

Ventricular Septal Defect

Albert Koja¹, Xhentila Doka²

^{1,2}Department of Pediatrics, University Hospital Center "Mother Teresa", Tirana, Albania

Abstract: *Septal defects (ASD, VSD, Atrioventricular defects) account for 35-40% of the congenital heart defects. Ventricular septal defect (VSD) is one of the most common congenital heart lesions. It occurs in almost 50 percent of all patients with congenital heart disease (CHD), with a reported prevalence of 4 per 1000 live births. [1] There are three main anatomic components of interventricular septum and VSD may occur at various locations in any of the three components (the septum of AV canal, the muscular septum, the parietal band or distal conal septum). [2] The aim of the study is to compare data such as right heart enlargement, pulmonary hyperflow persistence, rhythm disturbances and FS values in pre and post operatory patients. This is a prospective study performed in the General Pediatric Service, the Department of Cardiology for the period 2010-2014. A total of 66 cases were diagnosed with VSD from 2010 to 2014. By comparing all the variables, we conclude that all the estimated parameters (enlargement of right cavities, pulmonary hyperflow, FS contractility) are significantly improved after the intervention.*

Keywords: ventricular septal defect, congenital heart disease (CHD), pulmonary artery pressure, pulmonary hypertensive vascular disease, enlargement of right cavities, pulmonary hyperflow

1. Introduction

Clinical presentation

Most patients with VSD present in the neonatal period. However, the clinical presentation varies depending upon the size of the defect and may range from an isolated murmur that is detected incidentally at a health supervision visit to severe heart failure. [3] Infants with small, restrictive VSDs usually remain asymptomatic. In contrast, infants with moderate to large VSDs usually manifest signs of heart failure by three to four weeks of age. [1] These infants with moderate or large VSDs may present with tachypnea, increased work of breathing, poor weight gain or failure to thrive, and diaphoresis particularly with feeding at approximately two to eight weeks of age. [4]

Diagnosis

Anamnesis and physical examination of any case that occurs with complaints such as fatigue, weight loss or signs of heart failure with systolic murmur, gives a suspicion of septal defect of the heart. The diagnosis is made by echocardiography which determines the defect size and position, but also left-to-right shunt. Other diagnostic examinations are: chest X-ray, electrocardiogram, cardiac catheterization and in some cases CT scan or MRI. [2]

Management

Regular follow-up should occur throughout the first year of life for all infants with moderate to large VSDs, even if symptoms are lacking. Patients with pulmonary artery pressure PAP > 50 percent of systemic arterial pressure at risk of developing pulmonary hypertensive vascular disease (PHVD) and they should be referred for surgical closure by six months of age and no later than one year of age. For symptomatic patients, medical intervention may postpone and possibly avoid the need for surgical correction. Management of symptomatic patients includes nutritional support and pharmacologic treatment of heart failure (eg, diuretic therapy). Measures to prevent respiratory tract infections are also an important component of management. [2] Decisions regarding closure of VSDs and the type of

procedure are made on a case-by-case basis. The goal of VSD closure is to alleviate or prevent heart failure symptoms and to intervene before irreversible PHVD develops, which may occur as early as six months in high risk patients. Surgery is not typically required for asymptomatic patients with residual shunts in the absence of left ventricular or elevated PAP.

For most children who require a VSD closure intervention, direct patch closure under cardiopulmonary bypass is the procedure of choice. [5] The approach (eg, transatrial, transaortic, right ventriculotomy, apical ventriculotomy) depends upon the type and location of the defect, as well as the preference of the surgeon. [6]

2. Material and Methods

This is a prospective study performed in the General Pediatric Service, the Department of Cardiology for the period 2010-2014.

A total of 66 cases were diagnosed with VSD from 2010 to 2014. The number of cases distributed by gender in VSD cases is almost the same, with a slight predominance in males (53% vs 47% females) but without a statistical difference ($p = 0.135$).

With regard to the age of diagnosis, the average age is 1.7 months, the youngest child is 1 month and the oldest is 2 years old. A large part of the cases have been diagnosed at birth (75% of cases). The average age of surgical intervention is 18.5 months, the youngest child is 5 months old and the oldest 11 years old. 3% of cases have VSD associated with other anomalies (Down syndrome).

Clinically, these patients had: all cases had cardiac murmurs, 95% recurrent pulmonary infections, 91% diaphoresis, 67% dyspnea, 64% with poor weight gain, 15% with rhythm disorders, 6% asymptomatic.

All the cases had membranous defects, 2 cases had also ASD,

and 1 case had besides membranous defect and muscular defect. The cases with restrictive VSD were 5 and the average size of the VSD resulted 7.5 mm

Echocardiographically 100% of cases had pulmonary hyperflow and 20% had enlargement of right cavities. Correction of the defect in this study was done surgically by direct patch closure. 27% of cases had postoperative complications such as: cardiac rhythm disturbances, pericarditis and death (one case).

Statistical analysis

All the collected data was downloaded to the computer in Microsoft Excel, where they were then expounded in Statistical Package for Social Sciences (SPSS) 20.0, a program in which all statistical analysis was performed. The statistical procedures and techniques applied in the analysis of the data of this study are described in detail below:

- For all categorical variables (nominal including binary / dichotomous and ordinal), the absolute numbers and percentages were calculated.
- For all numeric variables, when the data subject to normal distribution was calculated the arithmetic averages \pm respective standard deviations.
- Differences between groups for discrete variables, non-parametric data, were performed by Hi-square test.
- The presentation of the data was realized through tables and graphs of different types. Significant values were $p \leq 0.05$.

3. Results and Discussion

After surgical correction of ventricular defect, data such as right heart enlargement, pulmonary hyperflow persistence, rhythm disturbances and FS values were compared as pre and post operator variables. The results are: only 6% of cases had an enlargement of right cavities one month after correcting the defect. All cases had no pulmonary hyperflow after correction, and only 39% of cases had systolic murmur one month after the defect correction.

The comparison of the pre and post interventions in VSD cases (right heart enlargement) is shown in Table 1:

Table 1: Right heart enlargement

Right heart enlargement	pre-op	post-op	P values
yes	13(19.7)	4 (6.1)	P <0.013
no	53(80.3)	62(93.9)	
Total	66(100.0)	66(100.0)	

Wilcoxon Test=5.2

Wilcoxon test shows that there is a statistically significant difference in right heart enlargement before and after surgical intervention ($p = <0.001$). After surgery, the number of patients with the enlargement of right cavities (19.7% vs 6.1%) decreased significantly.

Comparison of echocardiographic data pre and post interventions in VSD cases (pulmonary hyperflow) is presented in Table 2.

Table 2: Pulmonary hyperflow

Pulmonary hyperflow	pre-op	post-op	P values
yes	66 (100.0)	0 (0.0)	P < 0.001
no	0 (0.0)	66 (100.0)	
Total	66 (100.0)	66 (100.0)	

Wilcoxon test=8.12

The Wilcoxon test shows that there is a statistically significant difference in pulmonary hyperflow before and after surgical intervention ($p < 0.001$). After the intervention there is no patient with pulmonary hyperflow (100.0% vs. 0.0%).

The comparison of the number of cases with VSD with pre and postoperative rhythm disorder is presented in Table 3.

Table 3: Rhythm disturbances

Rhythm disturbances	pre-op	post-op	P values
yes	10 (15.2)	12 (18.2)	P=0.637
no	56 (84.8)	54 (81.8)	
Total	66 (100.0)	66 (100.0)	

Wilcoxon test=0.47

The Wilcoxon test shows that there is no statistically significant difference in the rhythm disturbances before and after surgery ($p = 0.637$).

Comparison of pre and post operator FS values is shown in Table 4:

Table 4: Fractional shortening (FS)

FS (%)	pre-op	post-op	P value
	32.36 \pm 1.93	32.72 \pm 1.73	P< 0.009

T-Test=2.7

Through the student's test for two paired samples it is seen that there is a statistically significant difference in FS contractility before and after surgical intervention ($p < 0.001$). After the intervention there is a tendency to increase the average value of the FS contractility, compared to the pre-intervention value (indirectly, 32.36 \pm 1.93 vs. 32.72 \pm 1.73).

4. Conclusion

By comparing all the variables, we conclude that all the estimated parameters (enlargement of right cavities, pulmonary hyperflow, FS contractility) are significantly improved after the intervention.

References

- [1] Rudolph AM. Ventricular Septal Defect. In: Congenital Diseases of the Heart: Clinica-Physiological.
- [2] Kliegman RM, Stanton BF, Schor NF, Geme JW, Behrman RE. *pediatria di nelson* 19th ed.2013;1627-28.
- [3] Gumbiner CH, Takao A. Ventricular septal defect. In: *The Science and Practice of Pediatric Cardiology*, 2nd ed, Garson A, Bricker JT, Fisher DJ, Neish SR (Eds), Williams & Wikins, Baltimore 1998. P. 1119.

- [4] Fyler DC, Rudolph AM, Wittenborg MH, Nadas AS. In: ventricular Septal Defect in infants and children; a correlation of clinical, physiologic, and autopsy data.
- [5] Bol-Raap G, Weerheim J, Kappetein AP, Witsenburg M, Bogers AJ. In: Follow-up after surgical closure of congenital ventricular septal defect.
- [6] Lincoln C, Jamieson S, Joseph M, Shinebourne E, Anderson RH. In: transatrial repair of ventricular septal defects with reference to their anatomic classification.

Author Profile



Albert Koja, Pediatrician, Division of General Pediatrics, Pediatric Rheumatology and Cardiology, Department of Pediatrics, University Hospital Center “Mother Teresa”, Tirana, Albania



Xhentila Doka, Pediatric Resident, Department of Pediatrics, University Hospital Center “Mother Teresa”, Tirana, Albania

