

## ORIGINAL ARTICLE

# PEDIATRIC SUBTYPES OF VENTRICULAR SEPTAL DEFECTS WITH PERCENT CLOSURE AT IBN-SENA TEACHING HOSPITAL IN THE CITY OF MOSUL – IRAQ

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### Summary

The ventricular septal defect (VSD) is a congenital lesion characterized by the presence of an opening between cardiac chambers. The treatment might involve medical therapy to control symptoms or in certain cases, surgical resuscitation might be required.

**Objectives:** The study was conducted to establish a database about the prevalence and pattern of VSD and their prognosis in children referred to by echocardiography in Ibn-Sena Teaching Hospital over the period of March 2019 to January 2020. **Method:** The present study is a prospective descriptive study conducted on all patients diagnosed with cardiac lesions revealed by echocardiography. The sample included in the study involves newborns (day 1) to 14-years-old children.

**Result:** Out of 500 children included in the study; most of these cases were cyanotic congenital heart lesions and out of which two-third were perimembranous defects.

**Conclusion:** The study concluded a higher prevalence of non-cyanotic lesions and peri-membranous type is the commonest VSD lesion.

*Key words:* ventricular septal defect; echocardiography; congenital; cardiac

### Introduction

The most common acceptable definition for ventricular septal defect (VSD) is the presence of a hole between ventricles due to a congenital defect. This definition has been introduced by the International Society for the Nomenclature of Pediatrics and Congenital Heart Disease (ISNPCHD) (1). However, different types have existed with no clear distinction between them and there is no consent about their nomenclature. The diagnosis involves clinical diagnosis through observed signs and symptoms together with chest radiography, and ECG. The size of the defect and relative vascular resistance of the pulmonary versus systemic beds will determine the severity of the symptoms. Small lesions show proper prognosis with no morbidity and mortality apart from using certain medication in cases of association with congestive heart failure (2).

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There is controversy in the terminology and classification of different types of VSD; however, clinically four classifications are acceptable, these include, anatomical classification, classification based on the size of lesion or defect, classification based on the pressure, and classification based on appearance on ventriculography. To clarify these types and determine the differences between them; more details are required. Based on anatomical classification; the defect is either a small membranous part (the lesion is simple and the defect is on the membrane) or a large muscular part, the large muscular part could be sub-classified based on the involved part of either inlet septum, outlet septum, or trabecular muscle septum (1-3). When the defect extends to the peri-membranous system, the most popular type of VSD and represents 70% of total defects (1). The perimembranous defect is a defect that extends beyond the membrane into muscular tissues resulting in different types based on the anatomical site of involved tissue of trabecular muscle (3). However, sometimes the defects are represented as a multiple muscular lesion called Swiss cheese defect; this type of defect is associated with poor prognosis (4), moreover, the lesion could be presented in the joining portion between left ventricular tissue and right atrium precisely in the atrioventricular septum portion; this type of lesion is rare and is clinically known as Gerbode defect (2).

The classification based on size is determined in relation to the aorta orifice. Small VSD means lesion is smaller than aorta (less than one-third) or up to one-half will be medium lesion or equal to aorta lesion and called large VSD. The third classification was based on the pressure induced by the presence of lesions into (Restrictive VSD, moderately restrictive VSDs, and nonrestrictive VSDs). The fourth classification is based on the ventriculography into a. tubular, window, aneurysmal, and infundibular (2,3).

The patient's clinical manifestation depends on the type of lesion or defect. Infants with small lesions are asymptomatic associated with no morbidity or mortality, normally developed, and are acyanotic alongside normal ECG (3). Conversely, infants with moderate-large lesions are severely affected and presented with cyanosis and sign of CHF, additionally, they show signs of morbidity with delayed growth or reduced weight gain. ECG of Infants with large VSD presented with left-ventricular and atrial hypertrophy or biventricular hypertrophy (5,6). The hypertrophy in the heart could be further documented by radiography showing enlarged cardiac chambers. The location of the defects could be confirmed by echocardiography (3).

The available treatment modalities for VSD infants were directed toward controlling symptoms of CHF using diuretics and vasodilators and the addition of digoxin in severe cases or oxygen in an emergency situation (2). Trabecular VSD infants required an amendment of the opening by using a closure device because the device can easily be made to be attached to muscle; however, this technique is not practical in the case of perimembranous VSDs due to proximity to the valve. The surgical step is valuable only in the case of the left-right shunt. Albeit, the surgery is indicated it could be postponed to the age of 12 years old unless otherwise urgently indicated as the case with the presence of pulmonary vascular resistance (2,3).

The present study was conducted to evaluate the common types of VSD in our locality and determine the rate of each type of VSD in relation to the total number of patients presented to the clinic. The study also shows the prognosis percentages of each type and the fate of overall patients.

## **Patients and methods**

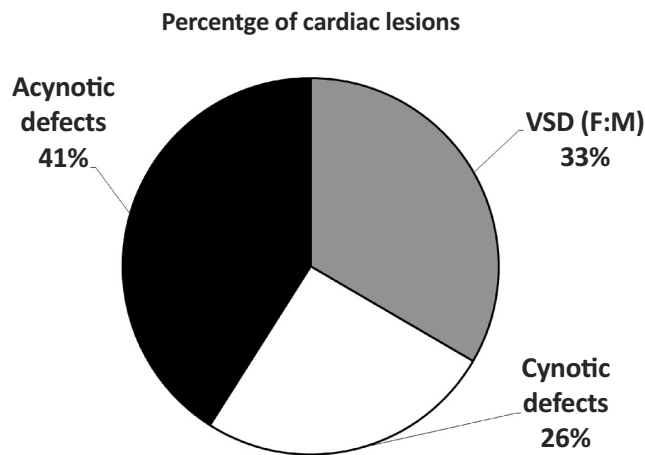
The prospective descriptive study was conducted in the Pediatric echo-cardiac clinic from March 2019 to January 2020 in Ibn-Sena Teaching Hospital in Mosul- Iraq and this study has been approved by ethical committees approval in the Clinical pediatric department, Nineveh College of Medicine- University of Mosul.

All patients who were diagnosed with congenital heart disease were enrolled in this study, and these include newborns at (day 1) to 14-years-old children. For each patient; age, sex, and type of lesion were recorded. Premature newborns and newborns with inherited heart diseases (such as rheumatic heart or mitral valve prolapsed) were excluded from the study.

The size of the aortic valve annulus is a frequent standard value. The lesion is a large lesion if its size is equal to the diameter of the aortic annulus, lesion size equal to half the diameter of the aortic root described as moderate lesions, and those that are less than half the diameter of the aortic root is considered small lesions (1).

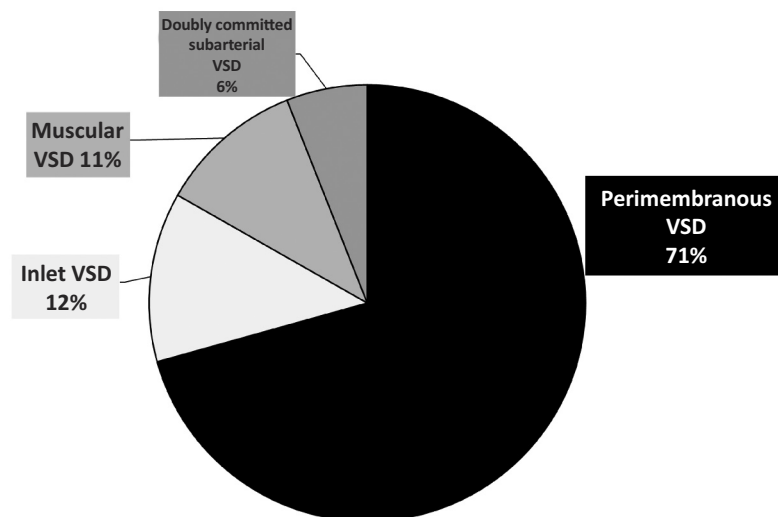
## Results

The number of patients with VSDs was compared to those with noncyanotic and cyanotic congenital heart disease (Figure1). Out of 500 children who participated in the study, 33% of these participants had VSD with a large portion of acyanotic congenital heart lesions and the ratio of these lesions were slightly higher in female's patients compared to males (57.5% vs 42.5%, respectively)



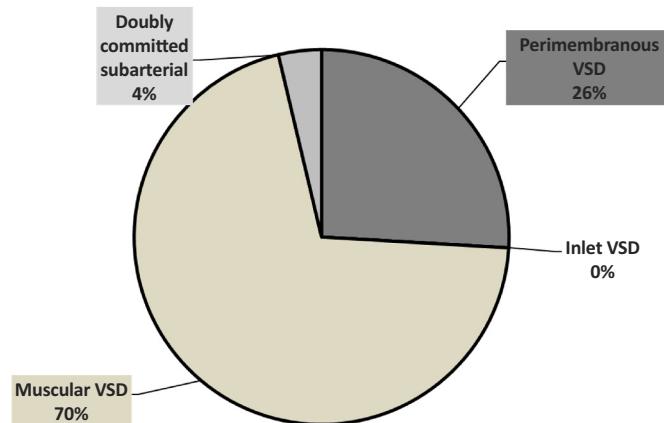
**Figure 1.** Distribution of cardiac lesions in the studied group (n=500), the percentage of VSD is higher than other lesions. VSD=ventricular septal defects.

The commonest VSD lesion was perimembranous defect (about 71%), whereas less common lesions were by A-V canal (inlet), Muscular and doubly committed subarterial ventricular septal defect (outlet) type lesions represented up to 12.57%, 10.77%, and 6% respectively (Figure 2).



**Figure 2.** Distribution of VSD types.

Regarding closure rate, the muscular type had more tendency for closing then followed by perimembranous, doubly committed subarterial VSD as counted 70%, 26%, 4% respectively were A-V canal (inlet) showed no tendency for closure as shown in Figure 3.



**Figure 3.** Closure rate of different types of VSD.

## Discussion

Congenital heart defects affect about 1% of all newborns. Approximately 30% of congenital heart disease is caused by two defects: ventricular septal defect (VSD) and atrial septal defect (ASD): VSD accounts for 20% and ASD accounts for 10%(1). VSDs are the most prevalent congenital cardiac defects. They represent up to 30-60% of overall births with congenital heart diseases or 2-6 per 1000 births(6, 7). Given that most trabecular VSDs close spontaneously(8), it's unclear whether isolated, small muscular VSDs should even be consider pathologic conditions. According to prospective research, trabecular VSDs close shortly after birth in 80-90 percent of cases, with a frequency of 2-5 per 100 births (9).

This study aimed to demonstrate the different sub-divisions of VSD in Mosul province in Iraq and how they vary from other medical centres which may reflect physicians' experience and familiarity with diagnosis and prognosis, and how it compares to other studies. It is widely acknowledged that improved diagnosis, education, or recognition by general paediatricians, as well as early referral to pediatric cardiologists, has resulted in a rise in the reported prevalence of Congenital Heart Diseases (CHD) (10).

According to the findings of this report, "VSD" as "CHD" is still a significant pediatric cardiac issue in Mosul city-Iraq that requires further focus for early diagnosis. To the best of our knowledge, there is no other local research or records regarding cases of VSD subtypes in Mosul city and the Ninevah governorate in general. A total of 500 cases were included in this study, with 74.4 % (372) of them being diagnosed as acyanotic CHD." This was in close agreement with international studies (5, 9). It is likely that certain cases of CHD will go missed and unaddressed, the majority of them are neonates born at home or who perish without medical treatment. In our sample, VSD was found to be the most common acyanotic CHD" (167 (33.4 %)). This is consistent with findings from other research (5,6,9,11). As seen in Table 1, the Prince Hashem Hospital observed a higher prevalence than other reports (9). VSD is the most "common acyanotic CHD" reported worldwide, accounting for 25-30% of all CHD. Given that many genetic factors, including chromosomal, single gene, and polygenic inheritance, have been identified as causes of VSD(12). Variation in genetic makeup and ethnicity may account for this disparity. However, Non-inherited risk factors, such as maternal infection (Influenza, measles, and febrile illness), maternal diabetes mellitus, and phenylketonuria, have been linked to the progression of VSDs. VSDs have also been related to contaminants like alcohol, tobacco, cocaine, and certain drugs like metronidazole and ibuprofen (6,13).

**Table 1.** Comparative study of "VSD" lesions with other studies.

Types of "CHD"	Prince Hashem Hospital	Fuad Abbag (Saudi Arabia)	Alberta Heritage pediatric cardiology program (Canada)	Mary K.M. Shann (Taiwan)	Ibn-Sena hospital Mosul
Ventricular Septal Defect	43.4%	32.5%	34.4%	39.3%	33.4%

As the child grows, small VSDs may close spontaneously. A larger VSD almost always necessitates surgery. Regardless of the type, if a VSD is diagnosed, the child's clinical condition must be monitored by a cardiologist regularly to determine if the spontaneous closure occurs. If the VSD has not closed on its own, it will be repaired to prevent lung problems from developing from prolonged exposure to extra blood flow (14). The natural progression of a VSD can range from spontaneous closure (commonest) to congestive heart failure (less common) to death in infancy (rare) and is largely determined by the size of the deform, which represents the sort of symptoms identified, the severity of the condition, and the onset of age at which they first appear (15).

A considerable number of small size defects (30–50%) "close spontaneously," most commonly throughout the first two years of life. Small muscular VSDs have a higher chance of closing (up to 80%) than membranous VSDs (up to 35%)(16). The outlet VSD has a low rate of spontaneous closure, whereas inlet VSD does not close spontaneously very often (16,17). These clinical data are almost identical to our findings. Although "spontaneous closure" has been reported in adults, the overwhelming majority of lesions have been followed up to close before the age of four(18). The majority of children with minor defects are asymptomatic, with no signs of increased heart size, pulmonary arterial pressure, or resistance (18). However, infective endocarditis has been described as a long-term complication(1). Several long-term investigational studies reported a higher incidence of arrhythmia, sub-aortic stenosis, and exercise intolerance in adults with unoperated small VSDs (19,20,21,22).

The prognosis of a VSD is typically excellent when diagnosed and repaired early, and minimal follow-up is required. The outlook is usually much more grim when a large VSD is discovered in adulthood age, if complications arise after surgical closure, or if a large VSD is never repaired (23). The development of pulmonary hypertension or Eisenmenger's syndrome would also be a possibility in such cases (23,24). In order to avoid infective endocarditis, necessitates maintaining good dental hygiene and antibiotics used as prophylaxis. Kidd *et al.* also found a small but significant risk of malignant ventricular arrhythmia. In children with the profoundly committed and per membranous type of VSD, the prevalence of aortic leaflet prolapse and aortic insufficiency increases with age (25).

The majority of children who have endured corrective surgery of VSD will live healthier lifestyles. As a result, most children's activity levels, appetite, and growth rate will return to normal. In the general population, the prevalence of congenital heart disease is about 0.8%. Yet, it rises to 2–6% for second pregnancy after the birth of a child with "congenital heart disease" or if a parent is affected (12). Hence why parents who have a child with "congenital heart disease" should seek genetic education about the likelihood of developing a cardiac lesion in the next newborn. The type of lesion in the first child has a big impact on the likelihood of recurrence. When two first-degree families have "congenital heart disease," the risk for a subsequent child can be as high as 20% to 30% (12). When a second child is diagnosed with congenital heart disease, it usually falls into the same category as the lesion in a first-degree relative. However, the degree of severity, as well as the presence of associated defects, may be considerably different. A thorough echocardiography screening of first-degree relatives will often reveal mild forms of CHD that were previously undetectable (26). The most credible way of providing the family with up-to-date information about the likelihood of recurrence is to consult with a competent genetic counsellor. Given that the CHD is inherited in a multifactorial manner, genetic counselling for patients with a family history of congenital heart diseases is critical (26).

## Conclusions

This study provides an overview of the subtypes of VSD as non-cyanotic "congenital heart disease" at Ibn-Sena Teaching Hospital in Mosul city-Iraq. The majority of patients diagnosed with "congenital heart disease", have VSD non-cyanotic lesions. This study also showed that early detection of "congenital heart diseases" is crucial for proper management and avoidance of complications, and "two- dimensional-echography" with Doppler examination appears to be the gold standard for diagnosis. Our study could contribute to the creation of a national pediatric cardiac database. For the diagnosis of congenital heart disease, echocardiography is the best diagnostic investigation to be considered.

## Adherence to Ethical Standards

The study was approved by an ethical committee in the university of Mosul.

## Conflict of Interest

The authors have no conflicts of interest regarding the publication of this article.

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