American Journal of Pediatrics
2025, Vol. 11, No. 3, pp. 166-172
https://doi.org/10.11648/j.ajp.20251103.18

Research Article

Epidemiological Profile, Diagnostic Approach and Associated Complications of Ventricular Septal Defect in a Pediatric Population

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Abstract

Ventricular septal defect (VSD) is the most common congenital heart defect in children, associated with important considerations for disease epidemiology, diagnosis, and long-term outcomes. VSD results from a deficiency in growth or the failure of alignment or fusion of the components of the ventricular septum. This study aimed to describe the epidemiological profile, diagnostic approach, and associated complications of VSD in a pediatric population. This cross-sectional study was conducted in the Department of Pediatric Cardiology, Combined Military Hospital, Dhaka, Bangladesh, from January 2018 to December 2018. This study included 46 pediatric patients less than 12 years old with isolated VSD. The mean age of the participants was 2.78 ±3.44 years, with a slight male predominance (male-to-female ratio of 1.09:1). Most patients (60.87%) were under one year of age. A family history of congenital heart disease was reported in 13.04% of cases. Common presenting symptoms included feeding difficulty (69.57%), cough (60.87%), poor weight gain (54.35%), and head sweating (50%). Respiratory distress was the most frequently observed clinical sign (71.74%), followed by tachycardia (47.83%) and failure to thrive (41.30%). Echocardiography was the definitive diagnostic tool, performed in all cases. Other investigations included ECG (82.61%), chest X-ray (76.09%), and cardiac catheterization (8.70%). Perimembranous VSD was the most prevalent type (67.39%), and the majority of defects were small in size (58.70%). Pulmonary hypertension was the most common complication, noted in 58.70% of patients, followed by aortic cusp prolapse (23.91%) and aortic regurgitation (17.39%). Ventricular septal defect remains a significant pediatric cardiac condition, predominantly affecting infants. Early diagnosis, with the help of a clinical picture and widely and easily accessible diagnostic tools like echocardiography (ECHO), will help initiate timely treatment and decrease mortality. Pulmonary hypertension was the most frequent complication, followed by aortic cusp prolapse and aortic regurgitation.

Keywords

Ventricular Septal Defect, Echocardiography, Pulmonary Hypertension

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Received: 30 June 2025; **Accepted:** 10 July 2025; **Published:** 28 July 2025

1. Introduction

Congenital heart disease (CHD) refers to structural and functional abnormalities of the heart present at birth. These abnormalities may involve the heart itself or the adjacent great blood vessels and can be detected either at birth or later in life. [1] The current global prevalence of CHD is estimated at approximately 8 cases per 1,000 live births, representing about 1.35 million affected newborns each year. [2]

Ventricular septal defect (VSD) is the most common type, accounting for up to 40% of all cardiac malformations. [3, 4] A VSD is a defect in the septum that separates the right and left ventricles, and is often described colloquially as a "hole in the heart". [5] They result from either a deficiency in the growth of the septum or a failure in the alignment or fusion of its parts. [6] Isolated VSD accounts for approximately 25–45% of all congenital heart diseases. [7, 8]

The most widely accepted and practical classification of VSD was proposed by Soto et al. (1980), which categorizes VSD into perimembranous, muscular, and doubly committed subarterial (DCSA) types. [9] Perimembranous defects are the most common, accounting for around 80% of ventricular septal defects. [10] These are often associated with aneurysms or pouches of the septal leaflet of the tricuspid valve, which can partially or completely occlude the defect. [11] Muscular VSD accounts for 5–20% of cases and generally has a better prognosis, as they tend to close spontaneously earlier than perimembranous defects. [12] Doubly committed subarterial VSD represents 5–7% of cases in surgical and autopsy series. [13]

Initial screening tools like chest X-ray and electrocardiogram (ECG) play a supportive role in diagnosing ventricular septal defects (VSDs); however, echocardiography remains the gold standard for definitive diagnosis. It not only confirms the presence of a VSD but also assesses the degree of hemodynamic disturbance and guides the decision between medical, surgical, or transcatheter intervention. [14] On chest X-ray, findings such as cardiomegaly and increased pulmonary vascular markings are commonly seen in moderate to large VSDs, while small defects may present with a normal radiographic appearance. Similarly, ECG results are usually normal in small VSDs but may show left atrial and left ventricular hypertrophy in moderate cases, and biventricular hypertrophy in larger defects. [15, 16]

Echocardiographic assessment, particularly through two-dimensional imaging and color Doppler, is highly effective in pinpointing the location of defects within the ventricular septum. [17] This modality also assists in evaluating left atrial and ventricular dilation, which typically correlates with defect size. [18] Furthermore, echocardiography can reveal aneurysmal tissue formation, which may aid in the spontaneous closure of the VSD. [19] Doppler studies are valuable for visualizing shunting across the defect, with peak Doppler flow velocities being inversely proportional to VSD size. Higher velocities generally indicate smaller defects. [14] Although advanced imaging techniques like CT and MRI are helpful in assessing other congenital heart diseases, they are rarely necessary for diagnosing VSDs. Historically, cardiac catheterization and selective cineangiography were essential for preoperative evaluation. However, with advances in echocardiographic techniques, these invasive procedures are now typically reserved for selected cases, particularly those where elevated pulmonary vascular resistance is suspected. [17]

The natural history of VSD is associated with various complications, among which pulmonary hypertension and prolapse of the aortic valve cusp are particularly notable. This complication occurs more frequently in doubly committed sub-arterial VSDs and less commonly in perimembranous outlet types. [20] Aortic cusp prolapse can progress to secondary aortic insufficiency, which is observed in approximately 5% of patients with VSD. [21] When the aortic leaflet prolapses, it can partially close a moderate or large VSD, thus limiting the left-to-right shunt. Although the prevalence of this complication increases with age, it can also manifest in children younger than six years of age. [8]

Aortic regurgitation may also develop, with its severity varying among individuals. The anatomical and hemodynamic characteristics of DCSA ventricular septal defects significantly influence the risk of developing aortic valve deformities and subsequent aortic regurgitation. [8] Deformities in the aortic cusp can predict progressive aortic regurgitation. [8] Aortic regurgitation tends to be progressive, and even mild regurgitation or isolated aortic valve prolapse without regurgitation is considered an indication for surgical intervention. [22]

Perimembranous outlet VSDs are also associated with infundibular hypertrophy, which can lead to progressive right ventricular outflow tract obstruction requiring surgical management. [23] Although infective endocarditis is rare in children under two years of age, it remains a concern. [23] Additionally, discrete fibrous subaortic stenosis may occasionally occur in association with VSDs, most frequently with perimembranous types, and can appear after either spontaneous or surgical closure of the defect. [23, 24]

Therefore, in this study, we aimed to describe the epidemiological profile, diagnostic approach, and associated complications of VSD in a pediatric population.

2. Materials and Methods

This cross-sectional study was conducted in the Department of Pediatric Cardiology, Combined Military Hospital, Dhaka, Bangladesh, from January 2018 to December 2018. In this study, we included 46 pediatric patients less than 12 years of age with Doppler ECHO findings showing isolated VSD, a defect in the ventricular wall.

The following criteria were used to determine eligibility for

enrollment as study participants:

Inclusion Criteria

a) Patients aged less than 12 years;
b) Patients having an isolated Ventricular Septal Defect;
c) Patients having minor associated anomalies, like a small patent ductus arteriosus, a small secundum atrial septal defect, and mild mitral regurgitation.

Exclusion Criteria

a) Patients with incomplete medical records;
b) Patients having VSD with other complex congenital structural heart disease;
c) Patients whose parents or guardians were not willing to participate in the study.

Data Collection Procedure: The legal guardians or parents of neonates were invited to participate in the current study. Informed written consent was obtained after the study procedure was explained. This cross-sectional study used a structured questionnaire to gather relevant data. The questionnaire included socio-demographic and clinical data at baseline, along with investigation findings and associated complications. Treatment, whether medical or surgical, was selected based on the patient's categorization.

Statistical Analysis: The qualitative data from this study were presented as frequency distributions and percentages, while the quantitative data were expressed in terms of mean and standard deviation. The data were analyzed using SPSS 25 (Statistical Package for Social Sciences) for Windows Version 10. This study was approved by the ethical review committee of the Directorate General Medical Service (DGMS), Dhaka Cantonment.

3. Results

Figure 1 presents the age distribution of the study subjects (n = 46). The majority of participants were under 1 year of age (60.87%), followed by 21.74% aged 6–12 years, and 17.39% were aged 1–5 years.

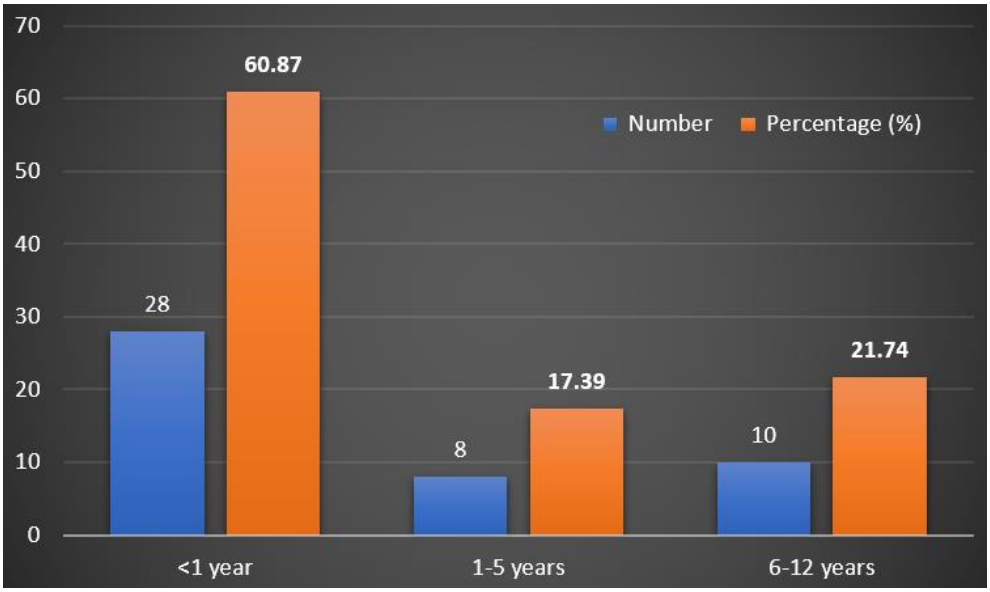

Figure 1. *Age Distribution of Study Subjects (n = 46).*

Table 1. *Demographic and epidemiological characteristics of the study population (n = 46).*

Variable	Number	Percentage (%)
Age (Mean ±SD, years)	2.78 ± 3.44	
Sex		
Male	24	52.17
Female	22	47.83
Residence		
Urban	28	60.87
Rural	18	39.13
Family History of CHD		
Present	6	13.04
Absent	40	86.96

Table 1 shows that the mean age of the children was 2.78 ± 3.44 years. Of the total participants, 52.17% were male and 47.83% were female. The male and female ratio was 1.09:1 in

our study. A majority (60.87%) of the children were from urban areas, while the remaining 39.13% resided in rural settings. Family history of congenital heart disease (CHD) was present in 13.04% of the participants, whereas 86.96% reported no family history of CHD.

Table 2. *Baseline findings of study participants (n = 46).*

Baseline Findings	Number	Percentage (%)
Symptoms		
Head sweating	23	50.00
Dyspnea on exertion	22	47.83
Cough	28	60.87
Feeding difficulty	32	69.57
Poor weight gain	25	54.35
Asymptomatic	14	30.43
Signs		
Respiratory distress	33	71.74
Tachycardia	22	47.83
Fever	11	23.91
Failure to thrive (FTT)	19	41.30
Precordium Findings		
Bulge of the precordium	17	36.96
Cardiomegaly	16	34.78
Thrill	33	71.74
P_2 palpable	5	10.87
Loud P_2	7	15.22
Pansystolic murmur	32	69.57
Ejection systolic murmur	13	28.26

Table 2 summarizes the baseline findings of the study participants (n = 46). Among symptoms, feeding difficulty was reported in 69.57% of participants, cough in 60.87%, poor weight gain in 54.35%, and head sweating in 50%. Notably, 30.43% of participants were asymptomatic. Regarding the clinical signs, respiratory distress was observed in 71.74% of subjects, tachycardia in 47.83%, fever in 23.91%, and failure to thrive in 41.30%. In terms of precordium findings, thrill was mostly present in 71.74% of cases, followed by pansystolic murmur in 69.57%, bulge of the precordium in 36.96%, cardiomegaly in 34.78%, and an ejection systolic murmur in 28.26% of subjects.

Table 3. *Diagnostic modalities used for VSD detection in study subjects (n=46).*

Diagnostic Tool	Number	Percentage (%)
Chest X-ray	35	76.09
ECG	38	82.61
Echocardiography	46	100.00
Cardiac Catheterization	4	8.70

Table 3 outlines the diagnostic tools used among the study population. Echocardiography was performed in all cases (100%), which is the most universally applied diagnostic modality for VSD detection. Electrocardiography (ECG) was conducted in 82.61% of the children, while Chest X-ray was used in 76.09% of cases. Cardiac catheterization was performed in a limited number of patients (8.70%).

Table 4. *Distribution of study patients by type and size of VSD (n = 46).*

Type	Number	Percentage (%)
Perimembranous	31	67.39
Muscular	8	17.39
Inlet	4	8.70
Outlet	3	6.52
Size		
Small (<5 mm)	27	58.70
Moderate (5-10 mm)	15	32.61
Large (>10 mm)	4	8.70

Table 4 shows the distribution of study participants by types of ventricular septal defect (VSD). The majority of participants had perimembranous VSD (67.39%), followed by muscular VSD (17.39%), inlet VSD (8.70%), and outlet VSD (6.52%). The majority of lesions were of small size (<5 mm), accounting for 58.70% of cases. Moderate-sized (5-10 mm) lesions were observed in 32.61% of participants, while large lesions (>10 mm) were present in 8.70% of cases.

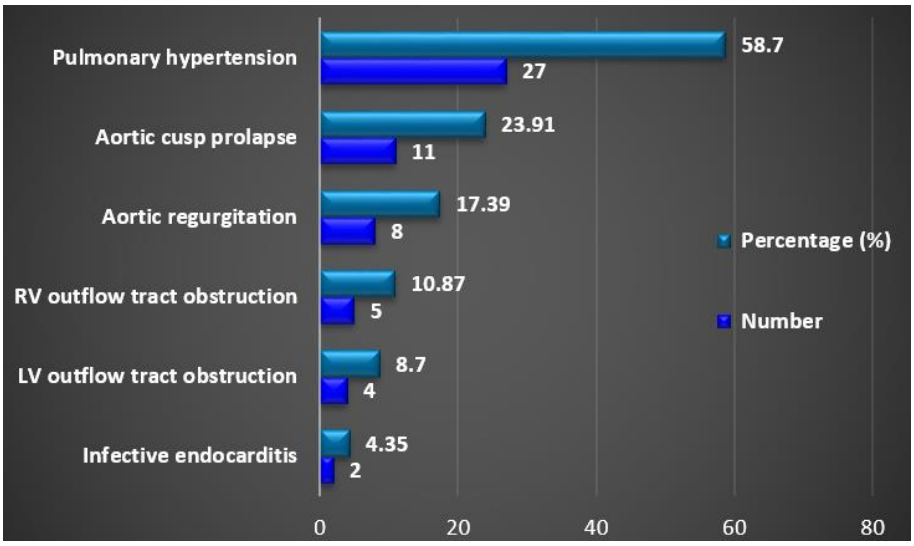

Figure 2. *Associated complications in VSD patients.*

The bar chart illustrates that pulmonary hypertension was the most common complication, observed in 58.70% of participants. Other complications included aortic cusp prolapse (23.91%), aortic regurgitation (17.39%), right ventricular outflow tract obstruction (10.87%), left ventricular outflow tract obstruction (8.70%), and infective endocarditis (4.35%).

4. Discussion

Ventricular septal defect (VSD) is the most common congenital cardiac malformation, accounting for approximately 25% of all congenital heart diseases. [8, 25] It results from deficient growth or failure in the alignment or fusion of the components of the ventricular septum. The natural history of VSD spans a wide clinical spectrum from spontaneous closure to severe complications such as congestive heart failure and early infant mortality. [26] One study analyzing the natural and modified history of isolated VSD reported that VSD constituted 25.5% of all congenital heart defects. In cases of moderate to large VSD, delayed physical growth, decreased exercise tolerance, recurrent pulmonary infections, and congestive heart failure are relatively frequent during infancy. [27]

VSD can be anatomically classified into perimembranous, outlet, inlet, and muscular types. [28] In the current study, perimembranous VSD was the most frequent subtype, observed in 67.39% of participants, followed by muscular (17.39%), inlet (8.70%), and outlet (6.52%) types. These findings are consistent with earlier studies. For instance, Chaudhry et al. (2011) reported that among 1,276 patients, 79.3% had perimembranous VSD, 9.8% had muscular VSD, 6.7% had doubly committed subarterial (DCSA) VSD, and 4.2% had inlet VSD. [23] Similarly, Kazmi et al. (2009) found that the most common type was perimembranous, followed by muscular (11.7%) and DCSA (2.9%) VSDs. [8] These findings align with Western literature, where perimembranous VSDs are also the most common, followed by muscular and DCSA types. [13]

There are limited local studies on the distribution of VSD types. In a study conducted at the National Institute of Cardiovascular Diseases (NICVD) in Karachi, Aziz et al. (2000) found that 92% of VSDs were perimembranous, 7% were DCSA, and only 1.7% were muscular. [29] Sadiq et al. (2002) reported a 32% incidence of VSD among all congenital heart diseases in a tertiary care pediatric cardiology unit. [7] Contrastingly, studies in Southeast Asian children suggest a higher prevalence of DCSA, reaching up to 29–30% of all VSDs. [8] A Sri Lankan study also reported VSDs accounting for 27.5% of all congenital heart diseases. [10]

The clinical presentation of VSD primarily depends on the size of the defect. Large VSDs may remain asymptomatic in the neonatal period, while smaller defects often produce loud murmurs and are thus detected early in infancy. [28] In the present study, the majority of lesions were of small size (58.70%), followed by moderate-sized (32.61%) lesions, and large lesions (>10 mm) were present in 8.70% of cases. In a study conducted by Layangoo et al. (2008), the distribution of small, moderate, and large defects was 62.5%, 15.9%, and 21.6%, respectively. [30]

Management of VSD depends on the defect size and associated complications. Small VSDs often do not require intervention, whereas moderate defects may behave similarly to large ones and may necessitate surgical or catheter-based procedures. [28] Device closure is recommended for selected muscular VSDs. [31]

In this study, echocardiography was the definitive diagnostic tool and was performed in all cases. Other investigations included ECG (82.61%), chest X-ray (76.09%), and cardiac catheterization (8.70%). Kazmi et al. (2009) performed echocardiography as the primary diagnostic tool for detecting ventricular septal defects. All of their patients underwent transthoracic echocardiography, utilizing 2D imag-

ing, continuous wave Doppler, and color Doppler techniques. [8] A study by Chaudhry et al. (2011) reported that the diagnosis was primarily made using echocardiography. The size, number, and exact location of the defect, as well as the magnitude of the shunt, were identified using two-dimensional and Doppler echocardiography. [23]

Regarding complications, pulmonary hypertension was the most frequently observed, affecting 58.70% of participants. Other notable complications included aortic cusp prolapse (23.91%) and aortic regurgitation (17.39%). These findings are consistent with those reported by Kazmi et al. (2009), who observed 76 cases of right coronary cusp (RCC) prolapse among 729 patients with perimembranous VSDs (10.4%). [8] Lue et al. (1981) reported aortic valve prolapse and regurgitation in 11.9% of their VSD patients [20], while Brauner et al. (1995) noted aortic valve prolapse in over 5% of affected children. [32] Ando et al. (1986) found RCC prolapse in 16% of VSD cases [33], and Chiu et al. (2005) observed that the prolapsed cusp in DCSA was always the RCC. In perimembranous outlet VSDs, the non-coronary cusp (NCC) was involved in 16.5% of cases. [22] Somanath et al. (1990) identified RCC prolapse in 48% of their patients with perimembranous VSD, NCC in 41%, and both cusps in 11%. [34]

Detection of RCC prolapse is critical in patients with outlet VSD, as it can lead to permanent aortic regurgitation. [35] Surgical intervention is warranted in patients with clinically significant aortic regurgitation or right ventricular outflow tract (RVOT) obstruction [36], and early repair may prevent progression of aortic insufficiency. [37] Glen et al. (2004) reported infundibular stenosis in 5.8% and aortic valve prolapse in 3.6% of their VSD patients. [38]

These findings highlight the diverse clinical presentations and potential severity of VSD, underscoring the importance of early detection, precise classification, and timely intervention to prevent complications.

5. Conclusion and Recommendations

The study findings highlight that VSD often presents with significant clinical symptoms, including respiratory distress, feeding difficulties, and poor weight gain, although a notable proportion of cases remain asymptomatic. Early diagnosis, based on clinical signs and use of simple investigation tools like X-ray and echocardiography (ECHO), may reduce the life-threatening complications and mortality. Pulmonary hypertension emerged as the most frequent complication, affecting more than half of the participants. Other notable complications included aortic cusp prolapse, aortic regurgitation, right & left ventricular outflow tract obstructions, and infective endocarditis.

Further study with a prospective and longitudinal study design, including a larger sample size, needs to be done to validate the findings of this study.

Abbreviations

CHD	Congenital Heart Disease
VSD	Ventricular Septal Defect
ECHO	Echocardiography
ECG	Electrocardiogram

Author Contributions

Mohammad Moniruzzaman is the sole author. The author read and approved the final manuscript.

Ethical Approval

This study was ethically approved.

Funding

No funding sources.

Conflicts of Interest

The author declares no conflicts of interest.

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