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Recommended Citation

Ashfaq, A., Zia, H., Amanullah, M. (2010). Is early correction of congenital ventricular septal defect a better option in a developing country. *Journal of the Pakistan Medical Association*, 60(4), 324-7.

Available at: http://ecommons.aku.edu/pakistan_fhs_mc_surg_cardiothoracic/19

Is early correction of congenital ventricular septal defect a better option in a developing country?

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Abstract

Objective: To assess and evaluate the effectiveness of early ventricular septal defect (VSD) repair in a developing country by comparing the outcomes in two age groups.

Methods: A total of 39 patients underwent VSD repair at a tertiary care hospital in Karachi over a period of 2.5 years. Patients were divided into Group 1 (< 2 years) and Group 2 (2-18 yrs). Perioperative and postoperative variables were reviewed retrospectively for each patient and compared with the development of complications and the effect on postoperative recovery times. The data was analyzed using Mann Whitney test for continuous variables and Fischer's exact test for categorical variables.

Results: Mean age at surgery was 10.1 ± 7.0 months and 108.5 ± 54.0 months for both groups, respectively. Two patients died. Procedure time was not affected by decreasing age (p=0.622) but Cardio pulmonary Bypass duration (p=0.040) and Aortic Cross Clamp time (p=0.063) were significantly affected. No associated significance was obtained when hospital stay (p=0.085) was observed. Increased duration of ventilation (p=0.000) and length of ICU stay (p=0.003) were highly significant for the younger age group. Presence of residual VSD (p=0.025) was also significantly affected by lower age. There was no significant difference in the number of patients with complications (p=1.000) among the 2 groups.

Conclusion: Age does not increase morbidity rates after VSD repair although postoperative recovery times are increased with decreasing age. Hence, there is no justification in delaying VSD repair (JPMA 60:324; 2010).

Introduction

Over the years, there has been a shift towards operating children with ventricular septal defects (VSD) earlier. This trend may be explained mainly by two observations: Firstly, pediatric cardiologists are referring patients to surgeons for repair of symptomatic VSDs as early as possible and secondly, an increase in the awareness of the general population, has led to a greater and earlier influx of patients compared to the past.

Ventricular septal defect (VSD) is one of the most common congenital heart defect, accounting for 25% to 30% of all children born with structural heart disease.¹ Large VSDs cause significant morbidity and mortality in infants because of congestive heart failure and recurrent lung infections. If left untreated, large VSDs typically result in significant failure to thrive, which often does not respond to nutritional supplementation alone.²⁻⁴ Delay in closure of large VSDs during infancy is also associated with a risk for development of pulmonary vascular obstructive disease.⁵ Usually, infants can be haemodynamically stable if the surgery is performed before the age of 6 to 9 months.⁵⁻⁷ Studies from developed countries have demonstrated that adverse preoperative factors do not significantly affect the postoperative outcome of infants undergoing VSD closure.⁸⁻¹⁴ It is also known that earlier

intervention prevents these patients from heart failure and helps to maintain proper development and growth without increased pulmonary resistance.

However, in developing countries, there is a clear difference with respect to the patient population. Usually, patients with VSDs present very late, often several months to years after they start developing symptoms. This exposes them preoperatively to a high risk from a cardiac point of view and also predisposes them to recurrent lung infections. Moreover, environmental and nutritional factors expedite the development of such risks as most of these patients come from under-privileged classes with poor hygiene and parallel malnutrition. Concern about the postoperative complications is also one of the major hindrances to early surgery. However, the trend is changing as the public is getting more aware of the developments and technologies of health sector as a result of better health awareness programmes. This has led to an increase in the number of infants with VSDs being referred for surgery.

To address this debate and evaluate the effectiveness of early VSD repair, we compared the outcomes of VSD closure in different age groups.

Methods

The study was conducted retrospectively at the Aga

Khan University Hospital, a tertiary care hospital in Karachi, Pakistan, for a period of 2.5 years, from July 2006 to December 2008. We reviewed preoperative, operative and postoperative data from the medical records of the patients, and comparisons were made thereafter to observe the outcomes. A total of 39 patients underwent VSD closures that were divided into two groups based on their ages: Group 1 (n = 21, <2yrs of age) and Group 2 (n = 18, 2-18 yrs of age). Patients with single as well as multiple VSDs were included, as were infants with associated Atrial Septal Defect (ASD), Patent Ductus Arteriosus (PDA), Aortic Regurgitation (AR), Double Outlet Right Ventricle (DORV) and Coarctation of Aorta (CoA). Patients with atrioventricular canal defects, Tetralogy of Fallot and other concomitant defects were excluded from the analyses.

The following variables were collected from each patient's medical record: patient's first and last name, medical record number (unique to each patient in the AKUH clinical database), date of birth, sex, height and weight.

The pre-operative echo data which included type of VSD, any concomitant defects and the size of VSD were obtained.

Hospital data included date of surgery and date of discharge (to calculate the length of hospital stay), previous pulmonary artery banding, use of any medications, and evaluation with right heart catheterization (RHC) and comparison with the echocardiogram.

Operative variables included duration of surgery (time from incision to time of dressing), Cardiopulmonary bypass (CPB) time and aortic cross clamp (ACx) time were recorded where ACx is inclusive of CPB time. Minimum temperature achieved during surgery, type of VSD closure and the approach used by the surgeon were also among the data obtained.

Post-operative data included the presence of any residual VSD/s (through a post-op echo), the type of residual VSD, any inotropic support required by the patient, duration of ventilation, length of ICU stay and any complications that occurred postoperatively.

All data for each patient was collected by 2 independent investigators. Upon completion, both the investigators randomly selected 1 in 10 medical record files and quality controlled them by verifying the information a second time.

The data was not normally distributed and was analyzed for group differences using nonparametric methods. The data is presented as arithmetic mean \pm standard deviation. The chosen p value was two tailed, and a p value of < 0.05 was considered significant. Continuous variables such as duration of surgery, CPB time etc were analyzed using Mann Whitney

test and categorical variables such as presence of complications, presence of residual VSDs, etc were analyzed using Fisher's exact test (equivalent of chi-square for non-normally distributed data).

Results

The results are summarized in Table-1. The mean age at the time of surgery for group 1 was 10.1 ± 7.0 months and for group 2 was 108.5 ± 54.0 months. VSDs were categorized as membranous/perimembranous (76.9%), inlet type (5.1%), outlet type (0) and muscular (17.9%). None of the patients had a combination of these VSDs. The most common associated defect was PDA (50%), second to which was ASD (35.9%). The size of VSD was 8.6 ± 2.3 mm and 9.4 ± 5.3 mm for both groups, respectively which was not associated with decreasing age ($p=0.691$).

Operative data included CPB, ACx and surgery times. Duration of surgery was not affected by increasing age

Table-1: Summary of the variables.

	Group I* n = 21	Group II n = 18	p value+
Preoperative variables			
Height (cm)	69.5 \pm 26.4++	127 \pm 25.2	
Weight (kg)	6.6 \pm 4.0	27.2 \pm 13.6	
Age (months)	10.1 \pm 7.0	108.5 \pm 54.0	
Size of VSD (mm)	8.6 \pm 2.3	9.4 \pm 5.3	0.691
Operative variables+++			
Duration of surgery (min)	234.5 \pm 72.4	234.9 \pm 58.9	0.622
CPB time (min)	121.9 \pm 37.2	99.6 \pm 35.2	0.040
Aortic cross clamp time (min)	69.4 \pm 26.1	54.3 \pm 22.0	0.063
Postoperative variables			
Duration of ventilation (hr)	65.8 \pm 32.0	14.0 \pm 33.4	0.000
Length of ICU stay (hr)	136.6 \pm 145.9	63.2 \pm 33.0	0.003
Length of Hospital stay (days)	7.3 \pm 5.6	5.6 \pm 2.5	0.085
Postoperative results			
Complications (%)	66.7 (14)#	66.7 (12)	1.000
Residual VSDs (%)	66.7 (14)	27.8 (5)	0.025

*Age groups

+p<0.05 is considered significant

++A.M. \pm S.D. (95% CI)

+++Anaesthesia time not included

(n)

($p=0.622$) but CPB ($p=0.040$) and ACx time ($p=0.063$) were significantly affected by decreasing age. There was a tendency for younger age group to have a longer CPB and ACx times, as shown in Table-1. Postoperative data included duration of ventilation, length of ICU and hospital stay. No associated significance was obtained when looked at the hospital stay ($p=0.085$), although there was increase in the duration of ventilation ($p=0.000$) and the length of ICU stay ($p=0.003$) with increasing age which were very highly significant.

Other postoperative variables included the presence of residual VSD and complications. The presence of residual

Table-2: Frequency of all complications observed.

Complications	Group I	Group II	Total
SVT	0	0	0
Atrial flutter	0	0	0
Junctional rhythm	0	2	2
Bradycardia	5	0	5
Blood loss requiring transfusion	4	1	5
Transfusion without blood loss	3	1	4
Wound infection	2	0	2
Haemodynamics			
Hypotension	4	1	5
Metabolic acidosis	1	0	1
Cardiac arrest	2	1	3
Fever > 38°C	3	6	9
Multiorgan failure	1	0	1
Readmission (within 30 days)	1	2	3
Post op sepsis	1	0	1
Pneumonia	3	1	4
Reintubation	5	1	6
Chylothorax	0	0	0
Pleural effusion	4	1	5
Pericardial effusion	2	2	4
Death	2	0	2
Total	43	19	62

Table-3: Detail of the pattern of the complications observed.

	Group I	Group II	Total
n > 1*	9	8	17+
n = 1++	5	4	9+++
n = 0#	7	6	13**
GC***	14	12	
Total****	21	18	39

- * No. of patients with more than one complication
- + Total no. of patients with more than one complication
- ++ No. of patients with only one complication
- +++ Total no. of patients with only one complication
- # No. of patients with no complications
- ** Total no. of patients with no complications
- *** Total no. of complications in each group
- **** Total no. of patients in each group

VSD (p=0.025) was significantly decreased by increasing age (more older patients with residual VSD). On postoperative echocardiography, all those patients who had residual VSDs (n= 19) were found to be of residual-patch margin type and none had a residual VSD ≥ 2 mm in size. None of the patients required reoperation for residual VSD/s in the early post-operative period. There were 62 adverse events in 26 patients including postoperative fever $>38^{\circ}\text{C}$ (n=9) which was managed effectively, readmission within 30 days of surgery (n=3), re-intubation (n=6) and cardiac arrest (n=3). In the case of cardiac arrest, patients were revived after defibrillation except one who contributed towards mortality as well. There were 2 mortalities, one male and one female, of ages 5 months each with weights 3.7 kg and 4.1 kg, respectively. Both of these patients were severely ill preoperatively and never recovered, ultimately dying secondary to multi-organ failure.

There were no patients with heart block requiring temporary or permanent pacing. Few patients developed pleural effusion requiring drainage (n=5) and pericardial effusion requiring drainage (n=4) who developed no further complications. There was no case of chylothorax or arrhythmias. Even though the total number of complications were significantly different in both the groups; Group I (n=43) and Group II (n=19), as shown in Table-2; there was no significant difference in the number of patients who encountered complications (p=1.000) among the 2 groups. In Group I and Group II there were 13 out of 21 and 11 out of 18 patients who had complications, respectively. This was possible as some of the patients had more than one complication, Group I (n=9) and Group II (n=6). On analysis, there was no significant difference (p=0.431) between the groups with respect to the presence of more than one complication. This is explained, in detail, in Table-3.

Discussion

There is little published information from the developing world to assess and evaluate the effectiveness of early VSD repair. The purpose of this study was to compare the outcomes of VSD repair in infants with non-infants, primarily focusing on the development of complications postoperatively and postoperative care.

The preoperative characteristics of our study sample clearly reflect the kind of patient population we encounter. Almost half of the study sample in each category proved the later influx of VSD patients. Even in Group 1, the mean age of the patients who presented with VSDs was 10.1 ± 7.0 months, which is late when looked at the recommended timeline. Usually, when children reach 6 months of age with a large, single VSD, elective surgery is warranted.^{5,15,16} The indications for surgical closure of a VSD, uncomplicated by other congenital cardiac defects, depend on the size of the defect and its haemodynamic consequences.¹⁷ In the absence of significant symptoms, surgical closure can be delayed because pulmonary vascular obstructive disease rarely develops ≥ 2 years.^{15,17} Nevertheless, there are many exceptions to this rule, and it is difficult to predict which children will develop an early and accelerated form of pulmonary vascular disease.¹⁵

Occasionally, these children will suffer from severe, intractable congestive heart failure despite aggressive in-hospital medical treatment and require early surgical intervention within the first or second month of life.¹⁶ Children become increasingly tachypnoeic, particularly related to feeding, and fail to gain weight. Left untreated, they are likely to fail to thrive and progressively fall off the growth curve.¹⁵ These children must be referred for early surgery as they become progressively symptomatic within the first 6 months. And as long as the repair is done before the

development of increased pulmonary vascular resistance, the optimal time of repair is unclear.¹⁴

Looking at the outcomes of our study, although there was no significant difference in the number of patients who had complications among the two groups, the total number of complications was significantly different. This is expected as younger children are more likely to report with increasing complications. Children of increasing age (2-18 years), if they survive are likely to develop physiological changes that suit them, hence they are expected to report with less complications compared to infants. However, our mortality rate was (5.1%) which is much higher than the rates reported recently from the developed nations (<1%).^{5,14,15,18} Studies from developed countries have shown that in term infants, young age is not a risk factor for adverse postoperative outcomes after surgical closure of large VSDs.⁸⁻¹⁴ However, in our study, there is clearly a difference in the postoperative recovery times (duration of mechanical ventilation and ICU stay) and a higher rate of infectious complications, assessed by the development of fever, pneumonia and wound infections. It can also be proposed that the slower recovery of such patients is, in part, from longer durations of CPB. Earlier studies done from the developing world¹⁹ also report similar postoperative recovery times, although their rate of mortality was higher (6%) than reported by us.

In conclusion, we believe that once infants with symptomatic VSDs reach 6 months of age, early surgical intervention should be done. Our data clearly shows that earlier intervention can be undertaken safely and effectively in smaller children without adversely affecting outcomes. Although both the mortalities were from the infant group, their condition was already severe, and a larger study sample could relate better to this issue. Performing surgery earlier can avoid unnecessary aggressive medical management with increased diuretics, high caloric feeds, or even risk of developing pulmonary vascular disease. More studies should be undertaken to point out the exact correlation between peri-operative variables and the development of complications; and assess whether these factors play any role in decreasing the immediate as well as long term complication rate of individuals.

References

1. Bernstein D. Epidemiology of congenital heart disease. In: Behrman RE, Kleigman RM, Arvin AM, editors. Nelson textbook of pediatrics. 15th ed. Philadelphia (PA): WB Saunders Co, 1996; pp 1286-7.
2. Ackerman IL, Karn CA, Denne SC, Ensing GJ, Leitch CA. Total but not resting energy expenditure is increased in infants with ventricular septal defects. *Pediatrics* 1998; 102: 1172-7.
3. Granzotti JA, Falha SL, Nunes MD. Nutritional index in heart diseases in childhood. *Arq Bras Cardio* 1990; 55: 371-3.
4. Salzer HR, Haschke F, Wimmer M, Heil M, Schilling R. Growth and nutritional intake of infants with congenital heart disease. *Pediatr Cardiol* 1989; 10: 17-23.
5. Rabinovitch M, Keane JF, Norwood WI, Castaneda AR, Reid L. Vascular structures in lung tissue obtained at biopsy correlated with pulmonary hemodynamic findings after repair of congenital heart defects. *Circulation* 1984; 69: 655-67.
6. Cartmill TB, DuShane JW, McGoon DC, Kirklin JW. Results of repair of ventricular septal defect. *J Thoracic Cardiovasc Surg* 1966; 52: 486-501.
7. Jarmakani JM, Graham TP Jr, Canent RV Jr, Capp MP. The effect of corrective surgery on left heart volume and mass in children with ventricular septal defect. *Am J Cardiol* 1971; 27: 254-8.
8. Bastos P, Nascimento R, de Sousa AR, Pinho P, Cunha D, Gomes MR. Surgical treatment of ventricular septal defect in the first year of life. *Rev Port Cardiol* 1992; 11: 339-45.
9. Cabrera Dyro A, Martinez Corrales P, Llorenta Urcullo A, Aramburu Arriaga N, Rodrigo Carbonero D, Alcibar Villa J, et al. Surgical correction of symptomatic ventricular septal defects in patients less than 6 months of age. *An Esp Pediatr* 1999; 51: 353-6.
10. Kuo SM, Kang PL, Lyu JJ, Cheng KK, Heih KS, Meng CC. Surgical repair of ventricular septal defect without ventriculotomy in the first 12 months of life. *J Formos Med Assoc* 1992; 91: 400-4.
11. Hardin JT, Muskett AD, Canter CE, Martin TC, Spray TL. Primary surgical closure of large ventricular septal defect in small infants. *Ann Thorac Surg* 1992; 53: 397-401.
12. Rizzoli G, Blackstone EH, Kirklin JW, Pacifico AD, Barger LM Jr. Incremental risk factors in hospital mortality after repair of ventricular septal defect. *J Thorac Cardiovasc Surg* 1980; 80: 494-505.
13. Rizzoli G, Rubino M, Mazzucco A, Rocco F, Bellini P, Brumana T, et al. Progress in the surgical treatment of ventricular septal defect: an analysis of a twelve years experience. *Thorac Cardiovasc Surg* 1983; 31: 382-8.
14. Kogon B, Butler H, Kirshbom P, Kanter K, McConnell M. Closure of symptomatic ventricular septal defect defects: how early is too early? *Pediatr Cardiol* 2008; 29: 36-9.
15. Jonas R. Ventricular septal defect. In: Jonas R (ed) *Comprehensive surgical management of congenital heart disease*. London UK: Hooper Arnold Publication 2004; pp 242-55.
16. Mavroudis C, Backer C, Jacobs J. Ventricular septal defect. In: (eds) *Pediatric cardiac surgery*. Philadelphia: PA Mosby, 2003; pp 298-320.
17. Graham G. The outlook for children after cardiac surgery: ventricular septal defects. *Schweiz Med Wochenschr* 1983; 113: 1639-44.
18. Meijboom F, Sztatmari A, Utens E, Deckers JW, Roelandt JR, Bos E, et al. Long-term follow-up after surgical closure of ventricular septal defects in infancy and childhood. *J Am Coll Card* 1994; 24: 1358-64.
19. Vaidyanathan B, Roth SJ, Rao SG, Gauvreau K, Shivaprakasha K, Kumar RK. Outcome of ventricular septal defect repair in a developing country. *J Pediatr* 2002; 140: 736-41.