e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(10); 310-314

Original Research Article

Clinical Spectrum of Ventricular Septal Defect in Children in a Tertiary Care Hospital

Sandeep Mogre

Professor, Department of Pediatrics, Pacific Institute of Medical Sciences (PIMS) Umarda, Pacific Hospital Udaipur, Rajasthan, India

Received: 25-07-2024 / Revised: 23-08-2024 / Accepted: 26-09-2024
Corresponding Author: Dr. Sandeep Mogre
Conflict of interest: Nil

Abstract:

Introduction: The most prevalent congenital cardiac condition in children is Ventricular Septal Defect (VSD). The purpose of our study was to examine the clinical characteristics, size, and type of VSD in paediatric patients who were hospitalised at Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, Rajasthan India.

Materials and Methods: The study was conducted in the Department of Paediatrics, Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, Rajasthan India, during August 2023 to September 2024. A thorough history, anthropometric measurement and along with chest X-ray and ECG were done in all the cases. Anthropometric procedures were performed according to standard WHO procedure. Data was entered and analysed by using SPSS-16.

Results: Regarding the clinical presentation, 55.5% of patients presented with cough and fever. Other presentation includes breathlessness (43.3%), feeding problem (37.7%), failure to thrive (37.7%) and fatigue (33.3%). Out of the thirty one patients of small VSD cases in the study, 12 (33.3%) patients were asymptomatic. The major signs in order of frequency were pansystolic murmur (100%), tachypnoea (51.1%), tachycardia (51.1%), crepitations (46.6%), subcostal retraction (37.7%), hepatomegaly (28.8%), and wheeze (26.6%)

Conclusion: In our findings, Most of the patient of VSD was presented in infancy. Perimembranous was the commonest type of VSD. The clinical presentation of VSD varies from asymptomatic to severe symptoms, depending on the size of VSD. Patients of small VSD presented with mild symptoms or were asymptomatic. Moderate and large VSD mainly presented with severe symptoms. Complications like CCF, pulmonary hypertension, malnutrition and failure to thrive were mostly present in patient with moderate-to-large VSD.

Keywords: Ventricular Septal Defect (VSD), Developmental Defect, Ventricular Septum Congenital Heart Diseases (CHDs).

Introduction

Ventricular Septal Defect (VSD) is a developmental defect of the interventricular septum resulting from a deficiency of growth or a failure of alignment or fusion of component parts of ventricular septum. [1] Congenital heart diseases (CHDs) are the most common type of congenital birth defect and the leading cause of infant morbidity and mortality worldwide. The incidence of CHD in different studies varies from approximately 4/1,000 to 50/1,000 live births. [2]

Ventricular Septal Defect (VSD) is the most common cardiac malformation of the heart accounting for 25% of Congenital Heart Disease (CHD). [3] Isolated VSD affects approximately 2–6/1000 live births. [4,5] Ventricular Septal Defect (VSD) is a developmental defect of the interventricular septum resulting from a deficiency of growth or a failure of alignment or fusion of component parts of ventricular septum. [6] Soto et al [5] classified VSD into 3 types: perimembranous, muscular and Doubly Committed Subarterial (DCSA) types depending upon the location. Perimembranous defect is the commonest accounting for about 70–80% of cases. [7,8] The natural course of VSD depends to a large degree on the size of the defect. Small VSD with trivial left to right shunt and normal pulmonary arterial pressure are asymptomatic and is usually found during routine physical examination. Large VSD with excessive pulmonary blood flow and pulmonary hypertension are responsible for dyspnoea, feeding difficulties, poor growth, profuse perspiration and cardiac failure in early infancy. [3]

CHD symptoms may present at birth or may appear later in life. Signs and symptoms of CHDs vary from mild to severe depending on the severity and type of the heart defect. Some heart defects might have little or no symptoms while others might have

serious symptoms, including blueish skin, lips, or nails (cyanosis), hypersomnia, troubled breathing or fast breathing, poor circulation, getting unusually tired or breathing difficulty when exercising, heart murmur (a whooshing or swishing sound made by turbulent blood flow through the heart valves), and pounding heartbeat or weak pulse.

Approximately 10 % of infant mortality in our country is due to CHDs. In developing countries CHDs causes deaths of thousands of children. [9] Due to improved and more available diagnostic facilities many CHDs are diagnosed in initial years of life. In spite of early diagnosis overall prognosis is still poor because of lack of centres expert in correction of CHDs. Early, accurate diagnosis and timely intervention is the key for better prognosis in CHD. Emergence of cross-sectional echocardiography and then colour flow mapping in 1980s has provided a unique tool to study noninvasively the change in form and function of congenitally malformed hearts also the response and sequelae of interventions

Although a few studies have been conducted in India, CHD especially VSD in children have not been studied thoroughly in this part. Previous studies also lack information regarding clinical profile and size and type of lesion. Our study aims to present a single centre experience of VSD in children at Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, and Rajasthan India. The results of this study will be useful for initiating early diagnosis and proper management, which will result in decreasing the mortality and morbidity

Materials and Methods:

The study was conducted in the Department of Paediatrics, Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, Rajasthan India, during August 2023 to September 2024. All children aged 1 month to 12 years attending Paediatric OPD/IPD with clinical diagnosis of VSD were evaluated by echocardiography to confirm the diagnosis. Only cases of isolated VSD (Absence of other cardiac anomaly) were included in the study. Size and location of the defect were identified by two dimensional transthoracic, colour Doppler echocardiography in the Department of Cardiology. Patients were grouped into three different classes: small, moderate and large based on size of aortic root.

Lesions that approximate the size of the aorta are considered large; lesion one-third to two thirds of the diameter of aorta are moderate; and lesions less than one-third the aortic root diameter are considered small.8 VSD were classified as perimembranous, muscular, Doubly Committed Subarterial (DCSA), and inlet VSD according to Soto's classification. A thorough history, anthropometric measurement and along with chest X-ray and ECG were done in all the cases. Anthropometric procedures were performed according to standard WHO procedure. Data was entered and analysed by using SPSS-16.

Result

Amongst the 90 patients, 50 patients (55.5%) had their first symptom before the age of one year and only 12 (13.3%) had their first symptom after 5 years of age. Similarly, 42 (46.6%) patients were below one year of age at presentation (Table 1).

Table 1: Age of Patient (n=90)

Age Group	At Presentation	At First Symptom
1 month to <1 year	42 (46.6%)	50 (55.5%)
1 year to <5 years	25 (27.7%)	18 (20%)
5 years to 12 years	13 (14.4%)	12 (13.3%)
Total	**90 (100%)**	**90 (100%)**

Regarding the clinical presentation, 55.5% of patients presented with cough and fever.

Other presentation includes breathlessness (43.3%), feeding problem (37.7%), failure to thrive (37.7%) and fatigue (33.3%). Out of the thirty one patients of small VSD cases in the study, 12 (33.3%) patients were asymptomatic. The major signs in order of frequency were pansystolic murmur (100%), tachypnoea (51.1%), tachycardia (51.1%), crepitations (46.6%), subcostal retraction (37.7%), hepatomegaly (28.8%), and wheeze (26.6%) (Table 2).

Table 2: Signs and Symptoms (n=90)

Symptoms	N=90	Percentage
Cough	50	55.5
Fever	50	55.5
Breathlessness	39	43.3
Feeding problem	34	37.7
Failure to thrive	34	37.7

Fatigue	30	33.3
Asymptomatic	12	13.3
Pansystolic murmur	90	100
Tachypnoea	46	51.1
Tachycardia	46	51.1
Crepitations	42	46.6
Subcostal Retraction	34	37.7
Hepatomegaly	26	28.8
Wheeze	24	26.6

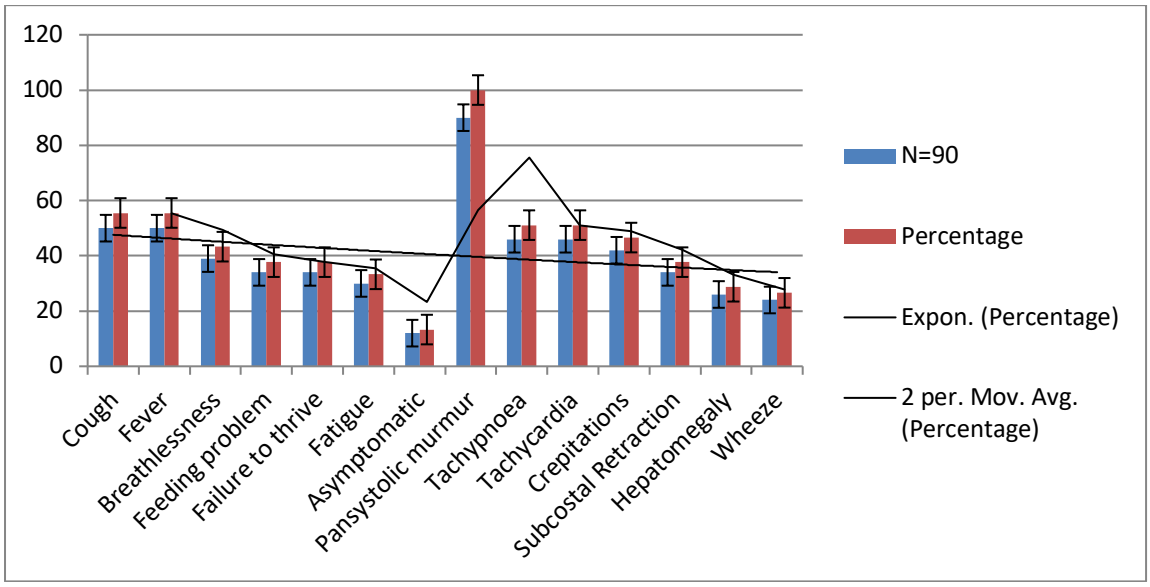

Figure 1: Signs and Symptoms

Among the complications that were frequently associated were pneumonia (44, 48.8%) followed by malnutrition (39,43.3%) and CCF (27, 30%). Pulmonary hypertension accounted for (12, 13.3%) of the cases (Table 3). Amongst the 39 cases of malnutrition, 27 patients (34.3%) had severe malnutrition and 12 patients had moderate malnutrition (13.3%).

Complications were mostly observed in moderate-to- large VSD.

Table 3: Complications of Patient with VSD

Complications	Small VSD (n=35)	Moderate VSD (n=24)	Large VSD (n=21)	Total (N=90)
Pneumonia	20 (57.1%)	13 (54.1%)	11 (52.3%)	44 (48.8%)
CCF	3 (8.5%)	9 (37.5%)	15 (71.4%)	27 (34.3%)
PHTN	Nil	4 (16.6%)	8 (38.0%)	12 (13.3%)
Malnutrition	8 (22.8%)	14 (58.3%)	17 (80.9%)	39 (43.3%)

Discussion:

VSD is the commonest acyanotic congenital heart disease in children. Knowing the clinical profile of VSD will help in early diagnosis and treatment leading to development of fewer complications. In our study, majority of the cases (46.6%) presented in infancy. Among the study subjects, 55.5 %had their 1st symptom below the age of 1 year. This is comparable with the studies done in Hussain M et al. [10]

Our study shows a male preponderance with a male: female ratio of 1.12:1. Chaudhry et al [11] also reported a predominance in males. In the present study regarding the clinical presentation, 55.5% of patients presented with cough and fever. Other presentation includes breathlessness (43.3%), feeding problem (37.7%), failure to thrive (37.7%) and fatigue (33.3%). Out of the thirty one patients of small VSD cases in the study, 12 (33.3%) patients were asymptomatic.

The major signs in order of frequency were pansystolic murmur (100%), tachypnoea (51.1%), tachycardia (51.1%), crepitations (46.6%), subcostal retraction (37.7%), hepatomegaly (28.8%), and wheeze (26.6%)These results were in keeping with what is found in Western literature and also in studies conducted in Pakistan., [10,11] Regarding clinical presentation, the most common symptoms were cough, fever, breathlessness, feeding problem, failure to thrive and fatigue.

These cases were admitted in hospital for other disease condition and incidentally diagnosed during routine examination. Recurrent chest infection has been found to be a frequent complication of VSD in several studies. Among the study group, about 51.4% had past history of recurrent pneumonia. These findings were consistent with the studies done by other workers. [12,13] In our study Among the complications that were frequently associated were pneumonia (48.8%) followed by malnutrition (39,43.3%) and CCF (27, 30%). Pulmonary hypertension accounted for (12, 13.3%) of the cases. Amongst the 39 cases of malnutrition, 27 patients (34.3%) had severe malnutrition and 12 patients had moderate malnutrition (13.3%).

The clinical signs, pansystolic murmur, tachypnoea, tachycardia, crepitations, subcostal retraction, hepatomegaly and wheeze were the most frequent findings. The same picture has been reported in studies done in other developing countries. Complications were mostly observed in moderate-to- large VSD. These cases were admitted in hospital for other disease condition and incidentally diagnosed during routine examination. Recurrent chest infection has been found to be a frequent complication of VSD in several studies. [14,15] Among the study group, about 51.4% had past history of recurrent pneumonia.

Cough, difficulty in breathing, poor weight gain and recurrent chest infection were major presenting symptoms in our study. Poor weight gain and malnutrition is common in children with congenital heart disease. [16] Other clinical observations are similar to findings in other studies. [17,18]

Conclusion

Epidemiological studies of congenital heart diseases (CHDs) are essential for early detection, evaluation, proper management and to understand natural course of disease.

In our findings, Most of the patient of VSD was presented in infancy. Perimembranous was the commonest type of VSD. The clinical presentation of VSD varies from asymptomatic to severe symptoms, depending on the size of VSD. Patients of small VSD presented with mild symptoms or were asymptomatic. Moderate and large VSD mainly presented with severe symptoms.

Complications like CCF, pulmonary hypertension, malnutrition and failure to thrive were mostly present in patient with moderate-to-large VSD. A detailed history, cardiovascular and other systemic examination and diagnostic modalities like chest X-ray, ECG and echocardiography is helpful in diagnosing VSD. Early diagnosis and management will help in preventing the associated complications, thereby reducing the mortality and morbidity in these children.

References

1. Praagh R, Geva T, Kreutzer J. Ventricular septal defects: how shall we describe, name and classify them? J Am Coll Cardiol 1989; 14(5):1298-9.
2. Hoffman JI, Kaplan S. The incidence of congenital heart disease. J Am Coll Cardiol. (2002) 39:1890–900. doi: 10.1016/S0735-1097(02)01886-7.
3. Bernstein D. Acyanotic congenital heart disease: The leftto-right shunt lesions. In: Behrhman RE, Kliegman RM, Jenson HB, et al. (eds). Nelson Textbook of Paediatrics. 17th ed. W.B. Saunders Co, 2004:1888-90.
4. Samanek M, Voriskova M. Congenital heart disease among 815, 569 children born between 1980 and 1990 and their 15-year survival: a prospective bohemia survival study. Paediatr Cardiol 1999; 20(6):411-7.
5. Tynan M, Anderson RH. Ventricular septal defect. In: Anderson RH, Baker EJ, McCartney FJ, (eds.) Paediatric Cardiology. London: Churchill Livingstone, 2002:983- 1014.
6. Praagh R, Geva T, Kreutzer J. Ventricular septal defects: how shall we describe, name and classify them? J Am Coll Cardiol 1989; 14(5):1298-9.
7. Wickramasinghe P, Lamabadusuriya SP, Nerenthiran S. Prospective study of congenital heart disease in children. Ceylon Med J 2001; 46(3):96-8.
8. Perloff JK. Clinical recognition of congenital heart disease. Philadelphia, PA. Saunders, 2003.
9. Cohen AJ, Tamir A, Houri S, Abegaz B, Gilad E, Omohkdion S, Zabeeda D, Khazin V, Ciubotaru A, Schachner A. Save a child's heart: we can and weshould. Ann Thorac Surg. 2001 Feb;71(2):462-8
10. Hussain M, Hussain S, Krishin J, et al. Presentation of congestive cardiac failure in children with ventricular septal defect. J Ayub Med Coll Abbottabad 2010; 22(4):135-8.
11. Chaudhry TA, Younas M, Baig A. Ventricular septal defect and associated complications. J Pak Med Assoc 2011; 61(10):1001-4.
12. Sharmin L, Haque M, Bari M, et al. Pattern and clinical profile of congenital heart disease in a teaching hospital. TAJ: J of Teachers Assoc 2008; 21(1):58-62.
13. Harshanghi S, Itagi L, Patil V, et al. Clinical study of congenital heart disease in infants in tertiary care hospital. Journal of Pharmaceutical and Scientific Innovation 2013; 2(1):15-8.
14. Ramachandran U, Alurkar V, Thaplia A. Pattern of cardiac diseases in children in Pokhara, Nepal. Kathmandu Univ Med J 2006; 4(2):222-7.
15. Memon Y, Majeed R, Memon F. Pattern of congenital heart disease at Liaquat university

hospital Hyderabad. Pak Heart J 2007; 40(1-2):9-13.
16. Cameron JW, Rosenthal A, Olsan AD. Malnutritionin hospitalized children with congenital heart disease. Arch Pediatr Adolesc Med. 1995 Oct; 149(10): 1098-102.
17. Silove ED. Assessment and management of congenital heart disease in the newborn by districtpediatrician. Arch Dis Child Fetal Neonatal Ed. 1994 Jan; 70(1):F71-4.
18. El Hag AI. Pattern of Congenital Heart Disease in Sudanese Children. East Afr Med J. 1994 Sep; 71(9): 580-6.