotation	1	2.4
Dextro-rotation	-7-	16.8
• Dextro rotation	2	4.8
• Dextro rotation + ASD	1	2.4
Type of CHD	**N**	**%**
Cyanotic	**19**	**45.2**
• Dextro rotation + ASD + VSD	2	4.8
• Dextro rotation + PFO	2	4.8
Total	**42**	**100.0**

Table (4): Change of body measurements among the studied children after nutritional intervention

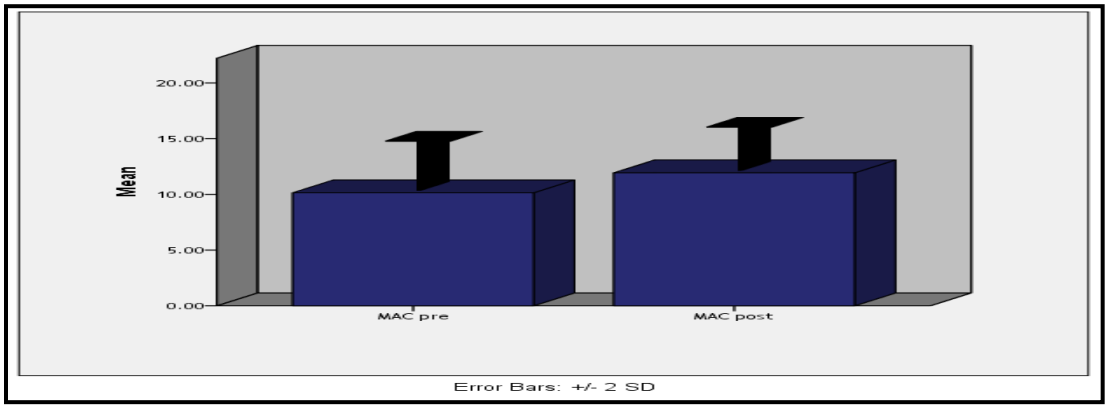
Variable	Pre (No=42)	Post (No=42)	Paired t	P
Length / cm	63.85±9.12	64.19±11.19	-0.187	0.852
Weight /Kg	5.87 ± 1.58	7.42 ± 1.85	-10.784	0.00**
MAC / cm	10.14±2.24	11.93±1.98	-10.093	0.00**
TSF / cm	0.56±0.25	0.82±0.27	-15.445	0.00**
HC / cm	40.54±3.35	42.07±2.92	-11.743	0.00**

HC: Head circumference; MAC: Mid-arm circumference; TSF: Triceps skin fold.

Table (5): Change in mineral and CBC levels among studied children after 2 months nutritional intervention

	Pre (42)	Post (42)	Paired t	P
Sodium "Na" (mEq/L)	137.64±2.21	137.08±2.89	0.414	0.621
Potassium "K" (mmol/L)	3.76±0.58	3.95±0.85	-1.505	0.140
Calcium "Ca" (mg/dl)	8.81±0.57	9.65±0.61	-9.126	0.00**
Phosphorus "Ph" (mg/dl)	3.61±1.06	3.82±1.01	-2.693	0.010*
Magnesium "Mg" (mg/dl)	2.17±0.34	2.31±0.22	-2.705	0.010*
White blood cells "WBCs" ($\times 10^9$/L)	11.62±2.62	11.92±2.47	-0.807	0.424
Hematocrit "HCT" (%)	28.06±4.97	31.51±3.55	-4.551	0.00**
Hemoglobin "HB" (g/dl)	9.41±1.07	10.18±1.14	-6.920	0.00**
Platelets "PLT" ($\times 10^9$/L)	474.97±109.98	421.45±104.66	2.529	0.015*

Figure (1): Change in Mid-arm circumference pre and post nutritional support intervention



DISCUSSION

Most of the infants with the cardiac defect have normal birth weight [14], but malnutrition and increased energy requirements expose them to an increased prevalence of growth failure [15]. Malnutrition leads to a decrease in weight for age z score – 0.8 and height for age z score – 0.5 [16].

Regarding basic demographic data, our results showed that this study was conducted on 21 (50%) female infants and 21 (50%) males, their ages were ranging between 4 – 18 months with mean age 8.58 ± 2.58 months. Their birth weights ranged between 2.4 – 3.3 kg with a mean weight of 2.86±0.25 kg. Similarly, El-Koofy N et al. [17] did a study on 50 patients having acyanotic cardiac defect for assessing nutritional status and for detection of the role of nutritional intervention. They reported that the mean age of their infants was (8.6±4.8 months) with a male: female ratio of 1:1.

In a study done by Tandberg BS et al. [18] on 60,600 mothers, they reported that between child aged 2–6 months, mothers of infants with CHD had a hazard ratio (HR) of 1.69 of weaning their child compared with mothers of controls. Mothers of infants with CHD with comorbidity weaned at an even faster rate (HR 3.54). At age 6 months, 9.9% of infants with CHD were fed with breast milk predominately, 64.1% continued to receive breast milk, and only 26% were fed no breast milk.

Regarding breastfeeding, our results showed that most of our malnourished infants (47.6%) were breastfed while only 14.3% of them were artificial feeders, 38.1% of them were combined feeders. As the artificial milk formula provides high calories and the breast milk may be scanty as the CHD child cannot make a good effort for suckling well so, breastfeeding is mainly dependent on the demand of the child.

Wong JJ et al. [19] stated that, although breast milk is the best nutrition increasing immunity due to a high level of immunoglobulin and having high nutritional value, formula feeding introduces excess calories, so it may be prescribed. Moreover, Labib NA et al. [20] reported that breastfed children on demand documented a higher prevalence of malnutrition compared to those receiving breastfeeding at regular intervals and the difference was statistically significant. However, El-Koofy N et al. [17] revealed that most of cases having FTT were formula feeding. This difference may be due to all cases in their study were acyanotic CHD.

Our results showed that 54.8% of cases had acyanotic congenital heart anomalies while 45.2% had cyanotic heart anomalies. The most common congenital anomaly in the acyanotic group was atrial septal defect “ASD” (33.3%) followed by ventricular septal defect “VSD” (21.5%) while in the cyanotic group, the most common anomaly was Tetralogy of Fallot “TOF” (28.7%) followed by dextrorotation (16.8%).

Saxena A et al. [21] agreed with our finding regarding the higher incidence of acyanotic CHD than the cyanotic CHD as they reported that 131 newborns had an acyanotic CHD (79.9%) and 33 a cyanotic CHD (20.1%). The incidence of different anomalies as they reported, VSD was the most common acyanotic CHD, present in 116 newborns, giving a prevalence of 5.7/1000 live births (0.57%). Among the cyanotic CHD, transposition of great arteries was most common with a prevalence 0.34/1000 live births (0.034%).

Kondapalli CS et al. [22] found that VSD is the most observed lesion (33.8%) followed by PDA (15%), ASD (10%), TOF (10%) and CoA (3.3%). Ravilala VK et al. [23] reported that in their study among the cyanotic CHDs, TGA was the commonest, followed by TOF. In the acyanotic group, VSD was the most common, followed by ASD. Miyague NI et al. [24] reported that the most frequent acyanotic congenital heart defects were as follows: ventricular septal defect (30.5%), atrial septal defect (19.1%) and persistent ductus

arteriosus (17%). Their most frequent cyanotic congenital heart defects were as follows: tetralogy of fallot (6.9%), transposition of the great vessels (4.1%) and tricuspid atresia (2.3%). These differences may be explained as our cases included associated lesions. The most common association was TOF + ASD as it was recorded in 6 (14.3%) of infants followed by ASD + VSD as it was recorded in 5 (11.9%) of infants.

Regarding the change in anthropometric measurements post to nutritional support intervention, our results showed that there was a significant improvement of all anthropometric measurements including body weight, mid-arm circumference "MAC", triceps skinfold "TSF" and head circumference "HC". There was a significant increase in weight all over the study follow-up intervals in comparison between weight measured at the start of the study and other weight measurements. There was no improvement in length. The unimproved length can be attributed to the short study interval (2 months interval in our study). Similarly, El-Koofy N et al. [17] found that there was a significant improvement in weight, MAC and TSF while the length showed no significant change.

Supplementation of infants by energy-dense formula early can prevent failure to thrive, this was documented by the study done by Marino LV et al. [25] on infants having CHD where it decreases fall of Z score of weight-for-age from birth also improve linear growth at 12 months of age.

Although Gongwer RC et al. [26] implanted a structured approach for nutritional intervention to infants with CHD and a significant increase in WAZ from baseline to 6 months the median WAZ was -3.5 and indicated overall the cohort was significantly malnourished.

In malnutrition various abnormalities occur in body electrolytes. The most common electrolyte abnormalities are those that involve sodium, potassium, bicarbonate, and water. In malnutrition with the edematous state, body water content is increased accompanied by sodium retention that is primarily extracellular, but serum sodium level is reduced in most children with malnutrition masking the sodium overload. Total body potassium is decreased in all malnourished by as much as 25% in overt malnutrition due to decreased intake and poor muscle mass. Both malnutrition and electrolyte disturbances are risk factors for death among children [27]. Diuretics used in the treatment of CHD cause increased urinary sodium, potassium, chloride, and calcium losses [28].

Regarding serum electrolytes levels pre and post to nutritional support, our results showed that there was a significant increase regarding serum calcium, phosphorus, and magnesium levels, while serum sodium and potassium levels increased but without significant difference indicating that our cases of CHD need further supplementation.

Nutritional deficiency may not be directly associated with anemia; however, it leads to certain changes in the body that make it susceptible to health hazards that may cause anemia. Children suffering from nutritional deficiency are more likely to have weaker immune systems which make them vulnerable to various illnesses and health hazards such as parasitic infections or chronic inflammation. Various factors influencing the anemia of malnourished infants including metabolic changes in the red cell, protein deficiency, adaptation anemia, iron deficiency, deficiency of vitamins (folic acid, B12, E, pyridoxine, riboflavin) or trace elements (copper, selenium, zinc), erythropoietin deficiency, infection, and chronic diseases [29].

Regarding CBC parameters pre and post to nutritional support, our results showed that there was a significant improvement in hemoglobin levels and hematocrit percent. In agreement with our findings, El-Koofy N et al. [17] reported that there was a significant increase in the mean level of hemoglobin after nutritional counseling and this leads to a decrease of anemia from 96% of cases to 80%. Hassan BA et al. [30] found a significant correlation between anemia and malnutrition. Also, Labib NA et al [20] found a statistically significant difference regarding malnutrition between anemic 95 (32%) and non-anemic 205 (68%). In the study done by Abdelmoneim HM et al. [31] they found that cases with CHD were having low hemoglobin concentration than control and reported that the significant contributing factors of malnutrition were poor dietary history, anemia, heart failure and pulmonary hypertension.

In the present study, there was a significant reduction of platelet count post to nutrition support, while there were no significant changes in WBCs count. Üner A et al. [32] found that platelet counts were increased before nutrition support as compared with after therapy. This finding considered that diminished platelet function has been balanced by increased platelet numbers. Mean platelet volume was also significantly decreased in patients with

PEM. These low values reached normal values after the patients recovered.

CONCLUSIONS

Nutritional intervention is very important to children with CHD. It prevents malnutrition which is a dangerous problem for them.

Conflict of interest: The authors declare there are no competing interests.

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