SJIF Impact Factor: 6.711

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 9
Issue: 5
Page N. 192-198
Year: 2025

Original Article

www.wjahr.com

ISOLATED VENTRICULAR SEPTAL DEFECT IN CHILDREN OF MOSUL

[1]*Manhal Zuhair Hammoodi, [2]Aysar Mohammed Taha and [3]Hasson Muhsin Hussein

[1]Al Wattar, M.B.Ch.B./F.A.B.H.S (Ped.), Department of Pediatric, Ibin Al Atheer Teaching Hospital.
[2]M.B.Ch.B./F.A.B.H.S (Ped.), Department of Pediatric, Ibin Al Atheer Teaching Hospital.
[3]M.B.Ch.B./F.A.B.H.S (Ped.), Department of Pediatric, Ibin Al Atheer Teaching Hospital.

Article Received date: 07 March 2025 **Article Revised date:** 28 March 2025 **Article Accepted date:** 18 April 2025

***Corresponding Author: Manhal Zuhair Hammoodi**

Al Wattar, M.B.Ch.B./F.A.B.H.S (Ped.), department of Pediatric, Ibin Al Atheer Teaching Hospital.

ABSTRACT

Background: Ventricular septal defect (VSD) is the most common cardiac malformation and accounts for one third of congenital heart disease. Defects may occur in any portion of the ventricular septum, but most are of the membranous type. It is often considered that congenital heart disease is due to polygenic or multifactorial inheritance, in which, as yet unknown, inherited and environmental factors combine to cause the malformation. **Objectives:** Is to find out how common the VSD among all CHD in Mosul. In addition; to find out the clinical features and the complications of VSD. **Methods:** This is descriptive, retrospective, cross-sectional study included Sixty patients diagnosed with VSD by echocardiography admitted to the department of pediatric medicine at Ibin Al Atheer Teaching Hospital, from January 2024 to the end of December 2024. All the patients were sent for CXR, ECG, two-dimensional echocardiography and color Doppler was done. Patients who were having more than a VSD were excluded from the study. Cardiac catheterization was not done to any patient. Twenty-five of normal children of matched age & sex with patients consulting the outpatient clinic were taken as a control group. **Results:** Out of (3951) patients admitted to Ibin Al Atheer Teaching Hospital during the period of the study, (194) had CHD and (60) of them had VSD which make (1.15%) of total admission and (30.92%) of CHD in this study. The age range was between 1 month - 12 years with the of mean 22.43 months and 60% of them were under 1 year of age ,30% between 1 and 5 years and 10% was older than 5 years, 31 were males and 29 were females. Tachypnea was the commonest presenting symptom followed by cough and poor weight gain. About half of the study patients have a weight below third percentile. Failure to thrive was found to be the commonest complications followed by recurrent chest infection and heart failure. **Conclusion:** VSD is the most common defect among all CHD. The clinical presentation and natural history of isolated ventricular septal defects in children depends upon the size of the defect. Prematurity and maternal diabetes mellitus were not significant risk factors but consanguinity was an important risk factor for the development of VSD.

KEYWORDS: Congenital heart disease, failure to thrive, Mosul, Iraq.

1- INTRODUCTION

Ventricular septal defect (VSD) is the most common cardiac malformation and accounts for one third of congenital heart disease. Defects may occur in any portion of the ventricular septum, but most are of the membranous type[1] The ventricular septum may be divided into a small membranous portion and a large muscular portion. The muscular septum has three components: the inlet septum, the trabecular septum, and the outlet (infundibular or conal) septum.[2] The trabecular septum (also simply called muscular septum) is further divided into anterior, posterior, middle, and apical portions.[3] Therefore, a VSD may be classified as a membranous, inlet, outlet or infundibular, midtrabecular or midmuscular, anterior trabecular or anterior muscular, posterior trabecular or posterior muscular, or apical muscular defect.[4] Although some CHD have different incidences in males and females, VSD appear to be equally distributed by sex.[5-6]

Environmental factors and genetic defects can be the cause of congenital heart disease, but in most children no specific cause is found.[7] In early pregnancy maternal infection, illness or ingestion of certain drugs (Alcohol and valproate) can result in cardiovascular abnormality.[8] There is also an increased incidence of a variety of lesions in the infants of diabetic mothers, with the risk

being minimized by good control of the diabetes in early pregnancy.[9-10]

Cardiac defects are associated with a wide range of chromosomal or genetic abnormalities, other syndromes and associations. It is thus appropriate to perform chromosomal analysis on infants and children with congenital heart disease who have dysmorphic features, low birth weight, other malformations, developmental delay, and failure to thrive or short stature. Single gene defects are increasingly being identified and some are associated with congenital heart disease.[11]

Additionally; Charge syndrome also has a dominant inheritance. Other relatively common conditions with a usually sporadic occurrence are VACTERL and CHARGE associations.[12] It is often considered that congenital heart disease is due to polygenic or multifactorial inheritance, in which, as yet unknown, inherited and environmental factors combine to cause the malformation.[13] Recurrence risk for a cardiac defect depends on the nature of the lesion and relationship of the affected person. However, if the mother is affected there is an even higher risk (5–15%) depending on the lesion.[14-15]

The aim of study is to find out how common the VSD among all CHD in Mosul. Moreover; to find out the most common presenting feature and the age of first presentation. Furthermore; to find out the relation between the size of the VSD and the growth parameters and to see the frequency of the complications of VSD.

PATIENTS AND METHODS

This is descriptive, retrospective, cross-sectional study included Sixty patients diagnosed with VSD by echocardiography admitted to the department of pediatric medicine at Ibin Al Atheer Teaching Hospital, from January 2024 to the end of December 2024.

Their age was between 1 month - 12 years (mean 22.43 months), 31 of them were males and 29 were females. Complete history was taken from all the patients by giving the parents a questionnaire form and a full physical examination was done to all the patients.

All the patients were sent for CXR, ECG, two-dimensional echocardiography and color Doppler was done. Patients who were having more than a VSD were excluded from the study. Cardiac catheterization was not done to any patient.

Twenty-five of normal children of matched age & sex with patients consulting the outpatient clinic were taken as a control group.

3. RESULTS

Out of (3951) patients admitted to Ibin Al Atheer Teaching Hospital during the period of the study, (194) had CHD and (60) of them had VSD which make (1.15%) of total admission and (30.92%) of CHD in this study.

1- Age of first diagnosis of the patients with VSD

Table 3.1: Shows age of the patients when they were firstly diagnosed.

Age	Number of Patients	%
At birth	0	0
1st month	11	18.33%
2nd month	23	38.33%
3rd month	6	10%
4th month	7	11.6%
5th month	2	4.33%
6th month	4	6.66%
7th month	3	5%
1.5 year	4	6.66%
Total	60	

2- Age of the patients at the recent admission

Table 3.2: Shows the age of the patients at the recent admission.

Age at admission	Number of patients	%
Birth to 12 months	36	60%
13 month - 5 years	18	30 %
> 5years - 13 years	6	10%

3- Gestational age of patients

Table 3.3: Shows gestational age of the patients.

Patients			Control Group	
Gestational age	No.	%	No.	%
Full term	56	93.33%	23	92 %
Post term	4	6.66 %	1	4 %
Pre term	0	0	1	4 %

4- Sex distribution

Table 3.4: Shows sex distribution of the patients.

Sex	Number of Patients	%
Male	31	51.66 %
Female	29	48.33 %

5- Maternal health during pregnancy

Table 3.5: Shows the maternal problems during pregnancy.

Maternal disease	Number of Patients	%
1- Un diagnosed fever	11	18,33 %
2- Urinary tract infection Proved by urine culture	3	5 %
3- Diabetes mellitus	1	1.66 %

6- Family history

Positive family history of CHD was found in (4) patients (6.66 %) and consanguineous marriage among parents was found in (19) patients (31.66 %), while consanguineous marriage among parents of control group was found only in one patient (4 %).

Table 3.6: Shows the consanguineous marriage & family history.

Patient	No.	%	Control No.	%
Positive family history	4	6.66 %	0	0
consanguineous marriage	19	31.66 %	1	4%

7- Presenting symptoms

Table 3.7: Shows presenting symptoms among patients with VSD in correlation with the size of VSD*.

Symptoms	Small VSD		Moderate & Large VSD		Total patients	
	No.	%	No.	%	No.	%
1- Tachypnea	4	26.66%	43	95.55 %	47	78.33%
2- Cough	9	60 %	36	80 %	45	75 %
3- Poor weight gain	5	33.3 %	33	73.33%	38	63.33 %
4- Dyspnea on exertion	3	20 %	20	57.89 %	23	38.33%
5- Feeding difficulties	4	26.66 %	20	44.44 %	24	40 %
6- sweating	2	13.33 %	32	51.11 %	36	60 %
7- Asymptomatic	4	26.66 %	0	0	4	6.66 %
8- Wheezes	0	0	1	2.22 %	1	1.66 %

8- Growth Parameters

Table 3.8: Shows weight, length or height percentile for age of patients with VSD & weight of control group.

Growth Parameter	< 3rd percentile		between 3rd and 10th percentile		between >10th and 50th percentile		above 50th percentile	
	No.	%	No.	%	No.	%	No.	%
Weight of the patients	28/60	46.66%	24/60	40 %	5/60	8.33%	3/60	5%
Length or height of patients	2/60	3.33 %	24/60	40 %	24/60	40 %	10/60	16.66 %
Weight of control group	1/25	4 %	8/25	32 %	14/25	56 %	2/25	8 %

Comparing weight < 3rd percentile between the patients (46.66%) and control group (4 %), (z= 5.5 $P < 0.001$ very highly significant).

9- Presenting signs

Table 3.9: Shows presenting signs of patients with VSD.

Signs	No. of Patients	%
1- feature of Down syndrome	3	5 %
2- Chest deformity	7	11.66 %
3- Visible apex beat	35	58.33 %
4- Palpable left parasternal heave	12	20 %
5- Systolic thrill	42	70 %
6- First heart sound:		
Normal	12	20 %
Cover by murmur	48	80 %
7- Second heart sound		
Normal	42	70 %
Loud	14	23.33 %
Diminish	4	6.66 %
8- Murmur:		
Pansystolic	60	100 %
Additional diastolic murmur	1	1.66 %
9- Character of murmur:		
Harsh	54	90 %
Blowing	6	10 %
10- Area of maximum intensity of the murmur:		
Lower left sternal border	60	100 %
Other area	0	0
11- Auscultation of the chest:		
Crepitation	31	51.66 %

Wheezes	1	1.66 %
12- Examination of the abdomen:		
Hepatomegaly (> 2 cm)	32	53.33 %
Splenomegaly	2	3.33 %

10- Presenting diseases

Table 3.10: Shows presenting disease which lead to diagnosis of VSD.

Presenting disease	No. of patients	%
1- Chest infections	30	50%
2- Accidental (by finding systolic murmur on auscultation)	12	20 %
3- Failure to thrive	13	21.66 %
4- Heart failure	5	8.33 %

11- Investigations of patients

Table 3.11: Shows the result of investigations which of our patients.

1- Chest X- ray:	No. of Patients	%
A/ Heart size:		
Normal	15	25 %
Border line	5	8.33 %
Enlarged	40	66.66 %
B/ Lungs:		
Normal	6	10 %
Increase pulmonary vascular markings	34	56.66 %
Pneumonia	25	41.6 %
Pulmonary edema	2	3.33 %
2- Electrocardiogram:		
Normal	16	26.66 %
Left Ventricular hypertrophy	30	50 %
Biventricular hypertrophy	14	23.33 %
Left atrial enlargement	20	33.33 %
Normal P- wave	45	75 %
Peak P- wave	15	25 %
3- Echo cardiogram:		
Small VSD	15	25 %
Moderate and Large VSD	45	75 %

12- Treatment of patients with VSD

Table 3.12: Shows types of treatment received by our patients.

Types of treatment	No. of Patients	%
a- Medical		
1- Antibiotics for chest infection	45	75 %
2- Diuretics	44	73.33 %
3-Captopril	41	68.33%
3- Digoxin	11	18.33 %
4- Antibiotics prophylaxis for infective endocarditis	22	36.66 %
b- Surgical	0	0

13- Complications and course of VSD

Table 3.13: Shows complications and course of VSD in our patients.

Complications and Course of the disease	No. of Patients	%
1- * Failure to thrive	48	80 %
2- ** Recurrent chest infection	45	75 %
3- Heart failure	41	68.33 %
4- Pulmonary hypertension	12	20%

(as evident from echo study)		
5- asymptomatic	4	6.66 %
6- infundibular Pulmonary stenosis (as evident from echo study)	0	0
7- Infective endocarditis	0	0
8- Eisenmenger syndrome	0	0

* Failure to thrive here defined as weight less than 80 % of the median weight for height or length for the same age and sex in the growth chart which is adopted from NCHS growth Charts by Ross Laboratories.
** Recurrent chest infection was found in 5 patients of the control group (8.33 %), (z=11.13 p< 0.001 very highly significant).

4. DISCUSSION

In this study, VSD is the commonest type of CHD account for (30.92 %). This result is in agreement with the finding of Constantine Mavroudis et al.[16] Moreover; the study found that the most common age of presentation and diagnosis was with in the first year of life and specifically after the age of 2 months probably due that; after birth in patients with a VSD, pulmonary vascular resistance may remain high, and thus the size of the left-to-right shunt may initially be limited. As pulmonary vascular resistance continues to fall in the first few weeks after birth because of normal involution of the media of small pulmonary arterioles, so the size of the left-to-right shunt increases and symptoms starts to appear.[17]

In this study, the majority of patients (93.33%) were full term similar to what was found by Wael Dakkak et al[18], but in other study sevenfold higher incidence of CHD in the VLBW neonates and an eleven fold higher incidence in the extremely low birth weight population, as compared to the reported incidence of 0.5–0.8% in the general population.[19]

There is no sex deference in the incidence of VSD in this study; Male to female ratio was 1.06: 1. This finding is similar to what was found by Saiful Islam et al.[20] Additionally; maternal diabetes mellitus history was found in one patient only which means that maternal diabetes mellitus is not related to VSD, which not in same way of Andreas S. Papazoglou et al meta-analysis.[21] Anyhow; small sample size might cause such findings. Furthermore; in the present study VSD seems to be linked to consanguineous communities. Consanguinity among the parents of my patients was found in (19) of patients, which is in agreement with finding of Mohammad Abdullah Al Mamun.[22]

The commonest presenting symptoms in my patients were tachypnea (78.33%), cough (75%), and poor weight gain (63.33 %). This is consistent with the study done by Sarah Keaney et al.[23] From the other hand; the size of the VSD play an important role in the development of the symptoms in the patients, about (26.66 %) of the patients with small VSD were asymptomatic, while none of patients with moderate and large VSD was asymptomatic. Tachypnea was found in (95.55 %) in those with moderate and large VSD but it was found in (26.66%%) in those with small VSD, this is may be due to the complication of heart failure and lower respiratory tract infection which are more likely in patients with moderate and large VSD. While; the body weight of (46.66 %) of the study patients fall below the third percentile for their age where as only (4 %) of the control group the body weight was below the 3rd percentile. But; only one patient (6.66 %) with small VSD his weight was below the 3rd percentile while other 27patients (60 %) with moderate and large VSD their weight was below the 3rd percentile. This mean that poor weight gain is more common in patient with moderate and large VSD than in patient with small VSD. The height and length of the study patients was not affected as the weight and it was between the third and 50th percentile of height for age in the majority of my patients (80 %), this result is similar to that by Kareem Mohammed Awd Ahmed et al.[24]

Down syndrome was the most common syndrome found in association with VSD in 3 of my patients (5%). This is similar to that found by Konstantinos Dimopoulos et al.[25]

Failure to thrive was the commonest complication among my patients which was found in 48 patients (80%), while; recurrent chest infection was the second most common complication which was found in 45 patients (75 %). Moreover; heart failure was found in 41 patients (68.33) this is consistent with the study Mavroudis et al.[16]

The small sample size is a major limitation of this study. A bigger sample size could have produced more accurate reporting. The study only included hospitalized children during the study period, which may have resulted in significant missing of asymptomatic cases.

5. CONCLUSION

VSD is the most common defect among all CHD excluding patent ductus arteriosus in preterm neonates, bicuspid aortic valve, physiologic peripheral pulmonic stenosis, and mitral valve prolapse. VSD was mainly diagnosed with in the first year of life mainly in the second month of life. The clinical presentation and natural history of isolated ventricular septal defects in children depends upon the size of the defect. Prematurity and maternal diabetes mellitus were not significant risk factors but consanguinity was an important risk factor for

the development of VSD. Failure to thrive was the commonest complication.

ACKNOWLEDGEMENT

We are grateful for the help provided by the medical team at Ibin Al Atheer Teaching Hospital as well as the careful consideration received from the Nineveh Directorate of Health. Without the help of each of these individuals, this study would not have been possible.

CONFLICT OF INTERTEST

About this study, the authors disclose no conflicts of interest.

REFERENCES

1. Chaithra S, Agarwala S, Ramachandra NB. High-risk genes involved in common septal defects of congenital heart disease. Gene, Oct. 5, 2022; 840: 146745.
2. Triposkiadis F, Xanthopoulos A, Boudoulas KD, Giamouzis G, Boudoulas H, Skoularigis J. The interventricular septum: structure, function, dysfunction, and diseases. Journal of clinical medicine, Jun. 6, 2022; 11(11): 3227.
3. Natarajan S, Cohen MS. Ventricular septal defects. Echocardiography in Pediatric and Congenital Heart Disease: From Fetus to Adult, Dec 27, 2021; 247-66.
4. Andugala S, Grant C, Powell J, Marathe S, Venugopal P, Alphonso N. Three-dimensional printing in the closure of multiple muscular ventricular septal defects. Operative Techniques in Thoracic and Cardiovascular Surgery, Jun. 1, 2024; 29(2): 184-201.
5. Eckerström F, Nyboe C, Redington A, Hjortdal VE. Lifetime burden of morbidity in patients with isolated congenital ventricular septal defect. Journal of the American Heart Association, Jan. 3, 2023; 12(1): e027477.
6. IPINGE LN. Clinical epidemiology of atrioventricular septal defects among children at Windhoek Central Hospital, 2015-2020 (Doctoral dissertation, University of Namibia).
7. Boyd R, McMullen H, Beqaj H, Kalfa D. Environmental exposures and congenital heart disease. Pediatrics, Jan. 1, 2022; 149(1): e2021052151.
8. Kaleelullah RA, Garugula N. Teratogenic genesis in fetal malformations. Cureus, Feb. 5, 2021; 13(2).
9. Ornoy A, Becker M, Weinstein-Fudim L, Ergaz Z. Diabetes during pregnancy: a maternal disease complicating the course of pregnancy with long-term deleterious effects on the offspring. a clinical review. International Journal of Molecular Sciences, Mar. 15, 2021; 22(6): 2965.
10. Patel DR, Kanubhai PA, Sethi DA, Dhangar DK. Clinical Profile and Outcome of Infant of Diabetic Mother in Tertiary Care Newborn Care Units. Res. J. Med. Sci., Jan. 27, 2025; 19: 262-8.
11. Wang H, Lin X, Lyu G, He S, Dong B, Yang Y. Chromosomal abnormalities in fetuses with congenital heart disease: a meta-analysis. Archives of Gynecology and Obstetrics, Sep. 2023; 308(3): 797-811.
12. Omer SO, Abu-Sulaiman RM, Alfadhel M. Genetic Basis of CHD and Management Guide for Specific Diagnostic Tests. InManual of Pediatric Cardiac Care, Jul 30, 2024; I: 485-496. Singapore: Springer Nature Singapore.
13. Rachamadugu SI, Miller KA, Lee IH, Zou YS. Genetic detection of congenital heart disease. Gynecology and Obstetrics Clinical Medicine, Sep. 1, 2022; 2(3): 109-23.
14. Maddhesiya J, Mohapatra B. Understanding the genetic and non-genetic interconnections in the aetiology of isolated congenital heart disease: an updated review: Part 1. Current cardiology reports, Mar. 2024; 26(3): 147-65.
15. Ison HE, Griffin EL, Parrott A, Shikany AR, Meyers L, Thomas MJ, Syverson E, Demo EM, Fitzgerald KK, Fitzgerald-Butt S, Ziegler KL. Genetic counseling for congenital heart disease–Practice resource of the National Society of Genetic Counselors. Journal of genetic counseling, Feb. 2022; 31(1): 9-33.
16. Mavroudis C, Backer CL, Anderson RH. Ventricular septal defect. Pediatric cardiac surgery, May 8, 2023: 317-60.
17. Toms R, Singh R. Cardiac Defects—Anatomy and Physiology. InPrinciples of Neonatology, Jan. 1, 2024; 299-316. Elsevier.
18. Dakkak W, Alahmadi MH, Oliver TI. Ventricular septal defect. InStatPearls [Internet] 2024 Oct 16. StatPearls Publishing.
19. Martins JG, Abuhamad A. Fetal Growth Restriction. InMaternal-Fetal Evidence based Guidelines, Mar. 29, 2022; 482-499. CRC Press.
20. Islam MS, Moniruzzaman M. Congenital heart diseases: a review of echocardiogram records. KYAMC Journal, May 9, 2018; 9(1): 35-8.
21. Papazoglou AS, Moysidis DV, Panagopoulos P, Kaklamanos EG, Tsagkaris C, Vouloagkas I, Karagiannidis E, Tagarakis GI, Papamitsou T, Papanikolaou IG, Christodoulaki C. Maternal diabetes mellitus and its impact on the risk of delivering a child with congenital heart disease: a systematic review and meta-analysis. The Journal of Maternal-Fetal & Neonatal Medicine, Dec. 12, 2022; 35(25): 7685-94.
22. Al Mamun MA, Hussain M, Khan MK. Consanguinity and Risk of Congenital Heart Defects in Bangladesh. Dhaka Shishu (Children) Hospital Journal, 2021; 37(1): 34-9.
23. Keaney S. Respiratory Viral Infection Unmasking Congenital Heart Disease. Journal of Pediatric Health Care, Feb. 17, 2025.
24. Ahmed KM, Azab SE, Kamel WI, Doghish AA. Outcomes Of Vsd Repair On Patients Weight Less Than 5 Kg. Ain Shams Medical Journal, Sep. 1, 2023; 74(3): 579-94.

25. Dimopoulos K, Constantine A, Clift P, Condliffe R, Moledina S, Jansen K, Inuzuka R, Veldtman GR, Cua CL, Tay EL, Opotowsky AR. Cardiovascular complications of down syndrome: scoping review and expert consensus. Circulation, Jan. 31, 2023; 147(5): 425-41.