The Outcome of VSD Repair in Older Children and Adults

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ABSTRACT

Objective: To evaluate the outcome of surgical repair of VSD in older children and adults.
Methods: It is a retrospective study of 47 patients who underwent VSD repair by the authors during a period of 5-years. The patients included 35 boys and 12 girls. The data was retrieved from the electronic database. The numeric variables were summarized as mean, median and standard deviation while the categoric variables were presented as frequency and percentage.
Results: The mean age was 10.57 years and mean body weight at the time of operation was 24.55 kg. The commonest variety of VSD was perimembranous which was found in 33 patients. The preoperative values of haemoglobin, left ventricular dimensions, Ejection Fraction and central venous pressure were within normal range. Six patients had severe pulmonary hypertension (PA pressure >60mmHg). Seven patients needed valve repair or replacement in addition to VSD repair. The mean bypass and aortic cross clamp times were 88.21 and 56.87 minutes respectively. The mean ICU stay was 32.28 hours and the median hospital stay was 7 days. Postoperative complications were observed in 12 patients. Five patients had residual VSDs, one patient developed complete heart block and six patients showed moderate tricuspid regurgitation.
Conclusion: VSD repair can be conducted in older children & adults with as good results as in infants.
Keywords: VSD; congenital heart disease; acyanotic heart disease

INTRODUCTION

Ventricular septal defect (VSD) is the most commonly recognized congenital heart defect, occurring in approximately 2 per 1,000 live births, and constitutes over 20% of all kinds of congenital heart diseases. The most common associated anomaly is patent ductus arteriosus (PDA) in 6%. The presence of perimembranous VSD has a negative impact on the aortic valve function and with of time can result in significant aortic regurgitation in approximately 5% of patients. The other major complication of untreated VSD is progressive pulmonary hypertension which may reach supra-systemic levels in later age. In the developed countries the VSDs are diagnosed and treated during infancy to avoid any long term complications. The early results of VSD repair in these patients are excellent without any residual leaks and the need for re-operation. The long-term follow-up has also proved good quality of life and exercise capacity after VSD repair. The late closure of VSD is likely to be more challenging and associated with less favourable outcome. Unfortunately, most of the patients in our socio-economic set-up present late and are treated at relatively much older age. We therefore conducted this study to evaluate our results of VSD repair.

MATERIAL AND METHODS

The data of all patients operated by the authors (AJ & TW) from December 2008 to December 2012 was reviewed retrospectively. The patients with VSD as a part of complex congenital heart defects like Tetralogy of Fallots, pulmonary atresia, tricuspid atresia and coarctation of aorta, were excluded. However, patients having VSD in association with PDA, atrial septal defect (ASD) and aortic regurgitation and mitral regurgitation were included. The search of our electronic database produced a record of over 47 patients fulfilling our inclusion and exclusion criteria. The preoperative operative and postoperative variables of these patients were summarized using SPSS.

Diagnosis was done by echocardiography. Preoperative echocardiography indicated the type of VSD, which was confirmed intraoperatively. The operative technique was uniform in all patients. Median sternotomy was done under general anesthesia. Direct bicaval venous and ascending aortic cannulation was performed, and extracorporeal circulation was established. The PDA whenever present was dissected and ligated intrapericardially before starting cardiopulmonary bypass. Systemic hypothermia was maintained at 28-30 degrees C. The procedure was carried out under moderate systemic hypothermia and intermittent antegrade cold blood cardioplegia was given for myocardial protection. The left side of the heart was vented by direct suction through the foramen ovale along with the aortic root vent. The ventricular septal defect...
(VSD) was approached through the right atrium via tricuspid valve or by detaching the septal leaflet of the tricuspid valve which was reattached in the end. The ventricular septal defect was closed in all patients with a Dacron patch with interrupted pledgeted polypropylene sutures. In patients with a concomitant ASD it was repaired with a pericardial patch. After closing the atrial septum and right atriotomy, de-airing was performed, the aortic crossclamp was removed, and the patient was rewarmed. Extracorporeal circulation was discontinued once the hemodynamic status was satisfactory. The patients in this series had clinical and echocardiographic assessment before discharge from the hospital. This included recording of the presence and severity of any residual VSD or tricuspid valve insufficiency and evaluation of right and left ventricular function.

RESULTS

There were 47 patients which included 35 boys and 12 girls. The mean age and body weight at the time of operation were 10.57 years and 24.55kg respectively. The commonest variety of VSD was perimembranous which was found in 33 patients (Table 1). Table 2 summarizes the pre-operative, operative and post-operative variables of these patients. It shows that patients were having satisfactory pre-operative values of haemoglobin, left ventricular dimensions, Ejection Fraction (EF) and central venous pressure. Majority of these patients had normal pulmonary artery pressure except 6 six patients who presented with severe pulmonary hypertension (PA pressure >60mmHg). Four out of these six patients had isolated VSD while one patient had VSD with severe aortic regurgitation and ruptures sinus of valsalva. Another patient with high PA pressure had large PDA in addition to VSD. All of these six patients had normal postoperative recovery without any complications. The table 3 shows other procedure which were performed in addition to VSD repair. Aortic valve showed regurgitation in 5 patients. In two of these patients the valve could be saved by doing aortic valve repair while 3 patients needed replacement of aortic vale. On patient had severe mitral regurgitation while another patient had tricuspid valve regurgitation. Both of these patient underwent successful repair as the main pathology of these valve was annular dilatation amenable to annuloplasty.

The study shows relatively long bypass and cross clamp times. The average values of these times do not represent the figures for majority of the patients. These are increased due to relatively higher values for the patients who underwent additional procedures like valve repairs or replacements.

The postoperative complications were observed in 12 patients. Five patients had residual VSDs which were small in size and were treated conservatively. One patient developed complete heart block and needed pacemaker insertion. Moderate tricuspid regurgitation was noticed in six patients and again it improved by conservative management. There was no re-operation or deaths in this series. All of these patients are having regular follow-up and no late deaths have been reported during the study period.

Table 1: Types of ventricular septal defects

<table>
<thead>
<tr>
<th>Type of defect</th>
<th>n=</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perimembranous outlet</td>
<td>33</td>
<td>70.21</td>
</tr>
<tr>
<td>Subaortic</td>
<td>4</td>
<td>8.51</td>
</tr>
<tr>
<td>Muscular</td>
<td>3</td>
<td>6.39</td>
</tr>
<tr>
<td>Doubly Committed</td>
<td>6</td>
<td>12.78</td>
</tr>
<tr>
<td>Inlet</td>
<td>1</td>
<td>2.13</td>
</tr>
</tbody>
</table>

Table 2: Preoperative, operative & postoperative variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Min</th>
<th>Max</th>
<th>Median</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>2</td>
<td>25</td>
<td>10</td>
<td>10.57</td>
<td>5.71</td>
</tr>
<tr>
<td>Haemoglobin (gm/dl)</td>
<td>8</td>
<td>14.5</td>
<td>11.9</td>
<td>11.76</td>
<td>1.55</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>10</td>
<td>76</td>
<td>18</td>
<td>24.55</td>
<td>15.33</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>45</td>
<td>170</td>
<td>122</td>
<td>119.67</td>
<td>33.14</td>
</tr>
<tr>
<td>Left ventricular internal dimension: diastolic (mm)</td>
<td>27</td>
<td>86</td>
<td>45</td>
<td>47.56</td>
<td>13.48</td>
</tr>
<tr>
<td>Left ventricular internal dimension: systolic (mm)</td>
<td>9</td>
<td>65</td>
<td>27</td>
<td>29.44</td>
<td>10.63</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>40</td>
<td>80</td>
<td>65</td>
<td>65.68</td>
<td>8.28</td>
</tr>
<tr>
<td>Central venous pressure (mmHg)</td>
<td>3</td>
<td>28</td>
<td>7</td>
<td>8.28</td>
<td>4.74</td>
</tr>
<tr>
<td>Bypass time (min)</td>
<td>42</td>
<td>222</td>
<td>83</td>
<td>88.21</td>
<td>34.31</td>
</tr>
<tr>
<td>Cross clamp time (min)</td>
<td>28</td>
<td>130</td>
<td>50</td>
<td>56.87</td>
<td>23.33</td>
</tr>
<tr>
<td>ICU stay (hours)</td>
<td>17</td>
<td>112</td>
<td>24</td>
<td>32.28</td>
<td>18.21</td>
</tr>
<tr>
<td>Ventilation time (hours)</td>
<td>2</td>
<td>72</td>
<td>4</td>
<td>5.47</td>
<td>10.03</td>
</tr>
<tr>
<td>Inotropes (hours)</td>
<td>0</td>
<td>65</td>
<td>5</td>
<td>12.02</td>
<td>15.21</td>
</tr>
<tr>
<td>Chest drainage (ml)</td>
<td>140</td>
<td>1370</td>
<td>360</td>
<td>428.83</td>
<td>263.54</td>
</tr>
<tr>
<td>Blood transfusion (no. of units)</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>1.93</td>
<td>0.96</td>
</tr>
<tr>
<td>Total hospital stay (days)</td>
<td>4</td>
<td>29</td>
<td>7</td>
<td>7.73</td>
<td>3.99</td>
</tr>
</tbody>
</table>
Table 3: Operative procedures carried out in addition to VSD repair

<table>
<thead>
<tr>
<th>Variables</th>
<th>=n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic valve repair</td>
<td>2</td>
<td>4.26</td>
</tr>
<tr>
<td>Aortic valve replacement</td>
<td>3</td>
<td>6.39</td>
</tr>
<tr>
<td>Mitral valve repair</td>
<td>1</td>
<td>2.13</td>
</tr>
<tr>
<td>Tricuspid valve repair</td>
<td>1</td>
<td>2.13</td>
</tr>
</tbody>
</table>

DISCUSSION

VSD is one of the most common congenital heart defects. Almost 30% of patients with congenital heart disease have a VSD, a quarter of infants with a large VSD develop congestive heart failure and 20% have progressive pulmonary vascular disease. Since the first series of direct vision closure reported by Lillehei and colleagues in 1955, the surgical repair of ventricular septal defects have evolved significantly. Advances in surgical technique and intraoperative and perioperative management have led to marked improvement in outcomes for these patients. Long-term survival and clinical outcomes for these patients are excellent. According to a study 92% of patients were in New York Heart Association class I more than 20 years after repair. Likewise for infants undergoing surgical closure of VSD, Kuribayashi and associates reported that all survivors grew normally with a good quality of life after more than 10 years. Surgical closure of an isolated VSD is a routinely performed operation and outcomes are excellent, with mortality and morbidity rates approaching zero in almost all centers internationally. Our study also supported this data with no reported mortality.

Surgical repair of VSD leads to excellent short and long-term results. However, residual VSDs can theoretically have negative impact on long term results and some authors have advised early re-operations for residual VSDs as small as 2mm on these theoretically grounds. However, the incidence of such residual VSDs is very high in early postoperative period and up to one-third of patients may be found to have residual VSDs immediately after the operation when diagnosed with the help of colour Doppler technique. Yang et al have also reported incidence of residual VSD as high as 33%, in their study but they suggested that residual defect on intra operative TEE measuring ≥4 mm predicts the need for immediate reoperation, while a 3mm residual VSD may be significant and requires additional intra-operative hemodynamic evaluation.

On the contrary in the Natural History Study-II (NHS-II) the prevalence of a residual VSD was as low as 19.5%. In our series of 47 patients only 5(10%) patients had a residual VSD post operatively. All of our patients with residual defects were asymptomatic and medication-free and were labeled as tiny to mild residual VSD on transthoracic echocardiography. Their quality of life was judged as normal to excellent with NYHA functions class I. Therefore they were kept on follow-up and no re-operations were carried out.

The conduction system is particularly at risk during closure of perimembranous VSDs. It is because of the close anatomic relationship of the atroioventricular node and the bundle of His to the inferoposterior margin of the VSD. Therefore complete heart block has been a serious complication of VSD closure and is associated with an increased risk of late death. According to Demirag et al the incidence of complete heart block ranged from 0–2.3% in various studies while ventricular arrhythmias were common after VSD repair in their study, in agreement with other reports. With increasing knowledge of the anatomy of the conduction system surgeons can now avoid the incidence of complete heart block in the vast majority of patients. Anderson and associates recently reviewed the results of a large series of patients undergoing VSD repair during a 26-year period. Based on the results of their study, the risk of iatrogenic complete heart block with VSD closure should be less than 1% and the expected mortality rates for these patients should be approaching 0%. The results of our series of patients undergoing VSD repair support the findings of Anderson and coworkers as there was only one patient (0.02%) who developed complete heart block requiring a permanent pacemaker and an operative mortality of 0%. Scully et al also supported our findings with no incidence of heart block and an operative mortality of less than 1%. Furthermore, overall morbidity was minimal, with no patient requiring reoperation for residual VSD in our study.

The incidence of AR with VSD is 4%–5% in the Western population, but it is higher in Orientals. The AR results from two etiological factors i.e., lack of aortic valve support and hemodynamic changes producing the Venturi effect. The Venturi effect may be an important phenomenon in the progression of regurgitation and prolapse of the aortic valve leaflets because of a restrictive VSD. Restrictive VSD leads to slow progression of AR which peaks at 5 to 8 years. According to Rathore aortic valve replacement is a preferred option in patients with moderate to severe AR and a high cardiothoracic ratio, dilated left ventricle, LV dysfunction, and older age. While in young patients, valve repair is preferred as biological valves have a high rate of degeneration. In our study three patients underwent aortic valve replacement while aortic valve was repaired in two patients with good post operative
results. In our study only one patient had mitral regurgitation which was repaired with ring annuloplasty while one patient has severe tricuspid regurgitation (TR) which was repaired. Both the patients had good post operative results. According to a study tricuspid regurgitation was almost universal that is seen in almost all the cases post operatively. In contrast only six patients had moderate TR in our series while no patient had severe TR.

CONCLUSION

This study shows that VSD repair can be conducted in relatively older children and adults with as good results as in infants.

REFERENCES