Growth and Nutritional Assessment in Pediatric Patients with Congenital Heart Diseases A cross sectional study

Dr. Inas Refay El-Sayed
Prof. Dr. Hanan Abd-Allah El-Gamal
Professor of Pediatrics
Institute of Post-graduate Childhood Studies
Ain Shams University
Prof. Dr. Ayman Mansour
Professor of Pediatrics
National Research center
Dr. Mahmoud Mohamed Nafie
Lecture Of Clinical Pathology
Ain Shams University

Abstract:

Detection of the impact of the presence of cardiac lesions on physical growth of pediatric patients suffering from CHD, investigating the association of gender effect and type cardiac lesions with the affection of growth indices.

Patients and methods:

A cross sectional study of 300 pediatric patients with established congenital heart disease chosen randomly from the Pediatric Cardiology Department, Cairo University, and National Cardiac Institute within one year. Congenital cardiac defects were diagnosed on the basis of two -dimensional echocardiography. The studied patients were selected randomly from Pediatric patients who were on conservative treatment and not submitted to definitive or palliative heart surgical correction. The study comprised also 50 apparently normal healthy age, sex & culture matched children; serving for control purposes, and they were obtained from preschool nursery. All studied patients were subjected to thorough history taking, physical examinations including anthropometric measures, laboratory investigations, imaging studies including echocardiography, radiological and electrocardiography. Karyotyping and FISCH technique were performed for dysmorphic patients. Data were statistically analyzed using SPSS package software version 14.0©.

Results:

This study was performed on 300 sick outpatients' infants and children suffering from congenital heart diseases (group I).

Our patients were subdivided into 2 subgroups:

- Subgroup IA which included patients with congenital cyanotic heart diseases 120 patients (40%).
- Subgroup IB which included patients with congenital acyanotic heart diseases 180 patients (60%).

Clinical examination revealed that:

- **(46) cases were dysmorphic.**
- **Growth retardation was evident in both cyanotic and acyanotic cases when compared to the controls.**
- **Weight retardation was more frequent and more evident than height retardation. Delayed and retarded development was of greater severity in males than females.**

Conclusion:

Pediatric Patients suffering from CHD are more prone to growth retardation. Weight retardation was more frequent and more evident than height retardation in all studied cases and was more pronounced in those with cyanosis. Early diagnosis and timely intervention will reduce the morbidity and mortality to a large extent. The maintenance of nutritional homeostasis is fundamental to normal health. They need regular monitoring so as to permit optimal growth and development.

Introduction:

Congenital heart diseases (CHD) are common pediatric problems that exist primarily at birth. They are characterized by gross structural abnormalities of the heart or the great vessels that actually interfere with normal cardiac function[1]. The general incidence is about 6:1000. Males and females are equally affected. Approximately 30% of cases are associated with congenital malformations and 10% with extracardiac malformations[2]. The known causes of congenital heart disease are scanty and largely based on small studies. Potentially identified factors include both environmental and genetic factors[3].

Congenital heart defects are classified into two broad categories: cyanotic and cyanotic lesions. Congestive heart failure and repeated chest infection are the primary concern in infants with a cyanotic lesions. Difficulty in feeding is common and is often associated with tachypnea, sweating and subcostal retraction[4]. In mild or early cases, central cyanosis is mainly exertional and appears only during crying, suckling or exertion. In established cases, cyanosis is constant and deepens with exertion. In longstanding cases, (dating more than 6 months), growth failure and clubbing of fingers occur[5].

Many factors contribute to growth impairment in infants with CHD, including a lower than normal birth weight, increased caloric requirements, reduction in adipose stores, feeding difficulty[6], and the presence of concomitant musculoskeletal, central nervous system, renal or gastrointestinal malformations. Chronic hypoxia is reported to affect growth. Mild steatorrhea and excess protein loss are common in infants with CHD[7].

Patients and methods:

Three hundreds pediatric patients with established congenital heart disease were chosen from the clinics of both Pediatric Cardiology Department, Cairo University and National cardiac institute within one year, starting from October 2008 to September 2009. Congenital cardiac defects were diagnosed by two dimensional echocardiography. According to history and physical examination, we found that the maximum number of cases with CHD was diagnosed early in infancy as having CHD. They were subdivided into 2 subgroups; 120 cases (40%) in subgroup IA with congenital cyanotic heart diseases and 180 cases (60%) with congenital acyanotic heart diseases in subgroup IB. The subgroup IA included 71 (59.2%) males and 49 (40.8%) females (M: F ratio of 1.45: 1), and the subgroup IB included 101 (56.1%) males and 79 (43.9%) females (M: F ratio of 1.27:1). They are compared with control group (group II) having the same age, sex, and culture. The group II considered 50 apparently healthy children. All studied patients were subjected to thorough history taking, including

onset of cyanosis, hypercyanotic spells, tachypnea, feeding difficulty, poor weight gain, repeated chest infections, skin infection, gastroenteritis, congestive heart failure, previous hospitalization, drug intake during pregnancy, family history and positive consanguinity. Information on socioeconomic level, parental education status, birth weight and nutrition history, number of siblings, and the timing, quality, and quantity of nutrients ingested during weaning period and at the time of the examination were obtained through interviews with parents.

The studied patients were subjected to comprehensive physical examination, checking their weight, length, head circumference, and physical signs of malnutrition, such as skin lesions, and thin and weak hair. Thereafter, BMI, as well as growth percentiles and Z- scores was calculated using CDC-2000 reverence values. Laboratory investigations entailed a complete blood picture, mainly differential white blood cell count, hemoglobin, and hematocrite concentration. Karyotyping and FISCH technique were performed for dysmorphic patients. The obtained data were compared to those of the control group which comprised 50 normal healthy ages, sex, and culture matched subjects. All studied data were statistically analyzed using SPSS package software version 14.0©. The local ethical committee approved the study protocol. Informed consents were obtained from the parents of the subjects.

Results:

This study was performed on 300 sick outpatients' infants and children suffering from congenital heart diseases (group I). Our patients were subdivided into 2 subgroups; subgroup IA which included patients with congenital cyanotic heart diseases 120 patients (40%) & subgroup IB which included patients with congenital acyanotic heart diseases 180 patients (60%).

Table (1) showed important physical findings of the study patients. The major clinical presentations were breathlessness (84%) both in subgroup IA and subgroup IB, tachycardia, (53.4%), anemia (63.4%), clubbing in 75 cases (25%), and polycythemia (23.3%).

Cyanosis was the main reason for referral to Pediatric Cardiology department that was present in 120 cases (40%), heart failure in 252 (84%), tachypnea in 90 cases (30%), poor suckling, and poor weight gain in 78 cases (26%).

Infective endocarditic was present in 18 cases (6%), pleural effusion in 3.3%, pericardial effusion in 8%, staphylococcal skin infection in 13 cases (4.3%), and history of neonatal septicemia in 6 cases (2%).

As regards associated congenital malformations, we found that:

- (46) cases were dimorphic.
- (35) cases had Down syndrome as proved by karyotyping.
- (2) cases had Williams' syndrome as proved by fluorescent in situ hybridization.
- (3) cases had Marfan syndrome.
- (2) cases had Alagille's syndrome.
- (1) case had Holt oram syndrome.
- (1) case had skeletal dysplasia
- (1) case had congenital hemihypertrophy syndrome.
- (1) case was diagnosed as Apert syndrome.

Table (1): Important physical findings of the study patients (N=300).

Physical Findings	Group I (N=300)		Subgroup IA (N=120)		Subgroup IB (N=180)	
	No. Of Patients	Percentage (%)	No. Of Patients	Percentage (%)	No. Of Patients	Percentage (%)
Dyspnea	252	84%	100	33. 4%	152	50.6%
Heart Failure	160	53.4%	55	18.4%	105	35%
Tachypnea	90	30%	30	10%	60	20%
Crepitation	90	30%	30	10%	60	20%
Cyanosis	120	40%	120	40%	-	-
Polycythemia	70	23.3%	70	23.3%	-	-
Anemia	190	63.4%	50	16.8%	140	46.6%
Enlarged Tender Liver	60	20%	10	3.4%	50	16.8%
Clubbing	75	25%	75	25%	-	-
Rhonchi	54	18%	28	9.34%	26	8.6%
Lower Respiratory Tract Infections	207	69%	69	23%	138	46%
Engorged Neck Veins	60	20%	10	3.4%	50	16.8%
Hypertension	16	5.3%	-	-	16	5.3%
Radio-Femoral Delay	16	5.3%	-	-	16	5.3%
Infective Endocarditis	18	6%	6	2%	12	4%
Pleural Effusion	10	3.3%	7	2.3%	3	1
Pericardial Effusion	24	8%	6	2%	18	6%
Staph.Skin Infection	13	4.3%	8	2.7%	5	1.7%
Neonatal Septicemia	6	2%	1	0.34%	5	1.66%
Down Syndrome	35	11.66%	-	-	35	11.6%
Genetic Syndrome	11	3.6%	-	-	11	3.6%

Basic data of the studied patients and comparison between subgroup IA, and subgroup IB were described in table (2).

There was a history of positive consanguinity in 108 cases (36%) in group I; 24% of patients having congenital cyanotic heart diseases, while 12% of patients having congenital a cyanotic heart diseases, and the difference were statistically significant. Positive family history of the same congenital cardiac lesions was present in 13 (4.4%) cases; 9 cases (3%) in subgroup IA, and 4 (1.3%) cases in subgroup IB.

A history of a +ve history of drug intake during pregnancy was present in 57(19%) cases; 33 cases (11%) in subgroup IA, and 24 (8%) cases in subgroup IB, and the difference was statistically significant.

There was a history of previous hospitalization in 207 patients (69%), which is significantly higher in subgroup IB. Positive history of gastroenteritis was present in 16%, and skin infections in 4.4%.

The annual incidence of respiratory infections in the acyanotic group was 40% which is significantly higher than that in the cyanotic group 21%, and the difference was statistically significant.

Table (2) : Basic data of the study patients with the comparison between subgroup IA, and subgroup IB.

Variables	Group I	Subgroup IA	Subgroup IB	X^2	P-Value	Odd' S Ratio	(95%Ci)
+Ve History Consanguinity	108 (36%)	72 (24%)	36 (12%)	4.07	0.044*	0.611	0.378 - 0.988
+ve Family history of cardiac lesion	13 (4.4%)	9 (3%)	4 (1.3%)	4.83	0.028*	0.28	0.084 - 0.932
+ve drug intake during pregnancy	57 (19%)	33 (11%)	24 (8%)	9.38	0.002*	0.406	0.225- 0.736
+Ve History Antifailure Medications	156 (53.3%)	87 (29%)	69 (23%)	29.52	0.001*	0.259	0.157- 0.426
+ve Previous history of hospitalization	207 (69%)	87 (29%)	120 (40%)	15.02	0.001*	0.345	0.199- 0.598
+Ve History Of Pneumonia	183 (61%)	63 (21%)	120 (40%)	8.12	0.005*	0.492	0.381- 0.804
+Ve History Of Gastroenteritis	48 (16%)	23 (7.7%)	25 (8.3%)	1.49	0.26	0.689	0.366- 1.265
+ve history of skin infections	13 (4.4%)	4 (1.4%)	9 (3%)	0.48	0.57	1.52	0.459- 5

Table (3), (4) show two dimensional echocardiography diagnoses of the study patients with comparison between males and females (N=300).The echocardiogram was diagnostic in all cases. Both M-mode, 2-D and Doppler echocardiography with color coding examinations were used. Among the identified medical diagnoses, the most frequent congenital diagnoses were: Interventricular communication (42%), followed by transposition of the great arteries (9%), persistence of the arterial channel (26%), Interatrial communication (5.6%), Coarctation of the aorta (16%), Tetralogy of Fallot (12%), Pulmonary stenosis (10.3%) and Total anomalous pulmonary venous drainage (1.6%) and others.

Table (3), (4): Two dimensional echocardiographic diagnoses of the study patients with comparison between males and females (N=300).

Cardiac Lesions	Male	Female	Total No.	Percent%	X2	P-Value
D- Tga	9	1	10	3.3%	34.72	0.05*
D- Tga +Pda	7	2	9	3%	3.2	0.03*
D- TGA +DORV+PS	5	4	9	3%	4.54	0.677
D- TGA +DORV+PAPVR	1	-	1	0.3%	7.63	0.43
T- TGA	2	1	3	1%	15.51	0.41
Fallot Tetrology	19	5	24	8%	17.09	0.03
Fallot tetrology+ PA+PDA	6	2	8	2.7%	13.91	0.05*
Fallot Tetrology+ PDA	1	-	1	0.3%	11.2	0.37
Pulmonary Atresia With IVS	2	3	5	1.6%	1.2	0.3
Pulmonary Atresia With PDA	2	1	3	1%	0.056	0.13
Tricuspid Atresia			4	1.3%	2	0.18
Ebstein Anomly	6	1	7	2.3%	9.18	0.042*
Truncus Arteriosus	2	2	4	1.3%	6.08	0.86
Single Ventricle	4	1	5	1.66%	3.39	0.089
Total Anomalous Pulmonary Venous Connection	3	2	5	1.66%	7.62	0.3
DORV	5	5	10	3.3%	23.65	0.87
Dorv+Ps	3	3	6	2%	4.79	0.64
Dextrocardia+other congenital cardiac anomalies	6	2	8	2.7%	0.05*	1.75
Aortopulmonary Window	1	-	1	0.3%	7.63	0.43
Coronary Arteriovenous Fistula	1	-	1	0.3%	15.51	0.41

* Echocardiographic findings of Subgroup IA

Cardiac Lesions	Male	Female	Total No.	Percent%	X2	P-Value
Patent ductus arteriosus (PDA)	8	18	26	8.6%	2.26	0.01*
Ventricular septal defect (perimembraneous)	15	27	42	14%	9.45	0.022*
Ventricular septal defect (muscular type)	3	9	12	4%	7.91	0.014*
Ventricular Septal Defect With PDA	5	10	15	5%	9.75	0.03*
Atrioventricular Septal Defect	1	4	5	1.6%	9.05	0.028*
Atrial Septal Defect	6	11	17	5.6%	16.13	0.01*
PAPVR	3	3	6	2%	2.59	0.25
Pulmonic Stenosis	11	20	31	10.3%	2.57	0.001*
Congenital Mitral Valve Prolapse	3	13	16	5.3%	7.91	0.034*
Aortic Stenosis	12	2	14	4.7%	9.75	0.028*
Coarctation Of Aorta	13	3	16	5.3%	11.26	0.047*

* Echocardiographic findings of Subgroup IB

Echocardiography revealed a male preponderance for D- TGA, aortic stenosis, coarctation of the aorta, Ebstein anomly, and Fallot tetralogy, whereas pulmonary stenosis, patent ductus arteriosis, atrial and ventricular septal defect are more common in females.

Table (5) displayed comparison of anthropometric measurements between patients in subgroup IA, and IB. In subgroup IA, there were statistically significant difference between male and female as regards weight for age, height for age, weight for height percentile and Z- score. The mean of significant difference was decrease as regards weight for age, height for age, and weight for height percentile, but there was no significant difference between male and female in subgroup IB.

Table (5): Comparison of anthropometric measurements between males and females in subgroup IA and subgroup IB

variables	Subgroup IA					Subgroup IB				
	Male		Female		P-Value	Male		Female		P-Value
	Mean	SD	Mean	SD		Mean	SD	Mean	SD	
Age(Mon.)	17.18	12.11	18.30	10.46	0.008*	32.37	23.2	36.69	26.85	0.04*
Ht /Age Percentile	8.62	16.51	10.26	18.59	0.01*	9.37	16.87	8.22	13.50	0.54
Wt /Age Percentile	8.96	12.79	13.80	18.92	0.05*	11.69	15.48	10.97	13.70	0.89
Wt/Ht Percentile	41.24	29.88	41.06	31.84	0.057*	39.48	32.76	39.03	31.91	0.81
Ht /Age Zscore	-2.07	1.29	-1.7	1.40	0.15	-1.80	1.27	-1.69	1.44	0.58
Wt /Age Zscore	-1.60	0.981	-1.38	1.17	0.068	-1.49	1.044	-1.68	.912	0.14
Wt/Ht Zscore	-0.40	1.084	-0.31	1.27	0.67	-0.51	1.24	-.37	1.244	0.53
BMI	15.97	2.357	16.08	2.10	0.65	15.70	2.64	15.80	2.138	0.23

Table (6): showed the growth percentiles in group I and group II. In group I, according to the height for age percentile, we found that, 57% were severely malnourished (less than 5th percentile), while 43% were severely malnourished according to the weight for age percentile. Meanwhile weight for height percentile revealed that, 34% were severely malnourished. We showed also, according to the height for age percentile, 25% were border line (between 5^{th} and 10^{th}), 35% were border line according to the weight for age percentile and the rest were normal. Retardation in height and weight was noted in all children with cardiac disease but was more pronounced in those with acyanotic. Stunted growth was more evident than under weight, indicating chronic malnutrition.

Table (6): Growth percentiles in group I and group II.

Groups	Variables	< 5th Percentile		< 5th -< 10th Percentile		>10th -<25th Percentile		25th -50th Percentile		>50th Percentile	
Group I	Ht/Age Percentile	171	57%	75	25%	84	28%	9	3%	9	3%
	Wt/Age Percentile	129	43%	105	35%	72	24%	42	14%	3	1%
	Wt/Ht Percentile	102	34%	21	7%	48	16%	66	22%	45	15%
Subgroup IA	Ht/Age Percentile	63	21%	24	8%	30	10%	3	1%	-	-
	Wt/Age Percentile	42	14%	39	13%	24	8%	12	4%	3	1%
	Wt/Ht Percentile	21	7%	24	8%	27	9%	48	16%	-	-
Subgroup IB	Ht/Age Percentile	90	30%	42	14%	42	14%	6	2%	-	-
	Wt/Age Percentile	72	24%	51	17%	36	12%	21	7%	-	-
	Wt/Ht Percentile	33	11%	27	9%	30	10%	48	16%	42	14%
Group II.	Ht/Age Percentile	-	-	-	-	18	36%	16	32%	16	32%
	Wt/Age Percentile	-	-	-	-	20	40%	15	5%	15	30%
	Wt/Ht Percentile	-	-	-	-	16	32%	20	40%	14	28%

Table (7) showed Z-score for group I and group II. In group I, we found that, weight for age Z-score <or= -2 in 51%, height for age Z-score <or= -2 in 50%; and weight/height Z-score <or= -2 in 41%. In group I, according to the height for age Z-score, we found that, 50% were severely malnourished Z-score <or=-2 while 51% were severely malnourished according to the weight for age Z-score

Meanwhile weight for height Z-score revealed that, 41% were severely malnourished. We showed also, according to the height for age Z-score, 15% were mildly malnourished (Z-score less than -2, and more than -1), 20% were mildly malnourished according to the weight for age Z-score and the rest were normal. Retardation in growth indices was noted in all children with cardiac disease, but was more pronounced in those with a cyanotic, where stunted growth was more evident than under weight, indicating chronic malnutrition.

Table (8): Z- score for group I and group II.

Groups	Variables	Z- Score <-3		Z- Score <-2,-3		Z- Score -2, ≤ -1		Z- Score -1, ≤ 2	
		No.	%	No.	%	No.	%	No.	%
Group I	Wt /Age Z-score	69	23%	84	28%	60	20%	99	33%
	Ht /Age Z-score	63	21%	87	29%	45	15%	90	30%
	Wt/Ht Z-score	57	19%	66	22%	78	26%	120	40%
Subgroup IA	Wt /Age Z-score	15	5%	27	9%	27	9%	51	17%
	Ht /Age Z-score	6	2%	24	8%	30	10%	60	20%
	Wt/Ht Z-score	18	6%	9	3%	30	10%	60	20%
Subgroup IB	Wt /Age Z-score	27	9%	54	18%	33	11%	99	33%
	Ht /Age Z-score	32	8%	45	15%	39	13%	96	32%
	Wt/Ht Z-score	18	6%	21	7%	57	19%	114	38%
Group II	Wt /Age Z-score	-	-	-	-	-	-	50	100%
	Ht /Age Z-score	-	-	-	-	-	-	50	100%
	Wt/Ht Z-score	-	-	-	-	-	-	50	100%

Table (9): Comparison of anthropometric measurements between patients in subgroup IA, IB and group II.

Variable	Subgroup IA N=120 Mean± SD	Subgroup IB N=180 Mean±SD	Group II N=50 Mean± SD	F	P
Ht /Age Percentile	7.96±1.26	7.47±4.54	10.9±4.27	1.66	0.19
Wt /Age Percentile	10.44±4.48	9.71±5.1	78.0±14.38	3.19	0.042*
Wt/Ht Percentile	30.08±20.81	33.23±27.2	46.2±2.8	2.19	0.13
Ht /Age Z- Score	-2.02±.119	-1.83±1.35	1.1±0.21	5.34	0.005*
Wt /Age Z- Score	-1.58±.136	-1.79±1.03	1.5±0.24	4.77	0.009*
Wt/Ht Z- Score	-1.80±.129	-0.80±0.12	2.0±0.8	3.65	0.027*
BMI	16.00±2.17	15.80±2.1	18.34±18.95	2.88	0.057*

By using analysis of variance "ANOVA" table (6) shows that compared cyanotic group with non cyanotic group, and control group as regards growth indices, we can found that, there were a higher significant difference between the three groups ($p<0.01$) as regards weight for age percentile, weight for age Z-score, height for age Z-score, weight for height Z-score, and body mass index.

Discussion:

Congenital heart diseases represent one of the important factors which retard the growth rate of different parts of human body. This is attributed to multifactorial causes. Inadequate caloric intake, malabsorption, and increased energy requirements caused by increased metabolism may all contribute. Our present study included 300 patients with CHD, 120 patients having congenital cyanotic cardiac lesions and 180 patients having acyanotic cardiac lesions. The mean age range was 2.5- 120 months. They were compared with a control group (50 children) of apparently normal healthy, age, sex, and culture matched subjects. Statistical analysis of our data revealed that, growth failure was evident in both cyanotic and in acyanotic CHD when compared to the controls.

A history of a positive consanguinity was present in 108 (36%) cases with statistically significant value with respect to the control; 36 cases (12%) in subgroup IB, and 72 cases (24%) in subgroup IA, and the difference between the two subgroups was statistically significant.

Amongst our studied patients, 46 cases were dysmorphic, 35 cases had Down syndrome, two cases had Williams' syndrome, 3 cases had Marfan syndrome, 2 cases had Alagille's syndrome, 1 case had Holt oram syndrome, 1case had skeletal dysplasia, 1 case had congenital hemi hypertrophy syndrome and 1 case was diagnosed as Apert syndrome. Olson, et al.,[8] have reported 87.92% as classic trisomy 21, mosaic in 7.69% and translocation in 4.39% with significant contribution of advanced parental age. Phenotypic changes of genetic diseases at birth include congenital malformations in chromosomes and single gene defects. However, the most common defects are usually multifactorial and result from a combination of genetic predisposition and environmental stimulus[9].

Positive family history with the same congenital cardiac lesions was present in 13 (4.4%) cases; 9 cases (3%) in subgroup IA, and 4 (1.3%) cases in subgroup IB. There was a history of maternal drug intake during pregnancy other than calcium and folic acid in 57 (19%), 8 of them due to chronic maternal illness, while the others had a past history of threatened abortion, and they received prescribed medications in the first trimester to continue their pregnancy and prevent fetal loss. This is in accordance with studies by[10].

There were gender differences in the occurrence

of congenital heart diseases as a whole that occurred with equal frequency in males and females, but some lesions such as aortic stenosis, coarctation of the aorta, transposition of the great arteries and Fallot tetralogy were more common in males with statistically significant p value, whereas pulmonary stenosis, patent ductus arteriosis, atrial and ventricular septal defect are more common in females, and the gender difference between them was statistically significant. This is in accordance with studies by[11].

In our present study, the left ventricular configuration and increased cardiothoracic ratio were the main radiological findings of the patients and it was present in 84% of patients. The pulmonary vasculature was increased in 210 patients (70%). The electrocardiographic findings of the patients showed that all cases were in sinus rhythm and had left ventricular hypertrophy (84%). The echocardiogram was diagnostic in all cases. Both M-mode, 2-D and Doppler echocardiography with color coding examinations were used. Among the identified medical diagnoses, the most frequent congenital diagnoses were: Interventricular communication (42%), followed by transposition of the great arteries (9%), persistence of the arterial channel (26%), Interatrial communication (5.6%), Coarctation of the aorta (16%), Tetralogy of Fallot (12%), Pulmonary stenosis (10.3%) and Total anomalous pulmonary venous drainage (1.6%) and others. This is in accordance with studies by[12].

In our study, the clinical presentation of patients in group I varied according to the type and severity of the defect. In neonatal period, the main presenting features were cyanosis (40%) (with or without respiratory distress), heart failure in 252 (84%) (with or without cyanosis), tachypnea in 90 cases (30%), crepitation in 30%, and an abnormal clinical sign detected on routine examination (e.g., absent femoral pulse or a heart murmur) in 16 cases (5.3%). Clubbing was present in 75 cases (25%). There was a history of recurrent chest infections in 207 patients (69%). The other common form of infections were infective endocarditis, that was present in 18 cases (6%), pleural effusion in 10 cases (3.3%), pericardial effusion in 24 cases (8%), staphylococcal skin infection in 13 cases (4.3%), and history of neonatal septicemia in 6 cases (2%). Cardiac findings revealed murmur with or without thrill and cardiomegaly (84%) as the most frequently observed feature. Breathlessness was the commonest symptom both in cyanotic and acyanotic heart disease. In our study, we found breathlessness in 84%, and congestive heart failure in 84% who were receiving anti-failure medications, as congestive heart failure is a common problem in patients with large shunts. These percentage were higher than those reported by[13] who found that, the most common clinical presentations were breathlessness (69%), lower respiratory tract infection (LRTI) (52%), congestive cardiac failure (CCF) (46%), cyanosis (20.2%), cyanotic spell (9.5%) and infective endocarditis (9.5%).

Failure to thrive (FTT) and growth delay is a major complication of CHD, the reason being high energy expenditure, inadequate food intake, and malabsorption, and or feeding difficulty. In addition, many children with congenital heart disease present nutritional difficulties during the first year of life, with vomiting as one of the most common problems. FTT and growth delay was seen in (89.9%) cases, this is in accordance to that reported by (13) as only 86.9% of their patients presented the previous complaint.

In our present study, we found that the maximum number of cases with CHD were picked up in infancy in 206 cases (68.7%), which was higher percentage compared to that reported by[14], who

found that, out of 84 CHD cases, CHD presented more frequently during infancy (46%).

Predictors of malnutrition for our patients were documented as weight for age Z-score <or= -2 in 51%, height for age Z-score <or= -2 in 50%; and weight/height Z-score <or= -2 in 41%. In group I, according to the height for age Z-score, we found that, 50% were severely malnourished (Z-score <or= -2), while 51% were severely malnourished according to the weight for age Z-score. Meanwhile weight for height Z-score revealed that, 41% were severely malnourished. We showed also, according to the height for age Z-score, 15% were mildly malnourished (Z-score less than -2, and more than -1), 20% were mildly malnourished according to the weight for age Z-score and the rest were normal. Retardation in growth indices was noted in all children with cardiac disease, but was more pronounced in those with acyanotic, where stunted growth was more evident than under weight, indicating chronic malnutrition. This might have occurred, because most of the study patients had severe cardiac lesions and were from families of a low socioeconomic level. Our findings concerning malnutrition are in accordance with[15].

In group I, according to the height for age percentile, we found that, 57% were severely malnourished (less than 5th percentile), while 43% were severely malnourished according to the weight for age percentile. Meanwhile weight for height percentile revealed that, 34% were severely malnourished. We showed also, according to the height for age percentile, 25% were border line (between 5th and 10th) , 35% were border line according to the weight for age percentile and the rest were normal. Retardation in height and weight was noted in all children with cardiac disease but was more pronounced in those with acyanotic. Stunted growth was more evident than under weight, indicating chronic malnutrition[16].

Davis, et al.,[17], found that, the cause of growth retardation in CHD was multifactorial. Inadequate caloric intake, malabsorption, and increased energy requirements caused by increased metabolism might all contribute. However, inadequate caloric intake appears to be the most important cause.

On the other hand, by using analysis of variance "ANOVA" comparing cyanotic group with non cyanotic group, and control group as regards growth indices, we can found that, there were a higher significant difference between the three groups ($p<0.01$) as regards weight for age percentile, weight for age Z-score, height for age Z-score, weight for height Z-score, and body mass index.

Retardation in height and weight was noted in our patients as a whole, but was more pronounced in those with acyanosis. Weight retardation was more frequent and more marked than retardation in height. Delayed and retarded development was of greater severity in boys than in girls. Our results coincide with[18], who found that, delay in weight maturation is considerably greater than linear growth in acyanotic group, while in the cyanotic group height and weight tend to be parallel to each others.

Conclusion:

Patients with CHD are prone to malnutrition and growth failure. Anthropometric measures are important health status indicators when assessing the nutritional state, and growth of these children, as they help to diagnose nutritional alterations, and to determine the prognosis. Retardation in height and weight was noted in all children with cardiac disease but was more pronounced in those with acyanotic. Stunted growth was more evident than under weight, indicating chronic malnutrition. Delayed and retarded development was of greater severity in boys than in girls. Early diagnosis and timely intervention will reduce the morbidity and mortality to a large

extent. The maintenance of nutritional homeostasis is fundamental to normal health. They need regular monitoring so as to permit optimal growth and development.

References:

1. Allen, Hugh D.; Driscoll, David J.; Shaddy, Robert E.; Feltes, Timothy F. Moss and Adams' **Heart Disease in Infants, Children, and Adolescents: Including the Fetus and Young Adults**, 7th Edition, 2008.
2. Knopper, Melissa, and Teresa Godle: "Congenital Heart Disease." **Gale Encyclopedia of Medicine** 2004
3. Pajkrt, E, et al: Fetal Cardiac Anomalies and Genetic Syndromes. **Prenatal Diagnosis**, 2004, volume 24, pages 1104-1115.
4. Robert H. E, Anderson JB, Ferguson J, et al: **Pediatric Cardiology**, 2nd ed.. Philadelphia, Churchill Living stone 2001.
5. McKesson et al: "**Congenital Heart Disease**." Clinical Reference Systems. McKesson Health Solutions LC, 2004. pg 783. Health Reference Center-Academic. 20 Feb. 2006.
6. Silva VM, Lopes MV, Araujo TL: **Evaluation of growth percentiles in children with congenital heart disease**. Rev Latino-Am Enfermagem; 2007; 15(2): 298-303.
7. Weintraub RG, Menahem S: **Growth and congenital heart diseases**. J Paediatr Child Health 2004; 29:95-8.
8. Olson LE, Richtsmeier JT, Leszl J, Reeves RH (2004)."A chromosome 21 critical region does not cause specific Down syndrome phenotypes". **Science Journal** 306 (5696): 687-90.
9. Bernstein D. **Congenital heart disease**. In Behrman RE, Kligman RM, Jenson HB, 17th ed. Nelson textbook of Pediatrics. Philadelphia: Saunders, 2004; 1499-1553.
10. Chadha SL, Singh N, Shukla DK. Epidemiological study of congenital heart disease. **Indian J. Pediatr** 2001;68:507-10.
11. Kasturi L, Kulkarni AV, Amin A, Mahashankar VA. Congenital heart disease: clinical spectrum. **Indian J. Pediatr**. 1999;36:953.
12. Begum NF, Ahmed QS. **Pattern of Heart disease among neonates and their outcome: one year experience in non- invasive cardiac laboratory of Combined Military Hospital**, Dhaka. Bangladesh J child health 2008; 25: 48-52.
13. Bloomfield P, Bradbury A, Grubb NR, Newby DE. **Cardiovascular Disease**. In: Boon NA, Colledge NR, Walker BR, 20th ed. Davidson's Principle and Practice of Medicine. Edinburgh: Churchill Livingstone, 2006; 519-646
14. Kitchiner D J. **Cardiovascular disease**. In: Mclntosh N, Helms PJ, Smyth RL, 6th ed. Forfer & Arneil's Textbook of Pediatrics. Edinburgh: Churchill Livingstone, 2003; 815-888
15. Vaidyanathan B, Nair SB, Sundaram KR, Babu UK, Shivaprakaste K, Rao SG, et al. Malnutrition in children with congenital heart disease (CHD): determinants and short-term impact of corrective intervention. **Indian J. Pediatr** 2008; 45:541-546.
16. Garza C, De Onis M: Rationale for developing a new international growth reference. **Food Nutr Bull** 2009; 25(1):S5-S14.

الملخص

تقييم النمو فى الاطفال المرضى بعيوب خلقية بالقلب

هذه الدراسة تهدف إلى تقييم النمو البدنى للطفل المصرى وإلى دراسة تأثير نوعية الأصابة القلبية على النمو والتغذية، من خلال قياس الوزن والطول والارتفاع، ومحيط الرأس، و تقييم وتيرة سوء التغذية لدى الأطفال المصابين بعيوب خلقية فى القلب، ومقارنتها مع الأطفال الأصحاء من نفس الفئة العمرية والجنس والثقافة والعلاقات المتبادلة بين جميع البارامترات التى درست بما فى ذلك التدابير أنثروبومترية للأطفال مع المئينات التى تمثل مؤشرات نموها، والسريرية والمختبرية، والبيانات. وتشمل الدراسة ٣٠٠ من الاطفال المرضى الذين يعانون من أمراض عيوب القلب الخلقية، ١٢٠ من المرضى الذين يعانون من العيوب الخلقية بالقلب المصحوبة بوجود زرقة و١٨٠ من المرضى الذين يعانون من العيوب الخلقية بالقلب الغير مصحوبة بوجود زرقة، وتتراوح الفئة العمرية من ١-١٢٠ أشهر، وتم مقارنة هذة الحالات مع المجموعة االضابطة المكونة من (٥٠ طفلا) والذين لديهم نفس العمر ومن نفس الثقافة والجنس والحالة الاجتماعية.

ولقد تبين من التحليل الإحصائى للبيانات، أنه يوجد خلل واضح فى نمو الاطفال المصابين بعيوب خلقية بالقلب بنوعيها من حيث الوزن والطول ووجد ايضا أن الذكور كانوا أكثر المتخلفين فى الوزن أكثر من الإناث وقد وجد ان الخلل فى النمو من حيث الطول والوزن كان اكثر وضوحا فى نمو الاطفال المصابين بعيوب خلقية بالقلب الغير مصحوبة وجود زرقة عن نمو الاطفال المصابين بعيوب خلقية بالقلب المصحوبة وجود زرقة، اما الاطفال المصابين بعيوب خلقية بالقلب الغير مصحوبة بوجود زرقة عند مقارنتهم بالاطفال الاصحاء المتساويين معهم فى نفس السن والجنس والحالة الاجتماعية فوجد انهم يعانوا من انخفاض ملحوظ فى وزن الجسم وذلك عند مقارنتهم مع الأطفال الأصحاء من نفس الفئة العمرية والجنس والحالة الاجتماعية وهذا الانخفاض فى الوزن موجود بنسبة متساوية فى كلا من الذكور والاناث الموجودين بنفس المجموعة. كما ان الدراسة تشير الى ارتفاع نسبة الاطفال الذين تعرضوا لحدوث فشل فى وظائف القلب. ويلخص البحث مدى اهمية تقييم معدلات النمو فى الاطفال المصابين بعيوب خلقية بالقلب حيث ان هؤلاء الاطفال هم اكثر عرضة لحدوث سوء التغذية وتاخر النمو وسوء الامتصاص وبالتالى تدهور فى حالتهم الصحية.

17. Davis D, Davis S, Cotman K, Worley S, Londrico D, Kenny D, Harrison AM. Feeding difficulties and growth delay in children with hypoplastic left heart syndrome versus d-transposition of the great arteries. **Pediatr Cardiol**. 2008 Mar; 29 (2): 328- 33. Epub 2007; Aug

18. Jolley CD: Failure to thrive. **Curr Probl Pediatr Adolesc Health Care** 2003; 33:183-206.