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Detection of Cardiac Remolding After Surgical Closure of Ventricular Septal Defect in Children Less Than One Year

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ABSTRACT

Background: Cardiac remodeling is an important issue after surgical closure of ventricular septal defect (VSD).

Objective: The purpose of the present study was to evaluate cardiac remodeling by echocardiography by measuring the ejection fraction, fractional shortening, left ventricular internal diameter in diastole (LVIDd) and left ventricular internal diameter in systole (LVIDs) after surgical closure of VSD defect in patients less than 1 year.

Methodology: This study was conducted in the department of pediatric cardiology, Maternity and Children Hospital (MCH), El Madina El Menwara, Saudi Arebia. Patients were divided into 3 groups according to the age. All patients presented with isolated perimembreanous VSD (PM-VSD) who were surgically treated at the age less than1 year. This study population was divided into 3 groups as group A from (0- 3) months, group B from (3- 6) months and group C from 6 months to 1 year. Echocardiographic variables such as ejection fraction, fractional shortening, LVIDd and LVIDs were taken pre operative and one month post operative in all groups. The echocardiography was repeated three months post operative in group B and group C.

Result: A total number of 100 patients were recruited for this study. All the above parameters were normal 1 month after VSD closure in group group A. However, in both group B and group C the follow up echocardiography was indicated. In both groups the time was needed to reach the normal vlues.

Conclusion: Cardiac remodeling is an important indicator of the ability of the cardiomyocyte to be changed to the normal shape and dimension. There are many factors that affect the cardiac cell. The presence of preload or afterload causes can result in delay of remodeling.

As long as the patient start to take medication for anti failure cause, the suspicion of remodeling will be delayed.

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Introduction

Ventricular septal defect (VSD) is one of the most frequent congenital cardiac abnormalities [1]. Among infants dying with congenital heart disease in the first month of life, ventricular septal defect is second to transposition of the great arteries as a cause of death in this age group [2]. Prevalence rate of ventricular septal defects (VSDs) is varied from 0.44 and 0.48 per 1000 in four successive years of study in elementary school children [3]. The traditional treatment of VSD is surgical repair [4]. Since 1954, when the first surgical closure of a PM- VSD took place, there have been considerable changes in the surgical strategy for closure in terms of timing, perfusion modalities and approach, making surgical closure a relatively low risk procedure [1]. In the last decade, percutaneous closure techniques have been developed for all types of ventricular septal defects [5]. Though the surgical approach is considered to be the gold standard; however, it is associated with morbidity and mortality, patient discomfort, sternotomy and skin scar [6-7]. Improvement in cardiac function has been monitored by observing the cardiac remodeling. Cardiac remodeling is the genome expression, molecular, cellular and interstitial changes which are manifested clinically as changes in size, shape and function of the heart after cardiac injury [8]. The process of cardiac remodeling is influenced by hemodynamic load, neuro-hormonal activation and other factors still under investigations. Our theory is medication as loop diuretic can affect the function of the cardiac myocyte. We name our theory as "Taiba theory". We think about this theory as long as the patients take medication more time is taken by the myocyte to return to normal function and dimension. Loop diuretic cause hyponatremia, hypokalemia and hypochoremia. This ionic imbalance can affect the cardiac myocyte. The mitochondria can be affected resulting in imbalance in cell function and dimension. Further research on the cell biology and laboratory tests are still under investigation. Therefore, many questions will be answered.

The myocyte is the major cardiac cell involved in the remodeling process [8]. The degree of compensatory changes in the heart depends on the size and flow across the VSD, the relative compliance, pressure and resistance to flow in the ventricles and the greater the duration of this left to right shunt, greater the adaptive changes.

Therefore, early intervention can prevent the pathological changes and the magnitude of cardiac remodeling. Thus it is important to focus on echocardiographic evaluation of cardiac remodeling after surgical closure of VSD to evaluate the beneficial effect of early closure. The present study was undertaken to evaluate cardiac remodeling by echocardiography by measuring the ejection fraction, fractional shortening, LVIDd and LVIDs after surgical closure of ventricular septal defect.

Methodology

This study was designed as prospective cohort study and was carried out from December 2012 to May 2017. This study was conducted in the Department of pediatric cardiology, Maternaty and children hospital (MCH), El Madina El Menwara. All patients presented with isolated PM- VSD who were surgically treated at the age below 1 year. Both patients and echocardiographic parameters are collected in (table 1).

Table 1: Shows the clinical and echocardiographic parameters for this study

• **Clinical parameters:**
Age <1 years.
Male: female, 54: 46
Heart failure grade 2 to grade 4 according to NHA.
Asyndromic.
History of sibiling with history of open heart surgery.
Medication monotherapy or multitherapy
• **Echocardiographic parameters:**
VSD size: large > 5 mm, moderate size 3-5 mm.
No pulmonary hypertension.
Patent ductus arteriosus.
Ejection fraction.
Fraction shortening.
Left ventricular internal diameter in systole.
Left ventricular internal diameter in diastole.

The patients were divided into 3 groups. Group A from 0-3 months, group B from 3-6 months and group C from 6 months to 1 year. The size of PM-VSD were measured and summarized in (table2).

Table 2: Summarizes the size of VSD, number of patients start medication and if there is PDA or not

Group	F:M	SIZE VSD	Associated with PDA	Patient Start Drugs
0	20	> 5 mm	12:14	A
30	10	4-5 mm	24:26	B
15	NO	3-4 mm	10:14	C
45	30		46:54	TOTAL

F number of females, **M** number of males,
VSD ventricular septal defects.
PDA patent ductus arteriosus.

The association of patent ductus arteriosus PDA is also evaluated. The association of PDA make more effort on the heart. After getting written informed consent from parents of the children, all data regarding demographic variables, echocardiographic variables were collected. M-mode and two-dimensional echocardiography recordings were performed. Echocardiography was done in The supine position using standard parasternal short- and long-axis and apical views.The left ventricle ejection indices like ejection fraction, fractional shortening, Left ventricular internal diameter during diastole (LVIDd) and systole (LVIDs) were recorded by echocardiography.

All patients were followed at 1 month and 6 months postoperative by echocardiography and compared with preoperative echocardiographic findings. Data were collected using a preformed data collection sheet. Baseline information was collected from the patient after exploration of different complaints and sign and symptoms of the cardiovascular diseases. Statistical analysis was performed by using window based computer software with Statistical Packages for Social Sciences (SPSS-15) (SPSS Inc, Chicago, IL, USA).

Results

A total number of 100 patients underwent PM- VSD closure. Patients were divided into three groups. Group A from 0 - 3 months, group B from 3- 6 months and group C from 6 months to 1 year. Ejection fraction reading were collected in (table 3).

Table 3: Ejection fraction for patients

	Postoperative		
Group	Preoperative	1m	3m
Group A	63.00±3.22	65.00±2.22	Not indicated
Group B	62.11±2.52	59.12±2.14	66.51±2.10
Group C	64.28±2.31	61.22±2.51	67.61± 4.21

In group A the mean (±SD) ejection fraction of heart at the time of preoperative and postoperatively after 1 month were 63.00 (±3.22) and 65.00(±2.22) respectively. In group B the mean (±SD) ejection fraction of heart at the time of preoperative and postoperatively after 1 month and 3 months were 62.11 (± 2.52), 59.12 (±2.14) and 66.51 (±2.10) respectively. In group C the mean (±SD) ejection fraction of heart at the time of preoperative and postoperatively after 1 month and 3 months were 64.28 (±3.31), 61.22 (± 2.51) and 67.61 (±4.21) respectively. The fractional shortening reading were collected in (table 4).

Table 4: Fraction shortening for patients

	Postoperative		
Group	Preoperative	1m	3m
Group A	30.12±2.12	32.00±3.11	Not indicated
Group B	31.51±3.21	28.21±3.11	32.00±4.71
Group C	31.67±2.12	27.22±5.11	33.21±5.11

In group A the mean (±SD) fractional shortening of heart at the time of preoperative and postoperatively after 1 month were 30.12 (±2.12) and 32.00 (±3.11) respectively. In group B the mean (±SD) fractional shortening of heart at the time of preoperative and postoperatively after 1 month and 3 month were 31.51(±3.21), 28.21 (±3.11)% and 32.00 (±4.71) respectively. In group C the mean (±SD) fractional shortening of heart at the time of preoperative and postoperatively after 1 month and 3 month

were 31.67 (±2.12), 27.22 (±5.11)% and 33.21(±5.11) respectively. The left ventricular internal diameter during systole reading was collected in (table 5).

Table 5: Left ventricle internal dimension during systole

	Postoperative		
Group	Preoperative	1 m	3m
Group A	25.40±3.12	23.50±2.14	not indicated
Group B	26.21±3.14	26.83±4.12	24.21±4.11
Group C	27.00±2.15	28.10±2.11	25.20±2.11

In group A the mean (±SD) left ventricular internal diameter during systole (LVIDs) of heart at the time of preoperative and postoperatively after 1 month were 25.40 (±3.12) mm and 23.50 (±2.14) mm respectively. In group B the mean (±SD) left ventricular internal diameter during systole (LVIDs) of heart at the time of preoperative and postoperatively after 1 month and 3 month were 26.12 (±3.14) mm, 26.83 (±4.12) mm and 24.21 (±4.11) mm respectively. In group C the mean (±SD) left ventricular internal diameter during systole (LVIDs) of heart at the time of preoperative and postoperatively after 1 month and 3 month were 27.00 (±2.15) mm, 28.10 (±2.11) mm and 25.2 (±2.11) mm respectively. The left ventricular internal diameter during diastole.

Table 6: Left ventricle internal dimension during diastole

	Postoperative		
Group	Preoperative	1 m	3m
Group A	36.62±4.16	36.21±2.61	not indicated
Group B	38.72±4.31	39.00±2.45	35.00±6.24
Group C	35.21±2.14	34.00±2.42	33.00±2.51

In group A the mean (±SD) left ventricular internal diameter during diastole (LVIDd) of heart at the time of preoperative and postoperatively after 1 month and were 36.62 (±4.16) mm and 33.21 (±2.61) mm respectively. In group B the mean (±SD) left ventricular internal diameter during diastole (LVIDd) of heart at the time of preoperative and postoperatively after 1 month and 3 month were 38.72 (±4.31) mm, 39.00 (±2.45) mm and 35.00 (±6.24) mm respectively. In group C the mean (±SD) left ventricular internal diameter during diastole (LVIDd) of heart at the time of preoperative and postoperatively after 1 month and 3 month were 35.21 (±2.14) mm, 34.00 (±2.42) mm and 33.00 (±2.51) mm respectively.

Disscusion

Ventricular septal defect (VSD) is a common congenital anomaly which requires early closure by various methods. In this study100 patients of different age group who had undergone ventricular septal defect closure by surgery were enrolled. Echocardiographic evaluation was done before operation, 1 month and 6 months after surgical closure of VSD. Cardiac remodeling was measured by the changes of the ejection fraction, fractional shortening, left ventricular internal diameter during diastole (LVIDd) and left ventricular internal diameter during systole (LVIDs) after surgical closure of ventricular septal defect. All patients presented with isolated PM- VSD who was surgically treated. The size of VSD is different in each patient group as seen in (table 2). This study population was divided into 3 groups as group A from 0- 3 months, group B from 3- 6 months and group C from 6 months to 1 year. In group A no medications were prescribed. In group B and C loop diuretic was started as shown in (table 2). For group A good remodling happen in short time that support our theory that named Taiba theory. This theory suggests that hypochoremia and hypokalemia affect the myocyte. This ionic imbalance can affect the cardiac myocyte. The mitochondria can be affected resulting in imbalance in cell function and dimension. Further research on the cell biology and laboratory tests are still under investigation. Therefore, many questions will be answered. Perimembranous defects are common type of VSD [9]. According to their location within the septum, defects can be classified as muscular, perimembranous and supracristal [10]. This PM-VSD is the most common hemodynamically significant VSD [1]. After I month of operation the ejection fraction was increasing by 3.17% in group A patients. These patients have no conceren.

Ejection fraction was decreased from preoperative to 1 month of postoperative values in B and C groups. After 1 month it was decreased by 4.8 % and 4.7 % in group B and group C respectively. Ejection fraction increased significantly from preoperative values to the third month of postoperative values of surgical closure of VSD. After 3 months ejection fraction was increased by 6.42 % and 5.18 % in group B and group C respectively. Fractional shortening was decreased from preoperative to 1st month of postoperative values in group B and group C. However, in group A there is 6.24% increase in the fractional shortening after 1month. The fractional shortening was decreased by 10-47%and 14.05%in group B and group C respectively. Fraction shortening significantly increased after 3rd month of postoperative values from preoperative values in each group and the increment was 1.5 % and 4.86% in group B and group C respectively.

LVIDs increased from preoperative to first month of postoperative values in group B and C. However, in group A the LVIDs decreased by 7.4%. After 1 month LVIDs was increased by 2.7% and 3.07% in group B and group C respectively. LVIDs was declined from preoperative values to 3rd month of postoperative values ($p<0.05$). After 3 months LVIDs was decreased by 4.07% and 6.66% in group B and group C respectively. This study was consistent with other study, where LVIDs were reduced in 1 year after closure of VSD [12]. LVIDd increased from preoperative to the first month of postoperative values in group B and C but reduced in group A by 9.31%. After 1 month LVIDs were increased by 2.8% and7.92% in group B and group C respectively. LVIDd was significantly declined from preoperative values to 3rd month of postoperative values ($p<0.05$). After 3 months LVIDd were decreased by 9.6 %and 6.27 % in group B and group C respectively. This study was consistent with the study of Yuan et al, where LVIDd was reduced 1 year after closure of VSD [12]. Remodeling was better in young age group then elder [13]. From this study it is evident that favorable left ventricular morphological changes occur when early surgical closure of VSD is done in relatively younger group of patients.

Conclusion

This studies permits to conclude that cardiac remodeling is better among the younger children with no medication than older children with medication. The ejection fraction, fractional shortening, left ventricular internal diameter during diastole (LVIDd) and left ventricular internal diameter during systole (LVIDs) is significantly improving after three months of surgery [14,15].

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