

Research

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Small Ventricular Septal Defect Considered not Requiring Surgical Closure: The Frequency of Developing Complications in Our Center

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ABSTRACT

Objectives: The study was conducted to check the frequency of development of complication in the patients with small Ventricular Septal Defects (VSDs) considered not requiring surgical closure during childhood.

Methods: A descriptive study was conducted on children from January 2015 to December 2015 at Children Hospital and Institute of Child Health, Lahore, Pakistan. The data with isolated VSDs considered too small to require surgery from 1 month to 15 years of age were reviewed. The data was analyzed with SPSS 20 version.

Results: The total of 883 patients of restrictive VSDs considered not to require surgery, 60.6% (n=535) were males and 39.4% (n=348) were females. The significant number of patients i.e. 18.7% (n=166) developed complications. Aortic cusp prolapsed developed in 13.6% i.e. $p \leq 0.05$, 2.3% developed aortic regurgitation secondary to aortic cusp prolapsed. 1.8% developed right ventricular track outflow obstruction (RVOT) i.e. $p \leq 0.05$ and 0.3% of patient developed left ventricular outflow track obstruction (LVOT) i.e. $p \leq 0.05$. Similarly 0.8% patients developed endocarditis. Regarding types of VSD, we found Perimembranous in 65.8%, muscular in 12.6%, Subaortic in 8.3%, doubly committed in 6.0%, Inlet in 5% and outlet in 1.7% of our patients.

Conclusions: Patients with small restrictive Ventricular Septal Defects (VSDs) generally been considered as do not required surgery, the data suggested that a significant percentage of these patients developed complications later in their life i.e., 18.7%.

KEYWORDS: Ventricular septal defect; Aortic cusp prolapsed; Aortic regurgitation; Right ventricular outflow track obstruction; Left ventricular outflow track obstruction.

ABBREVIATIONS: VSD: Ventricular Septal Defect; RVOT: Right Ventricular Outflow; LVOT: Left Ventricular outflow; AR: Aortic Regurgitation.

INTRODUCTION

Isolated ventricular septal defect occurs in approximately 2-6 of every 1000 live births and accounts for more than 15-20% of all congenital heart diseases.¹ Soto et al divided VSD into Perimembranous, Muscular and Doubly committed sub-arterial (DCSA) types.² It had been well accepted for many years that patients with a small ventricular septal defect (VSD) as defined by a left-to-right shunt of <50%, normal pulmonary artery pressure (PAP), and absence of symptoms do not require surgical repair.^{3,4} Operation were postponed on the observation that VSDs frequently close spontaneously.^{5,6} The patients with such small defects are unlikely to

develop pulmonary hypertension and that the clinical outcome was assumed to be good.⁴

The natural history of VSD is also characterized by many complications. Of special interest is prolapse of the aortic valve cusp, which classically occurs with doubly committed subarterial and less commonly with perimembranous outlet type.⁷ Secondary aortic insufficiency, is associated along with prolapse of aortic valve cusps. This complication is observed only in 5% of patients with ventricular septal defect.⁸ Aortic regurgitation occurs due to a poorly supported right coronary cusp combined with the Venturi effect produced by the ventricular septal defect jet, resulting in cusp prolapsed.⁹ Aortic regurgitation is progressive in nature and presence of even mild aortic regurgitation or aortic valve prolapse in the absence of aortic regurgitation is an indication for surgery.¹⁰ Perimembranous outlet VSD are also associated with infundibular hypertrophy, and right ventricular outflow tract obstruction can progress in severity. This also requires surgical intervention.¹¹ Discrete fibrous subaortic stenosis is occasionally associated with a ventricular septal defect. This complication is most often reported with perimembranous ventricular septal defects and can first appear after either spontaneous or surgical closure.¹² Infective endocarditis is rare in children younger than 2 years.⁶

METHODS

Data Collection Procedure

All echocardiography reports were reviewed from hospital record. Patients only having isolated restrictive ventricular septal defect (VSD) were included in the study. Associated complications like aortic valve prolapse and aortic regurgitation, acquired right and left ventricular outflow tract obstruction and infective endocarditis were noted.

Patient Population

The patient population represents the total number of patients visited in the Department of Cardiology for echocardiography in Children Hospital & Institute of Child Health, Lahore, Pakistan from January 2015 to December 2015. A total of 883 patients with small VSDs (male/female 535/348) were selected. All patients fulfilled the following inclusion criteria:

- 1) Surgical closure had not been performed for the following reasons:
 - a. Gradient across VSD more than 50 mm of Hg,
 - b. Normal LVEDD according to age,
 - c. Size of VSD less than 3 mm,
 - d. No PR (normal PAP) and
 - e. Asymptomatic children.
- 2) No additional hemodynamically related heart defects were present.

Echocardiography

All echocardiograms were performed and interpreted at one laboratory by consultant pediatric cardiologist. Standard transthoracic M-mode, two-dimensional and Doppler echocardiography were performed with GE VIVID-7 DIMENSION echo-machine. The VSDs were classified by their location into perimembranous, inlet, outlet, doubly committed (Supracristal), and muscular. A normal left ventricular end-diastolic diameter (LVEDD) according to age. The peak pressure difference between right and left ventricle (LV) was calculated from the continuous-wave Doppler-measured maximum VSD jet velocity using the simplified Bernoulli equation. Systolic right ventricular-to-right atrial pressure difference was calculated from the continuous-wave Doppler-measured peak tricuspid regurgitant velocity with the simplified Bernoulli equation. Systolic PAP was obtained by adding 5 mmHg for the right atrial pressure as long as the gradient did not indicate pulmonary hypertension and tricuspid regurgitation was not hemodynamically relevant. A systolic PAP ≤ 25 mmHg was considered normal.¹³⁻¹⁵ If no adequate Doppler signal of the tricuspid regurgitant flow could be obtained, PAP was calculated by subtracting the left ventricular to right ventricular systolic gradient (LV-RV gradient) from the systemic arterial pressure measured with cuff, provided that no aortic stenosis was present.¹⁵

Statistical Analysis

Appropriate statistical data analysis technique by using SPSS version 20 was applied. Qualitative variables were described by numbers and percentages while quantitative were described as mean and SD (standard deviations). Chi-square test was applied for categorical variables and independent sample t-test was applied for quantitative variables. Five percent level of significance was used.

RESULTS

The 883 patients of restrictive hemodynamically small VSDs considered not to require surgery were entered. There were 60.6 % (n=535) males and 39.4% (n=348) were females (Figure 1). Regarding types of ventricular septal defects (VSD), we found Perimembranous in 65.8%, muscular in 12.6%, Subaortic in 8.3%, doubly committed in 6.0%, inlet in 5% and outlet in 1.7% of patients (Table 1). Complications were predominantly found more in male in case of perimembranous and doubly committed VSD i.e. 67% and 7.1% respectively (Figure 1).

The significant number of patients i.e. 18.7% (n=166) developed complications. Aortic cusp prolapsed was found to be the commonest one i.e. developed in 13.6% (n=120) $p \leq 0.05$ of patients. Second common complication was aortic regurgitation secondary to aortic cusp prolapsed which was found in 2.3% (n=20) of patients. 1.8% (n=16) of patients developed right ventricular track outflow obstruction (RVOTO) $p \leq 0.05$ and 0.3% (n=3) of patient developed left ventricular outflow track obstruction (LVOTO) $p \leq 0.018$. Similarly 0.8% (n=7) patients

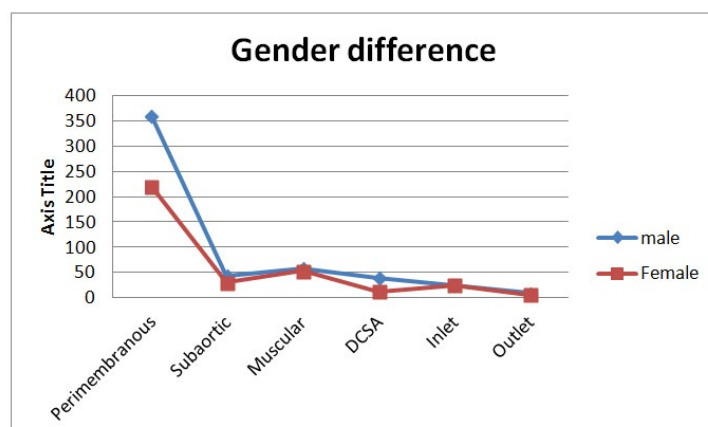


Figure 1: Gender difference with type of VSDs.

VSDs	Total	Percent %
Perimembranous	581	65.8%
Muscular	111	12.6%
Subaortic	73	8.3%
DCSA	53	6.0%
Inlet	50	5.7%
Outlet	15	1.7%
Total	883	100.0%

Table 1: Type of VSD (n=883).

developed echo based endocarditic (Tables 2 and 3).

Regarding complications aortic cusp prolapse was found commonly in perimembranous ventricular septal defect i.e. 11.09% as compared to doubly committed VSD which was 1.2% $p \leq 0.05$. Similarly aortic regurgitation was also commonly found in perimembranous types of ventricular septal defect (Table 4).

Patients were grouped into four groups according to age. Group-1 included patients of 1 day to 1 year, Group-2 included from 1 year to 5 years, Group-3 included from 6 years to 10 years and group-4 included more than 10 years of age.

DISCUSSION

The significant number of patients of restrictive small ventricular septal defect developed complications in our study. We found

it to be 18.7%. Associated complications related with VSD are already known in the literature, these included upto 25%, including infective endocarditis in 11 percent.¹⁶ Another follow-up study revealed that 22% subjects had major, VSD-related complications.¹⁷

The natural history has a wide spectrum, ranging from spontaneous closure to congestive heart failure (CHF) to death in early infancy.⁷ Spontaneous closure frequently occurs in children, usually by age of 2 years. Closure is most frequently observed in muscular defects (80%), followed by perimembranous defects (35-40%).⁵ Outlet ventricular septal defects have a low incidence of spontaneous closure, and inlet ventricular septal defects does not close.¹

Regarding complication in our study aortic cusp prolapsed was found to be the commonest one i.e. developed in 13.6% (n=120) of patients. Similar results were shown in local

Complications of VSD	Number of patients	Percentage %
Aortic cusp prolapse	120	13.6%
Aortic regurgitation	20	2.5%
RVOT obstruction	16	1.8%
Vegetation	07	0.8%
LVOT obstruction	03	0.3%

Table 2: Complications Observed with restricted VSDs (n=166).

Complications	Types of VSD					
	Perimembranous	Muscular	Subaortic	DCSA	Inlet	Outlet
Aortic cusp prolapse	98	0	10	11	0	1
Aortic regurgitation	16	0	1	2	0	0
RVOT obstruction	7	3	5	1	0	0
Vegetation	4	0	2	1	0	0
LVOT obstruction	0	1	2	0	0	0

Table 3: Complications Observed with Types of VSDs (n=166).

	Age				Total
	Day 1 to 1 year	1 year to 5 years	5 years to 10 years	10 to onward	
Subaortic	21	8	6	2	37
	9.3%	5.0%	7.3%	16.7%	7.7%
perimembranes	145	112	58	9	324
	64.4%	70.4%	70.7%	75.0%	67.8%
muscular	34	22	11	0	67
	15.1%	13.8%	13.4%	0.0%	14.0%
outlet	3	2	2	0	7
	1.3%	1.3%	2.4%	0.0%	1.5%
inlet	15	8	2	1	26
	6.7%	5.0%	2.4%	8.3%	5.4%
DCSA	7	7	3	0	17
	3.1%	4.4%	3.7%	0.0%	3.6%
	225	159	82	12	478
	100.0%	100.0%	100.0%	100.0%	100.0%

Table 4: Types of VSDs according to age groups.

studies by Uzma Kazmi et al¹⁸ and Masood Sadiq et al¹⁹ conducted in Lahore, Pakistan. This frequency is in keeping with other studies. Lue et al²⁰ found aortic cusp prolapse and aortic regurgitation in 11.9% of their patients with VSD. Brauner et al²¹ found aortic cusp prolapse in over 5% of children with VSD.

Classically Doubly committed sub-arterial type VSD is associated with progressive development of aortic cusp prolapse and aortic regurgitation.⁷ Contrary to this; our study showed that incidence of aortic cusp prolapse and aortic regurgitation with doubly committed VSD was 1.2%.

Development of aortic regurgitation (AR) or presence of AR at late follow-up has been reported within the wide range of 2% to 20% in previous studies.¹⁰ Patients with obvious prolapse of an aortic cusp into the VSD have the highest risk of developing progressive aortic regurgitation. We found 2.3% of our patients developed aortic regurgitation which was more common in case of perimembranous type of VSD.

Right ventricular outflow obstruction occurs in 3 to 7% patients and is due to hypertrophy of anomalous muscle bundles.¹¹ We found 1.8% of our patients with development of RVOT obstruction. Glenn et al²² found that 5.8% patients of

VSD developed infundibular stenosis. One study from Multan showed 1.6% of cases of their patients developed RVOT obstruction.

Discrete fibrous subaortic stenosis most often reported with perimembranous ventricular septal defects and can first appear after either spontaneous or surgical closure.¹² 0.3% of our patients developed LOVT obstruction.

The occurrence of endocarditis has been described as the major risk in small VSDs.⁶ Again, the incidence of this complication varies widely in previous reports, ranging from 1% to 15%.^{6,7} Variations reflect the difficulty of estimating the true incidence of this complication.⁵ Shah et al²³ estimated the risk of a 15-year-old with a VSD developing bacterial endocarditis by age 70 to be 11.5%. The highest percentage of 15% (5.7 per 1,000 patient-years) was reported by Otterstad et al.¹⁷ On the contrary, we found 0.8% echo based endocarditis in our patients.

LIMITATION

Limitation of this study is that it did not reflect the complications in total population as it was limited to one hospital attendance. Also, excluded were children not reaching a tertiary care centre

or admitted to critical care or ward, yet the results are comparable with other local and international studies. As it was a retrospective study, it was difficult to control bias and confounders. Also, we had to rely on the available written record. Results are, at best, hypothesis-generating.

CONCLUSION

Although patients with small VSDs have generally been considered not to require surgery, data suggests that a significant percentage of these patients developed complications later in their life i.e., 18.7%.

GRANT SUPPORT AND FINANCIAL DISCLOSURES: None.

CONFLICTS OF INTEREST: None.

ETHICS COMMITTEE APPROVAL

The study was approved by the Institutional Review Board and Ethical Committee of The Children Hospital and Institute of Child Health, Lahore, Pakistan and written informed consent was taken from both the parents of the patients.

REFERENCES

1. Sadiq M, Roshan B, Latif F, Bashir I, Sheikh SA, Khan A. Pattern of pediatric heart disease in Pakistan. *J Col Physicians Surg Pak*. 2002; 12(3): 149-153. Web site. https://inis.iaea.org/search/search.aspx?orig_q=RN:34015814. Accessed March 19, 2016.
2. Soto B, Becker AE, Moulaert AJ, Lie JT, Anderson RH. Classification of ventricular septal defects. *Br Heart J*. 1980; 43: 332-343. doi: [10.1136/hrt.43.3.332](https://doi.org/10.1136/hrt.43.3.332)
3. Bloomfield DK. The natural history of ventricular septal defects in patients surviving infancy. *Circulation*. 1964; 29: 914-955. doi: [10.1161/01.CIR.29.6.914](https://doi.org/10.1161/01.CIR.29.6.914)
4. Dickinson DF, Arnold R, Wilkinson JL. Ventricular septal defect in children born in Liverpool 1960 to 1969. Evaluation of natural course and surgical implications in an unselected population. *Br Heart J*. 1981; 46(1): 47-54. doi: [10.1136/hrt.46.1.47](https://doi.org/10.1136/hrt.46.1.47)
5. Campbell M. Natural history of ventricular septal defect. *Br Heart J*. 1971; 33(2): 246-257. Web site. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC487172/>. Accessed March 19, 2016.
6. Alpert BS, Cook DH, Varghese PJ, Rowe RD. Spontaneous closure of small ventricular septal defects: Ten-year follow-up. *Pediatrics*. 1979; 63(2): 204-206. Web site. pediatrics.aappublications.org/content/63/2/204?variant=long&sso=1&sso_redirect_count=1&nftstatus=401&nftoken=00000000-0000-0000-0000-000000000000&nftstatusdescription=ERROR%3a+No+local+token. Accessed March 19, 2016.
7. Keith JD, Rose V, Collins G, Kidd BS. Ventricular septal defect: Incidence, morbidity, and mortality in various age groups. *Br Heart J*. 1971; 33: 81-87. Web site. <http://heart.bmj.com/content/33/supplement/81.long>. Accessed March 19, 2016.
8. Corone P, Doyon F, Gaudeau S, et al. Natural history of ventricular septal defect. A study involving 790 cases. *Circulation*. 1977; 55(6): 908-915. Web site. <http://circ.ahajournals.org/content/55/6/908.full.pdf>. Accessed March 19, 2016.
9. Chaudhry TA, Younas M, Baig A. Ventricular Septal defect and associated complications. *JPMa*. 2011; 61(10): 1001-1004. Web site. http://jpma.org.pk/full_article_text.php?article_id=3021. Accessed March 19, 2016
10. Kobayashi J, Koike K, Senzaki H, et al. Correlation of anatomic and hemodynamic features with aortic valve leaflet deformity in doubly committed subarterial ventricular septal defect. *Heart Vessels*. 1999; 14(5): 240-245. doi: [10.1007/BF01747853](https://doi.org/10.1007/BF01747853)
11. Tomita H, Arakaki Y, Ono Y, Yamada O, Yagihara T, Echigo S. Imbalance of cusp width and aortic regurgitation associated with aortic cusp prolapse in ventricular septal defect. *Jpn Circ J*. 2001; 65(6): 500-504. doi: [10.1253/jcj.65.500](https://doi.org/10.1253/jcj.65.500)
12. Chiu SN, Wang JK, Lin MT, et al. Aortic valve prolapse associated with outlet-type ventricular septal defect. *Ann Thorac Surg*. 2005; 79(4): 1366-1371. doi: [10.1016/j.athoracsurg.2004.10.012](https://doi.org/10.1016/j.athoracsurg.2004.10.012)
13. Pongiglione G, Freedom RM, Cook D, Rowe RD. Mechanism of acquired right ventricular outflow tract obstruction in patients with ventricular septal defect: an angiocardiology study. *Am J Cardiol*. 1982; 50(4): 776-780. doi: [10.1016/0002-9149\(82\)91233-4](https://doi.org/10.1016/0002-9149(82)91233-4)
14. Zielinsky P, Rossi M, Haertel JC. Subaortic fibrous ridge and ventricular septal defect: role of septal malalignment. *Circulation*. 1987; 75(6): 1124-1129. doi: [10.1161/01.CIR.75.6.1124](https://doi.org/10.1161/01.CIR.75.6.1124)
15. Gabriel HM, Heger M, Innerhofer P, et al. Long-term outcome of patients with ventricular septal defect considered not to require surgical closure during childhood. *J Am Coll Cardiol*. 2002; 39(6): 1066-1071. doi: [10.1016/S0735-1097\(02\)01706-0](https://doi.org/10.1016/S0735-1097(02)01706-0)
16. Mehta AV, Goenka S, Chidambaram B, Hamati F. Natural history of isolated ventricular septal defect in the first five years of life. *Am J Cardiol*. 2000; 93(4): 136-138. Web site. <http://europepmc.org/abstract/med/10754804>. Accessed March 19, 2016.
17. Otterstad JE, Erikssen J, Michelsen S, Nitter-Hauge S. Long-term follow-up in isolated ventricular septal defect considered too small to warrant operation. *Circulation*. 1990; 228(4): 305-309. doi: [10.1111/j.1365-2796.1990.tb00237.x](https://doi.org/10.1111/j.1365-2796.1990.tb00237.x)
18. Kazmi U, Sadiq M, Hyder SN. Pattern of ventricular septal defects and associated complications. *J Coll Physicians Surg*

Pak. 2009; 19(6): 342-345. Web site. <http://www.jcpsp.pk/archive/2009/Jun2009/04.pdf>. Accessed March 19, 2016.

19. Sadiq M, Roshan B, Khan A, Latif F, Bashir I, Sheikh SA. Pediatric heart disease in Pakistan - epidemiological data on 6620 patients. *Pak Paed J.* 2001; 25: 63-70.

20. Lue HC, Sung TC, Hou SH, et al. Ventricular septal defect in Chinese with aortic valve prolapse and aortic regurgitation. *Heart Vessels.* 1981; 2(2): 111-116. doi: [10.1007/BF02059965](https://doi.org/10.1007/BF02059965)

21. Brauner R, Birk E, Blieden L, Sahar G, Vidne BA. Surgical management of ventricular septal defect with aortic valve prolapse: clinical considerations and results. *Eur J Cardiothoracic Surg.* 1995; 9(6): 315-319. doi: [10.1016/S1010-7940\(05\)80189-7](https://doi.org/10.1016/S1010-7940(05)80189-7)

22. Glen S, Burns J, Bloomfield P. Prevalence and development of additional cardiac abnormalities in 1448 patients with congenital ventricular septal defects. *Heart.* 2004; 90(11): 1321-1325. doi: [10.1136/hrt.2003.025007](https://doi.org/10.1136/hrt.2003.025007)

23. Shah P, Singh WS, Rose V, et al. Incidence of bacterial endocarditis in ventricular septal defect. *Circulation.* 1966; 34: 127-131. doi: [10.1161/01.CIR.34.1.127](https://doi.org/10.1161/01.CIR.34.1.127)