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Ventricular septal defect in children and adults by echocardiography study in Iraqi patients

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Abstract

Objective: A ventricular septal defect (VSD) is defined as a communication between the left and right ventricles or between the left ventricle and the right atrium. VSDs are amongst the most common abnormalities of the heart. They can be present in isolation or in association with other congenital cardiac abnormalities. This is study done with the aim to evaluate the types, size, associated CHD with ventricular septal defect in children and adolescent in two cardiac centers (Medical City Complex cardiac clinics, Ibn Al Nafaes teaching hospital) in Baghdad - Iraq

Methods: Prospective cross sectional study based on echocardiography done pediatric cardiology centers and included all children and adult (birth to 25 years old) with VSD during period from 1st of October 2021 till 1st of July 2022. The examination performed in the lateral decubitus position using the echocardiography device (GE VIVID 9), transducer M6Sc-D and M5Sc-D. All patients were examined standard transthoracic echocardiography (TTE) including two dimensional, colors Doppler and M -mode. The standard technique was used to obtain the measurements. Size and type of VSD were examined in different standards four chambers, five chambers, parasternal long and short axis and subcostal views. The sample was divided into two groups, group 1 isolated VSD; and Group 2 associated with other CHD. The data collected for patients with ventricular septal defect and the age (from birth to 25 years old).A convenient sample of 206 patients with VSD was selected from consultancy clinics and wards of selected hospitals.

Results: A total of 206 patients with ventricular septal defect were included in this study; 105 patients (51%) from Medical city complex, 101 patients (49%) from Ibn Al Nafeas teaching hospital. Males were 49% while females were 51%. The prevalence of isolated VSD 99(48%) and VSD associated with other CHD 107(52%). Symptomatic VSD were 60%, while Asymptomatic VSD diagnosed incidentally were 40%. The most common type of VSD was Perimembranous 73 followed by muscular 19%, Inlet (canal type) 7% and Outlet (subarterial) Subpulmonic VSD 1% respect. The most common associated CHD was PDA 33 (30.8%), followed by ASD Secundum 30 (28%), PS 10 (9.3%), Coarctation of aorta 8 (7.4%), MR 7(6.5%), TOF 6 (5.6%), Atrioventricular canal defect 3 (2.8%), DORV 2 (1.8%) and each of L-TGA, DTGA, Dextrocardia , truncus arteriosus and tricuspid atrasia had 1 (1%).

Conclusions: The most common VSD type of studied patients is perimembranous in followed by muscular, inlet type and the less common is outlet. Although multifactorial, Consanguinity marriage with positive family history plays a great role of recurrent CHD in families.

Keywords: Echocardiography; Ventricular septal defect; Children; Iraq

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1. Introduction

A ventricular septal defect (VSD) is defined as a communication between the left and right ventricles or between the left ventricle and the right atrium. VSDs are amongst the most common abnormalities of the heart. They can be present in isolation or in association with other congenital cardiac abnormalities. (1) The incidence ranges from 5 to 50 per 1,000 live births and 0.3 per 1,000 adults. (2)

The etiology of VSDs is likely multifactorial with both genetic and environmental factors. VSDs are

common in aneuploidy syndromes such as trisomy 13, 18, and 21 and in other genetic syndromes such as Holt-Oram (TBX5 mutation) (3). VSD in offspring has been associated with paternal use of marijuana and cocaine as well as paternal exposure to paint strping.(4’5).

Central perimembranous VSDs are located at the base of the heart, usually adjacent to the membranous ventricular septum. One margin of the defect almost always involves the area of fibrous continuity between the tricuspid and aortic valves. (6) Inlet VSDs are those defects opening into the inlet component of the right ventricle and extending along length of the tricuspid valve.(7, 8) Muscular trabecular defects are located within the more apically located segments of the muscular ventricular septum. They are further classified based on their location along the right ventricular surface of the ventricular septum with the most common descriptors including the midseptal ,apical, anterior, and inferior (posterior) areas of the muscular trabecular septum. (8)Outlet VSDs open into the outlet component of the right ventricle just under the pulmonary valve in hearts with ventriculoarterial concordance and almost never close spontaneously(1).

The physiology of a VSD is determined primarily by the direction and amount of flow across the defect. These variables are determined by the size of the defect and the relative resistances in the pulmonary and systemic vascular beds.(Therefore, the spectrum of pathophysiology ranges from large VSDs with significant left-to-right shunting across the defect resulting in left atrial and ventricular dilation to small VSDs with pressure-restrictive left-to-right flow to VSDs with acquired elevated pulmonary vascular resistance resulting in right-to-left shunting across the defect. (9)

The clinical presentation of the isolated VSD is primarily dependent on the size of the defect. Large defects often go unrecognized in the newborn period because they do not cause a significant heart murmur and the infant appears clinically well. Smaller defects that restrict flow from the left to the right ventricle typically cause loud heart murmurs and thus are often recognized early in infancy. (10)A large VSD can be primarily defined in several ways: when the defect is as large as the (normal) aortic valve and/or when the defect size equilibrates the pressure between the left and right ventricles.The typical presentation of a large VSD occurs by 4 to 8 weeks of age. As a result of the drop in pulmonary vascular resistance over the first few weeks of life, the infant with a large VSD will develop symptoms of congestive heart failure. (11)A heart murmur is a consistent component of the physical examination. Typically, an SI coincident holosystolic murmur is appreciated along the left sternal border and becomes decrescendo during the latter part of systole before closure of the aortic valve. The P2 component of the second heart sound is typically loud, and splitting is narrow (or S2 sounds single) as a result of the systemic pulmonary artery pressure. (12) (13)A small VSD is defined as a defect that is generally less than one-third the size of the normal aortic valve. The typical presentation of a small VSD is quite benign. Infants and children with small defects are essentially asymptomatic because the left-to-right shunt is minimal. An SI coincident holosystolic murmur that is typically grade 4 to 5/6 in degree and plateau in nature is heard at the left lower, mid, or upper sternal border depending on the location and jet direction of the VSD. The second heart sound is typically normal because the pulmonary artery pressure is not elevated . (13)A moderate VSD usually provides some resistance to pressure but variable resistance to flow. The right ventricular pressure is subsystemic but usually higher than normal. These defects will either behave like large VSDs and cause significant left-to-right shunting or become smaller over time and develop a higher gradient across the defect from the left ventricle to right ventricle.Patients with moderate-sized VSDs will usually present with milder presentation of congestive heart failure. The tachypnea and tachycardia may be subtle, and growth may be less affected than in those with large VSDs. The physical examination will be similar to those with large VSDs, except the holosystolic murmur will be louder because of the flow restriction across the defect. (13)

The electrocardiogram for a patient with a VSD depends on the size of the defoct and the age of the patient. The infant with a large VSD will typically exhibit right ventricular hypertrophy (upright T wae in VI, increased R voltage in VI, and/or increased S voltage in V6 or V7) or biventricular hypertrophy (increased biphasic voltages over the midprecordium (fig 6). In the infant who has a long-standing left-to right shunt, left atrial enlargement is typically present. Children with small VSDs usually have a normal electrocardiogram without evidence for ventricular

hypertrophy or atrial dilation. Those with elevated pulmonary vascular resistance will have evidence for right ventricular hypertrophy, sometimes with a strain pattern. (10)

The chest x-ray of an infant with a VSD is usually normal early in life, regardless of the size of the VSD. As pulmonary vascular resistance drops, the children with a large VSD exhibits increased pulmonary vascular markings and a prominent main pulmonary artery segment. Left ventricular volume overload will result in cardiomegaly. (10)Echocardiography can delineate the abnormal anatomy and hemodynamics of a VSD. Anatomic evaluation must include identification of the defect type as well as its location using the right ventricular septal surface as reference. Echocardiography is the primary diagnostic tool to evaluate a VSD and is frequently the only imaging modality required to determine the location and size of the VSD as well as the need for intervention. Cardiac catheterization to assess VSD size is rarely required in the present day. Serial echocardiographic studies can be performed on children with a VSD to determine progression or resolution of disease and whether associated abnormalities (such as a double-chambered right ventricle or aortic valve prolapse) are developing. (1)

For large VSDs, medical management is used to treat the congestive heart failure symptoms once they become evident. Medication does not alter the size or the natural history of the VSD. Furosemide or other loop diuretics are used to treat the pulmonary congestion that occurs with a large left-to right shunt. Systemic afterload reduction may also be used in the setting of a high Qp/Qs ratio, typically with angiotensin-converting enzyme (ACE) inhibition. (14)

1.1. Patients and methods

This study was a prospective cross sectional study conducted in Baghdad Medical City Complex cardiac clinics , Ibn Al Nafaes teaching hospital a in Baghdad - Iraq during period from 1st of October 2021 till 1st of July 2022.

All children and adult with ventricular septal defect (VSD) presented or admitted to consultancy clinics and wards of selected hospitals and clinics were the study population.

The examination performed in the lateral decubitus position. using the echocardiography device (GE VIVID 9),transducer M6Sc-D and M5Sc- D.

All patients were examined standard transthoracic echocardiography (TTE) including two dimensional, color Doppler and M -mode.

The standard technique was used to obtain the measurements. Size and type of VSD were examined in different standards four chambers, five chambers, parasternal long and short axis and subcostal views.

The size of VSD according to normal aorta of patient and pressure gradient(PG) across VSD were small which mean less than one-third aorta and pressure gradient (more than 70mmHg),medium between one- third to two third with pressure gradient (between 20_70 mmHg) and large more than two-third of aorta with pressure gradient (less than 20mmHg).

The patients also evaluate with clinical suspicion of pulmonary hypertension by Doppler echocardiography study, peak of TR velocity in continuous-wave was used to estimate the systolic pressure in the pulmonary artery plus added the right atrial pressure in the absence of PS.

Also was estimate by end-diastolic velocity of PR plus added the right atrial pressure.

The sample was divided into two groups, group 1 isolated VSD; and Group 2 associated with other CHD including PDA,ASDII and ASD primum,PS,COA,MR, TOF,DORV, AVCD, D-TGA and L-TGA, TRUNCUS ARTERIOSUS, TRICASPID ATRASIA and dextrocardia.

The data collected for patients with ventricular septal defect and the age (from birth to 25 years old).

A convenient sample of 206 patients with VSD was selected from consultancy clinics and wards of selected hospitals and clinics.

1.2. Data Collection

The data was collected directly from patients or from their parents. The questionnaire was designed by the researcher. The following information was checked in every patient:

- Demographic characteristics of VSD patients: Age and gender.
- Source of data collection of VSD patients.
- Age of mother and father.
- Maternal Hx. of Hypertention or Diabetes mellitus, drugs intake.
- History of IVF or normal pregnancy.
- Paternal use of marijuana and cocaine , exposure to paint stripping.
- If the diagnosis of VSD based on symptoms or discovered by routine checkup (incidentally).
- VSD characteristics: types, size in relation to native aorta and pressure gradient across the VSD.
- If the VSD isolated or associated with other CHD.
- If the VSD associated with complication like prolapsed aortic coronary cusp.
- Family history of CHD,consanguinity , the sequence of the child in the family.
- If child have genetic syndrome.

1.3. Statistical analysis

All patients' data entered using computerized statistical software; Statistical Package for Social Sciences (SPSS) version 22 was used. Descriptive statistics presented as (mean ± standard deviation) and frequencies as percentages. Multiple contingency tables conducted and appropriate statistical tests performed, Chi square test was used for categorical variables (Fishers exact test was used if the total of expected variables was less than 25% of total variables. In all statistical analysis, level of significance (p value) set at < 0.05.

2. Results

A total of two hundred and six patients with ventricular septal defect were included in this study; 105 patients (51%) from Medical city complex, 101 patients (49%) from Ibn Al Nafeas hospital. These findings were shown in table 1.

Table 1 The number of patients which had examined in each center

Name of center	Number of patients	Percent %
Medical city complex	105	51%
Ibn Al Nafaes hospital	101	49%
Total	206	100%

Table 2 The age and gander at which diagnosis of VSD confirmed by echocardiography

Variable	Number	Percent%
Age at diagnosis		
Less than 6 months	117	56.7%
From 6 to 12 months	36	17.4%
From 1 to 2 years	20	9.7%
Above 2 to 25 years	33	16.2%
Total	206	100%
Gender		
Male	100	49%
Female	106	51%
Total	206	100%

The age at diagnosis shows 117 patients (56.7%) were less than 6 month, 36 patients (17.4%) were from 6 to 12 months, 20 patients (9.7%) were from 1 to 2 years, 33 patients (16%) were above 2 years. The gender distribution of

patients in our study were in male 100 patients (49%) and 106 female patients (51%). All these findings were shown in table 2.

Regarding maternal drug history ,the mothers who not taken any drugs during pregnancy in patients with VSD were 185 patient (89.8%) while the mothers were taken antihypertensive drugs in 16 patients (7.8%) and insulin for diabetes were 5 patients (2.4%) .The drugs history in our study was statistically not significant (P=0.4)

Table 3 Maternal drug history during pregnancy

drugs intake or not	Number	Percent %	P value
Not taking any drugs	185	89.8%	0.4s
Antihypertensive drugs for hypertension	16	7.8%	
Insulin for DM	5	2.4%	
Total	206	100%	

* Fishers exact test, NS=Not significant

Regarding risk factors association, maternal hypertensive positive in 15 patients (7.3%) (p=0.5), diabetic positive in 5 patients (2.4%) (p=0.5), history of IVF in 9 patient (4.4%) (p=0.2) which statistically not significant. Paternal exposure to paint was positive in 5 patients (2.4%) (p = 0.5) which also statistically not significant.

There is spastically not significant in patients with VSD and paternal age (p=0.15) and maternal age (p=0.5)

Table 4 The risk factors (hypertension, DM, IVF, exposure to paints, paternal age, and maternal age) in patients with VSD in our study

Risk factors	Positive (%)	Negative (%)	Total	P value
Hypertension	15 (7.3%)	191(92.7%)	206	0.5'NS
DM	5 (2.4%)	201(97.6%)	206	0.5'NS
IVF	9 (4.4%)	197(95.6%)	206	
Paternal Exposure to paint	5 (2.4%)	201 (97.6%)	206	0.6'NS
Paternal age	>40 years	<40 years	206	0.045^
	58(28.2%)	148 (71.8%)		
Maternal age	>35 years	<35 years	206	
	31 (15%)	175 (85%)		

*Fishers exact test, NS=Not significant

Table 5 The relationship between VSD and family history of VSD in other member in the family and the consanguinity

Risk factors	Positive (%)	Negative (%)	Total	P value
family history for VSD	32(15.5%)	174(84.5%)	206	0.05*s
consanguinity	125(60.7%)	81 (39.3%)	206	0.003*s

* Fishers exact test, NS=Not significant, S=Significant.

There was significant association between positive family history of VSD patients (P = 0.05) as well as There was significant association among positive consanguinity (P= 0.003) as shown in table 5.

Causes of first echocardiography in our study were commonly symptomatic VSD in 124 patients (60%) most cases referred as chest infection symptoms of failure to thrive and heart failure and was significant (p=0.004), followed by incidentally diagnosis in 82 patients (40%).

All these finding were shown in(table 6) and (figure 1).

Table 6 Causes of first echocardiography

Causes of first echocardiography	Number	Percent %	P value
Symptomatic VSD	124	60%	0.004s
Asymptomatic VSD diagnosed incidentally	82	40%	

*Fishers exact test statistically, s= significant.

Table 7 Genetic syndromes in patients with VSD in our study

Variable	Number	%
VSD without Genetic syndrome	191	92.7%
VSD with Genetic syndromes		
Down syndrome (Trisomy 21)	10	4.8%
Turner syndrome	2	1%
Crouzon syndrome	2	1%
DiGeorge syndrome	1	0.5%
Total	206	100%

The VSD types of studied patients were commonly perimembranous in 150 patient (72.8%), followed by; muscular in 39 patients (19%),inlet type in 15 patients (7.2%) and the less common is outlet in 2 patients (1%). All these finding were included in table 8.

Table 8 The types of VSD in our study

Type of VSD	Number	%
Perimembranous VSD	150	73%
Muscular VSD	39	19%
Inlet (canal type),endocardial defect	15	7%
Outlet (subarterial) Subpulmonic VSD	2	1%
Total	206	100%

The VSD size of studied patients according to normal aorta size was distributed into small in 110 patients (53.4%),medium in 57 patients (27.7%),large in 39 (18.9%). All these finding were including in table 9.

Table 9 The size of VSD

Size of VSD	Anatomical size	No.	%
Less than 1/3 aorta	Small	110	53.4%
Between 1/3 to 2/3 aorta	Medium	57	27.7%
More than 2/3 aorta	Large	39	18.9%
Total		206	100%

Table 10 Pressure gradient (PG) across the VSD

Pressure gradient	Hemodynamic size	Number	%
Above 70 mmHg	Small	94	45.6%
Between 20 to 70 mmHg	Medium	48	23.3%
Less than 20 mmHg	Large	64	31.1%
Total		206	100%

Table 11 VSD associated with other congenital heart anomalies in patient with ventricular septal defect

Associated anomaly	Number	%
PDA	33	30.8%
ASDSECUNDUM	30	28%
PS	10	9.3%
COARCTATION OF AORTA	8	7.4%
MR	7	6.5%
TOF	6	5.6%
ATRIOVENTRICULAR CANAL DEFECT	3	2.8%
ASD PRIMUM	3	2.8%
DORV	2	1.8%
L-TGA	1	1%
DTGA	1	1%
DEXTROCARDIA	1	1%
TRUNCUS ARTERIOSUS	1	1%
TRICUSPID ATRASIA	1	1%
TOTAL	107	100%

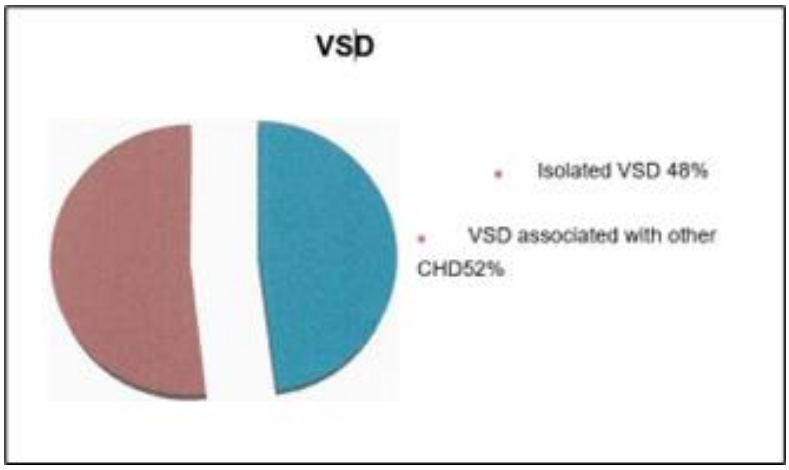

Figure 1 The prevalence of isolated VSD 99(48%) and VSD associated with other CHD 107(52%)

Table 12 VSD complications according to types and size of VSD

variables	PHT		AR		Prolapsed aortic coronary Cusp (RCC)		Subaortic ridge		Infective endocarditis	
	No.	%	NO.	%	No.	%	No.	%	No.	%
Type of VSD										
Perimembranous	8	4%	7	3.5%	7	3.5%	3	1.35%	1	0.45%
Muscular	1	0.45%			0		0	-	0	-
Inlet	2	0.9%			0	-	0	-	0	-
outlet	1	0.45%	1	0.45%	1	0.45%	0	-	0	-
Total	12	5.8%	8	3.9%	8	3.9%	3	1.35%	1	0.45%
Size of VSD										
small	0		0		0		0		0	
medium	3	1.5%	3	1.5%	3	1.5%	2	0.9%	0	
Large	9	4.3%	5	2.4%	5	2.4%	1	0.45 %	1	0.45%
Total	*12*	5.8%	8	3.9%	8	3.9%	3	1.35 %	1	0.45%

3. Discussion

Our study was prospective study where 206 patients enrolled in our study from Ibn Al-Nafees Teaching Hospital and Medical city complex in Baghdad city, to know types of VSD , associated CHD,whether the VSD symptomatic or not, if associated with complication and other echocardiographic features of VSD and the presumed risk factors for VSD for example the drug intake , consanguinity and family history.

VSD is the most common CHD worldwide and had many types and many variable associations with other CHD.[15][16]

In our study we divided VSD patients into two groups either isolated VSD which 99(48%) or VSD associated with other CHD which represented107 (52%), this is comparable to the same study of Humberto Morais b et al in Angola where the isolated VSD was (49%) and VSD with other CHD was (51%).[15][17]

In our study most VSDs were diagnosed at age group(less than 6 months) where 117 patients (56.7%), followed by age group (between 6 months and 12 months) were 36 (17.4%) and the 3rd age group was (above 2 years to 25years) 33 (16%) , while in Humberto Morais b et al study[15][17] showed, the mean age at diagnosis was (29 months) this variable age of first diagnosis is directly proportional to a worse prognosis, also inconsistency might be due to differences in health facilities and availability in specialist cardiac centers and cardiology department in hospitals between different countries.

The gender distribution of patients in our study were in male 100 patients (49%) and 106 female patients (51%) in comparing our results to other studies which showed there was no significant gender predominance like in Humberto Morais b et al study[17]

The symptomatic VSD in our study was 124 (60%) while asymptomatic VSD was 82 (40%) this indicates the severity of VSD, where the symptomatic VSD usually had more complications rate and worse prognosis, this is comparable to results of same studies in other countries like in Humberto Morais b et al study, Ejim EC et al study and Miyague et al study [17][18][19]. In the first year of life that the clinical manifestations of heart disease with left-to-right shunting appear, as inVSD, due to decreased pulmonary vascular resistance after birth and increased systemic resistance and right ventricular compliance, which causes the left-to-right shunt, leading to pulmonary hypertension and delay in development. [17]

The most common type of VSD in our study was Perimembranous VSD 150 (73%) followed by Muscular VSD 39 (19%), Inlet (canal type) endocardial defect 15(7%) and Outlet (subarterial) Subpulmonic VSD 2 (1%), these results are comparable with the results of other studies[15][16][17] where the Perimembranous VSD is the most common also these findings are close to results of Dawood study in Iraq which found that VSD types were perimembranous 80%, muscular (4%), outlet (6%) and inlet (10%), but differs from those in a study at Cathay General Hospital (Taiwan)[20] in which the authors found that 65% of VSDs were muscular, this may be a Southeast Asian characteristic.

Our study showed the most common VSD size was small in 110 patient (53.4%), followed by medium size (27.7%) and the large size VSD was (18.9%). These findings are similar to result in Iraqi study done in Ibn Al-Bitar cardiac center 2006.[21]

Regarding the family history in our study we found that 32 patients (15.5%) had positive family history of VSD, These findings are going with results of Hu et al study in China[22] which reported The familial recurrence rate of CHD among cases was approximately 16.3%.This indicate that genetics play critical role in the pathogenesis even the VSD is multifactorial in origin.

Regarding the positive consanguinity was found inl25 (60.7%) of VSD cases with p value (0.003), which is statistically Significant according to Fishers exact test, in comparing our results with Al-Fahham and Ali[23] study in Egypt which revealed that (44.6%) of patients with congenital heart diseases had positive consanguinity, also in other study from our country such as Ammar Hasan Abdul-Qahar et al [24] and Al- Ani studies [25] found that consanguinity is a significant risk factor in 58.5% and 43.3% respectively for congenital heart diseases. So in our Iraqi society consanguinity marriage and genetic factors had significant role as a cause of CHD and other genetic syndromes.

In our study the prevalence of isolated VSD 99(48%) and VSD associated with other CHD 107(52%). The most common associated CHD was PDA 33 (30.8%), followed by ASD Secundum 30 (28%), PS 10 (9.3%), Coarctation of aorta 8 (7.4%), MR 7(6.5%), TOF 6 (5.6%), Atrioventricular canal defect 3 (2.8%), DORV 2 (1.8%) and each of L- TGA, DTGA, Dextrocardia, truncus arteriosus and Tricuspid atrasia had 1 (1%). In comparing our results with Angola study where the most common associated CHD was PDA 50 (35%) followed by PS 24 (16.8%) and ASD SECUNDUM 12 (8.4%)[17].

Regarding genetic syndromes, patients with VSD in our study we found Down syndrome (Trisomy 21) in 10 (4.8%), Turner syndrome 2(1%), Crouzon syndrome 2 (1%) and DiGeorge syndrome 1 (0.05%), in comparing our results to Humberto Morais b et al study were the Down syndrome (Trisomy 21) represented 23(8%) of all cases of the study, while the Noonan syndrome represented 1 (0.4%)[17], this indicated the importance of echocardiography study for each case of genetic syndromes to exclude CHD.

Regarding the complications of VSD, the pulmonary hypertension is the commonest associated complication in 12(5.8%) VSD patients, followed by prolapsed aortic coronary cusp and aortic regurgitation in 8(3.9%), subaortic ridge in 3 (1.3%) patients and infective endocarditis in 1(0.45%), this goes with Tauseef Asma Chaudhry et al in Multan city in Pakistan were shows (22.5%) associated with PHT.[26][27]

Our study also found that the large VSD associated with sever pulmonary hypertension which progress to eisenmenger syndrome, indicate delay in proper surgical management. This finding is similar to result in Iraqi study done in Ibn Al-Bitar cardiac center 2006.[21]

Regarding paternal exposure to paints we found it not significantly associated with VSD 5 (2.4%) with P-value (0.6), this goes with the study of Vince Fazekas-Pongor et al which concluded that Even though parental occupational exposure to chemicals like solvents, polychlorinated organic substances and Phthalates. Phthalates that frequently occur in paints, plastics and cosmetics seems to have a minor impact on the occurrence of CHDs, the results of biological and environmental monitoring should be taken into consideration as well.[28]

Regarding to the drugs intake, maternal hypertension, DM, maternal age , paternal age and sequence of affected patients with VSD, all were assessed and statistically measures applied and the result showed no significant for VSD patients. These findings are comparable the result of Ammar Hasan Abdul-Qahar et al [24] which had been carried out in Ibn Al-Nafees Hospital at 2015.

4. Conclusions

- The most common VSD type of studied patients is perimembranous in followed by muscular,inlet type and the less common is outlet.
- Although multifactorial, Consanguinity marriage with positive family history play a great role of recurrent CHD in families.
- In our study VSD patients diagnosis most commonly below one year old because availability of pediatric cardiology units in the teaching hospital which support the neonatal care units for early diagnosis of CHD.
- Large VSD have risk of pulmonary hypertention and associated symptoms.
- Cardiac complication requiring frequent follow up or even hospitalization or intervention,including pulmonary hypertention, aortic regurgitation , endocarditis and arrhythmia.

Recommendations

- Screening echo for each suspected case of CHD in neonatal period and for all genetic syndromes to aid in early diagnosis and avoid late complications.
- Education of people about the Consanguinity marriage role in the inheritance of genetic diseases and CHD.
- Early diagnosis and referral to pediatric cardiologist and cardiology center for proper management and follow up is important to decrease and avoid the complication.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

Ethical approval for this study was done

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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