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Effect of congenital heart disease on child growth

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Abstract

Background: Children with congenital heart disease are prone to malnutrition and growth retardation. **Aim:** To compare growth status between children with CHD and healthy children.

Materials and Methods: This case–control study included 40 children with congenital heart disease and 40 healthy children matched in age and gender. The age was from 3 months to 36 months. Congenital heart disease patients grouped according to cardiac diagnosis: Ventricular Septal defect, Atrial Septal Defect, Tetralogy of Falot, and Transposition of Greater Arteries. Anthropometric measurements of weight (Kilogram), height (Centimeter), and head circumference (centimeter) were measured and recorded for both case and control groups. Descriptive and analytical statistics were performed using the by the Statistical Package for the Social Sciences (SPSS) version 23.0.

Results: Weight, height and head circumference were significantly lower in congenital heart disease children compared to healthy children (p-value <0.05).

Conclusion: Children with congenital heart disease experience early, simultaneous decrease in growth trajectory across weight, length, and head circumference.

Keywords: Children, congenital, disease

1. Introduction

Congenital Heart Defect (CHD) is one of the most common non-communicable diseases that occur due to developmental cardiovascular system in the embryonic stage ^[1]. CHD accounted as third congenital diseases and is a leading cause of infant mortality in the first year of life ^[2]. Its prevalence is about 5 to 8 per 1000 live births that varied in different parts of the world ^[3], but in a recent study the prevalence has been reported to be ranged from 4 to 50 cases per 1,000 live births ^[4]. Regarding to congenital malformations, cardiovascular disorders are the most debilitating diseases and CHDs are the leading causes of organic growth disorders in children, early rapid growth retardation detection in early age and rapid intervention would have a very important role in improving of their conditions. Furthermore, reported that CHD children have more developing growth disorders compared to healthy children ^[1, 2]. The most common form of CHD is ventricular septal defect (VSD) that involved 35% of CHD patients; and the considerable type of cyanotic CHD is Tetralogy of Falot (TOF)^[5]. Generally, several genetic and environmental risk factors introduced for CHD, but in most cases, the reasons are not fully understood ^[4, 5, 6]. These factors may include of mutations, alcohol drinking, using cocaine or abusing certain kinds of drugs during pregnancy, for example, thalidomide drug using by the pregnant mothers increases CHD in fetus ^[6, 7]. Many studies have found that cytokines have a strong effect on feeding, growth, weight and energy intake in patients with CHD ^[8, 9]. Impaired absorption can also play an important role of malnutrition in heart disease; so that, children with CHD have delay in growth due to increased work of cardiopulmonary and consequently, fatigue and loss of appetite, dyspnea, tachypnea, chronic hypoxia directed to malnutrition ^[1, 8, 10]. In addition, a series of serum factors such as leptin, ghrelin and tumor necrosis factor alpha $(TNF-\alpha)$ will be changed in these patients. Consequently, the rate of absorption of nutrients, growth, weight and energy consumption and storage are changing ^[11]. Children with CHD have normal growth when receiving more calories compared to healthy ones [9, 12]. Malnutrition in these effects on the metabolic response to injury and complications and outcomes of cardiac surgery including sepsis, renal dysfunction, necrotizing entero-colitis, hospitalization days ^[13]. And then increases the risk of mortality ^[14].

As we know, CHD divided into four major groups of cyanotic with and without an increase in pulmonary artery pressure and Acyanotic heart defect (also known as non-cyanotic heart defect) with and without an increase in pulmonary artery pressure. In our knowledge about the prevalence of growth retardation in these groups of heart diseases, revealed that the pattern of growth retardation not well understood in the area of the study. Therefore, this study aimed to evaluate the growth status in children with CHD compared with controls to have more accurately assessed towards planning, preventive and treatment of growth disorders in CHD children.

2. Methods

2.1 Study setting: In this case-control study, forty CHD children aged 3 months to 16 months old from those who were referred to Al-imamain Al-kadhumain Medical City, Baghdad city, Capital of Iraq, selected as case group after diagnosis and confirmation their CHD with echocardiography and cardiac catheterization. Following, 50 healthy children collected from those who were referred to Al-imamain Al-kadhumain Medical City for routine checkup, receiving vaccination or having diseases that do not affect their growth. The controls selection was with the consideration of not significant underlying disease which effect on growth disorders. The study performed In Alimamain Al-kadhumain Medical City in Baghdad, during the period of 20-7-2018 to 1-4-2019.

2.2 Inclusion and exclusion criteria: Inclusion criteria were, age from 3 months to 36 months and confirmed CHD by echocardiography and cardiac catheterization. Exclusion criteria were a history of pre-maturity, intrauterine growth retardation, known chromosomal abnormalities and genetic disorders and diseases that effect on growth, such as celiac disease, hyperthyroidism, chronic infection.

2.3 Methods of study: The two groups of children were matched based on age and gender. Children with CHD classified in two groups' accordance with their diseases. The groups were cyanotic and Acyanotic. A Questionnaire was given to the mothers after obtaining a full oral consent from them. Weights of participants, height and head circumference were measured.

2.4 Anthropometric measures: Participants' height, weight and head circumference were determined according to standard anthropometric methods. Participants' height over 2 years of age was measured to the nearest 0.1 centimeters (cm) in bare feet with participants standing upright against a mounted stadio-meter and for the participant lower than 2 years of age height was measured with a wooden scaled table in supine position. Weight was measured to the nearest 0.1 kilogram (kg) with participants lightly dressed using a portable digital scale (Tanita HD 309, Creative Health Products, MI, and USA).Weight of participants lower that 2 years age, measured by Mika Mark recumbent weighing scale made in Japan with an error factor of 10 gr. Head circumference measured with a flexible non-stretchable measuring tape.

2.5. Statistical analysis: After collecting the necessary information, data entered in Statistical Package for the Social Sciences (SPSS) version 23.0. To describe the data of central tendency and dispersion, mean and standard deviation (SD) were used. The correlation estimated with the Fisher's exact test. The level of significance less than 0.05 considered statistically significant.

3. Results

This case-control study conducted to evaluate growth indicators in children with congenital heart disease compared with controls. Table 1 showed the patients mean age (17.95 \pm 9.84 months) compared with controls' mean age (17.88 \pm 9.89 months), and resulted similarity (p=0.172). Also, the mean age of the patients' and the controls' mother was (30.62 \pm 7.51years) and (24.96 \pm 4.72 years) respectively. While the mean age of the patients' and controls' father was (33.60 \pm 8.40 years) and (27.43 \pm 5.35 years) respectively.

		Mean	Median	Std. Deviation
Age from 3 to	Controls	17.8825	16.0000	9.89280
36 months	Cases	17.9500	17.0000	9.84222
Mother age	Controls	24.9693	24.0000	4.72790
	Cases	30.6250	30.0000	7.51985
father age	Controls	27.4372	27.0000	5.35544
	Cases	33.6000	30.0000	8.40269

Table 1: Mean Age of the children and their parents.

Table 2: showed sex distribution, in patients 20 (50.0%) were girls and 20 (50.0%) were boys and in the controls, 20 (50.0%) were girls, and 20 (50.0%) were boys; the study shows there is no sex predilection for CHD (p-value=0.18). Regarding the maturity, in controls, 27(67.5%) mature and 13(32.5%) premature while in cases, 18(45.0%) mature and 22(55.0%) premature, so CHD were more common in premature children (55%) (P-value=0.11). Regarding the mode of delivery, in controls, 18(45.5%) delivered by caesarean section and 22(55.5%) while in cases, 27(67.5%) delivered by caesarean and 13(32.5%) delivered vaginally,

so CHD were seen more commonly in products of caesarean section (67.5%) than normal vaginal delivery (32.5%) (P-value=0.23). All the controls have no family history of CHD, while 23(57.5%) of patients have family history and 17(42.5%) do not (p-value=0.02).

All the controls did not admitted to the NCU, while 26(65.0%) of patients have admitted and 14(35.0%) do not (p-value=0.13). Regarding the types of CHD, 22(55.0%) of cases were having VSD, 5(12.5%) ASD, 8(20.0%) TOF, and 5(12.5%) TGA (p-value=0.05).

N=80	Cases		Controls		p-value	
IN=80	Frequency	Percent	Frequency	Percent		
Sex	male	20	50.0	20	50.0	0.18
Sex	female	20	50.0	20	50.0	0.18
	mature	18	45.0	27	67.5	0.11
Maturity	premature	22	55.0	13	32.5	
Mode of Delivery	C-section*	27	67.5	18	45.5	0.23
	Vaginally	13	32.5	22	55.5	
Family History	positive	23	57.5	0.0	0.0	0.02
Family History	negative	17	42.5	40	100.0	
NCU Admission	yes	26	65.0	0.0	0.0	0.13
NCU Admission	no	14	35.0	40	100.0	
Types of Congenital Heart Disease	VSD	22	55.0	0.0	0.0	
	ASD	5	12.5	0.0	0.0	
	TOF	8	20.0	0.0	0.0	0.05
	TGA	5	12.5	0.0	0.0	

*C-section: Caesarean Section

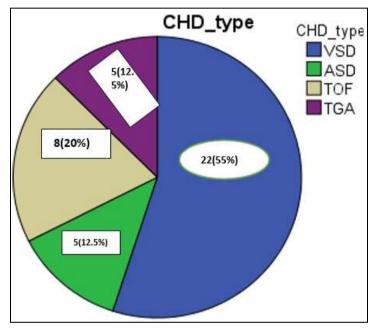


Fig 1: Distribution of CHD Types among cases.

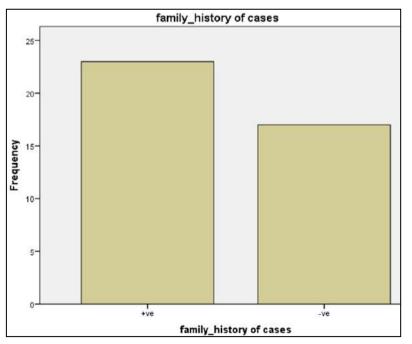


Fig 2: Family history of cases.

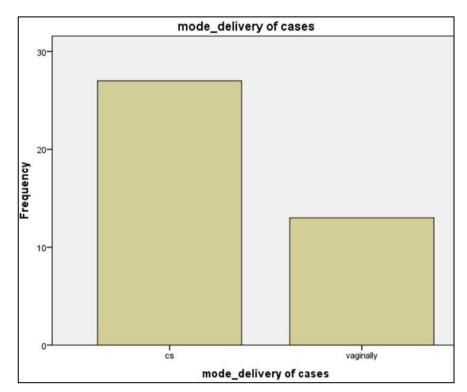


Fig 3: Classification of cases according to Mode of delivery.

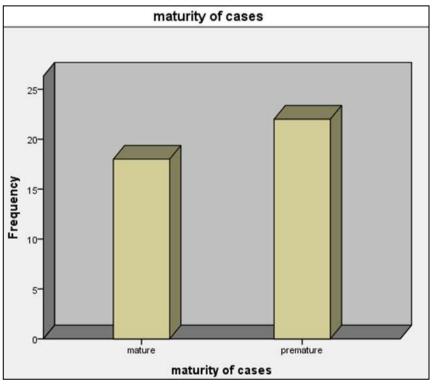




Table 3 shows means of weight, height, and HC were 8.50, 71.72 and 43.17 respectively for CHD children and were 11.30, 80.70 and 46.66 respectively for healthy ones. These

differences were significant except mean of height (p>0.05) that was similar between groups.

Table 3: The mean of the growth parameters of cases and controls.

N=80	Growth Parameters				
N=00	Weight Mean	Height Mean	Head circumference Mean		
Cases	8.5000	71.7250	43.1750		
Controls	11.3000	80.7000	46.6625		
P-value	0.001	>0.05	0.002		

Table 4 showed the frequency of different participants in different percentiles of height, weight, and HC using growth curve. Regarding height, the table showed that out of 40 of cases, 95%, 5%, and 0%, out of 40 of controls 0%, 65.0%, and 35.0% were dropped in the percentiles of $< 5^{th}$, between 5^{th} and 50^{th} and $> 50^{th}$, the p-value was < 0.0001. Regarding weight, the table showed that out of 40 of cases, 95%, 5%,

and 0%, out of 40 of controls 2.5%, 27.5%, and 70.0% were dropped in the percentiles of $< 5^{\text{th}}$, between 5^{th} and 50^{th} and $> 50^{\text{th}}$, the p-value was < 0.0001. Regarding head circumference, the table showed that out of 40 of cases, 97.5%, 2.5%, and 0%, out of 40 of controls 0%, 62.5%, and 37.5% were dropped in the percentiles of $< 5^{\text{th}}$, between 5^{th} and $> 50^{\text{th}}$, the p-value was < 0.0001.

Table 4: showed the frequency	y of different participants in	different percentiles of height,	, weight, and HC using growth curve.

	Percentiles					
Groups Of Participants	< 5 th Between 5 th and 50 th		>50 th	Total	D malma	
	Frequency (%)	Frequency (%)	Frequency (%)	Frequency (%)	P- value	
		Height				
Controls	0 (0%)	26 (65.0%)	14 (35.0%)	40 (100%)	< 0.0001	
Cases	38 (95.0%)	2 (5.0%)	0 (0.0%)	40 (100%)		
		Weight				
Controls	1 (2.5%)	11 (27.5%)	28 (70%)	40 (100%)	-0.0001	
Cases	38 (95.0%)	2 (5.0%)	0 (0.0%)	40 (100%)	< 0.0001	
		Head circumference				
Controls	0 (0.0%)	25 (62.5%)	15 (37.5%)	40 (100%)	-0.0001	
Cases	39 (97.5%)	1 (2.5%)	0 (0.0%)	40 (100%)	< 0.0001	

4. Discussion

Weight, height and head circumference were significantly lower in CHD children compared to healthy ones. Weight, Height and Head circumference were significantly lower in cvanotic and Acyanotic patients compared to groups' of CHD children. Children with CHD are malnourished and one of the most important reasons for these effects is storage reduction and increased energy consumption in their bodies ^[8]. Severe growth retardation in these patients maybe is a cause of failure to thrive even after surgery [8]. Genetic, environmental, nutritional, social, and frequent respiratory infections are determined major factors, but has not been specified which one more important [16]. In a study, pulmonary hypertension and cyanotic diseases were the most important factors of growth failure in patients with CHD ^[17]. In another survey the highest impact on growth failure has been observed in children with large ventricular septal defect and tetralogy of Fallot [18]. In Viviane Martins et al. study, growth and feeding status of CHD children less than one year old without surgery evaluated. The study results showed that the incidence of malnutrition was varied based on gender, cardiac abnormalities, height, and weight and head circumference at birth and in acute malnutrition, weight and height were most common in boys and girls ^[19]. In a retrospective cohort study by Daymont et al. on the growth status of children resulted that the most frequent disorders were weight for age and height for age in the first four months of life and continued till the age of 2 or 3 years ^[20]. In a case –control study by Hassan *et al.*, the status of feeding evaluated on cyanotic and Acyanotic children with non-operation surgery and compared with healthy ones. From the study resulted that the prevalence of underweight and short stature was higher in patients. A comparison in patients groups, they resulted that the prevalence of short stature and underweight was more in Acyanotic and cyanotic respectively. Hassan et al., also reported that the prevalence of malnutrition was 20% in controls and 84% in patients so that 71.4% had severe malnutrition. The prevalence of malnutrition in patients correlated with low levels of hemoglobin, low levels of arterial blood saturation, heart failure, and pulmonary hypertension ^[21]. Regarding the age of the mother and father, the study shows that the age of

the mother and father was higher in cases than that of controls and these results were similar to a study performed in south of Iran (In Ahwaz) that showed higher age of father and mother in cases than controls⁽²³⁾. Our study shows no sex differences (50/50) between males and females, these results were the same in Ahwaz study ^[23]. The current study shows that CHD are more common in premature patients (55.0%) than the mature ones (45.0%), these results were similar to the study (Noori et al.) [15]. Noori et al. conducted a study on CHD children aged 3 months to 36 months on some of growth parameters and resulted that a high percentage of patients were under the fifth percentile regardless of age classifications and type of CHD. Also, Noori study showed that family history was positive in 58% of cases which was similar to our study (57.5%) ^[15]. In our study, VSD was the most common type of CHD (55.0%) among cases followed by TOF the next most common and these results were similar to a survey by Chung and colleagues in Taiwan^[24]. In a survey by Chung and colleagues in Taiwan resulted that more than 50% of children with CHD, and 33% of normal children were below the 50th percentile of weight ^[24]. This difference can be attributed to differences in the factors affecting the growth of CHD patients, including gender [19], age [19, 20], cardiac abnormalities ^[18, 19, 25], simultaneous multiple congenital heart defects ^[16], and congestive heart failure ^[21]. Thus, the findings of this study, in line with other studies, a higher prevalence of growth retardation in children with CHD confirmed.

5. Conclusion

- 1. There was significant retardation in all growth parameters (weight, height and head circumference) in patients with congenital heart disease.
- 2. There was no sex predilection for occurrence of CHD and both sexes are equally affected.
- 3. Maternal and paternal age, prematurity, family history of CHD and mode of delivery are significantly associated with the occurrence of CHD.
- 4. The most common type of CHD was VSD followed by TOF.

6. Recommendations

- 1. Recommend that early surgical intervention and nutritional support can be fruitful in prevention of CHD complications.
- 2. It is very important to do antenatal screening for CHD in families with positive family history.
- 3. During pregnancy, there is crucial role of antenatal care in order to decrease the risk of prematurity and subsequently the prevalence of CHD.
- 4. There is great need for maternal programs including social maternal, T.V. and journals about the implementation and benefits of contraception at younger age to reduce the risk of CHD.

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