Does patch closure of subarterial ventricular septal defect affect the growth of pulmonary valve?∗

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Abstract

Objective: Patch closure of the subarterial ventricular septal defect requires suture placement at the pulmonary annulus. We aimed to identify whether patch closure of subarterial ventricular septal defect would affect the growth of pulmonary annulus in comparison with that of perimembranous ventricular septal defect.

Methods: Of 361 patients who underwent patch closure of ventricular septal defect from January 1992 to December 1999, 98 (51 subarterial, 47 perimembranous) had echocardiographic data available for measurement of both preoperative and postoperative (more than 5 years after operation) pulmonary and aortic annular diameters. The pulmonary/aortic annular diameter ratio and their growth rates in the subarterial group were compared with those in the perimembranous group. The perioperative variables correlated with the pulmonary annular growth in subarterial group were also identified.

Results: The mean follow-up duration was 7.2 years. Preoperative pulmonary/aortic annular diameter ratio was 1.45 (range, 0.94—2.31) in the subarterial group and 1.57 (range, 1.15—2.51) in the perimembranous group (p = 0.059). The latest postoperative ratio was significantly lower in the subarterial group [subarterial: 1.02 (range, 0.77—1.41) vs perimembranous: 1.36 (range, 1.11—1.75), p < 0.01]. Twenty-three patients (45%) in the subarterial group had the ratio less than 1. The pulmonary annular growth rate in the subarterial group was lower than that in the perimembranous group (subarterial: 0.34 mm/year, perimembranous: 1.03 mm/year, p < 0.01). Preoperative pulmonary/aortic annular diameter ratio (r = 0.885, p < 0.01), age at operation (r = −0.0417, p < 0.01), weight at operation (r = −0.357, p < 0.05), and ventricular septal defect size (r = 0.298, p < 0.05) were found to have correlation with pulmonary annular growth in the subarterial group.

Conclusions: Our data show that pulmonary annular growth after patch repair of subarterial ventricular septal defect is suboptimal compared with perimembranous ventricular septal defect. Careful attention must be paid to the possible late clinical implication caused by impaired pulmonary annular growth after patch repair of subarterial ventricular septal defect.

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1. Introduction

The subarterial ventricular septal defect (VSD) is located at the conal septum beneath the aortic valve (AV) and the pulmonary valve (PV) [1,2]. Most commonly, there is direct continuity between the two arterial valves, even though a thin fibrous ridge separating the valves can be occasionally found. This anatomic feature allows surgeons to anchor the patch to the pulmonary annulus [3—5]. We postulate that the suture placement at the pulmonary annulus in patch repair of subarterial VSD can affect the growth of the pulmonary annulus. No previous study on this topic was found in the literature. We conducted this study to elucidate whether patch repair of subarterial VSD would hamper the pulmonary annular growth compared to that of perimembranous VSD.

2. Materials and methods

2.1. Patient description and data collection

Three hundred and sixty one patients underwent patch repair for isolated VSD (including ASD or PFO) from January 1992 to December 1999 in Dong-A University Hospital, Busan, Korea. There were 181 patients with perimembranous VSD, 146 with subarterial VSD, and 34 with muscular VSD (mostly...
muscular outlet). Subarterial VSDs extending to the perimembranous area were included in the subarterial VSD group. Of these patients, 242 (subarterial and perimembranous VSD) had follow-up data for more than 5 years. We reviewed their videotapes of two-dimensional (2D) echocardiography for measurement of the pulmonary and aortic annular diameters. Many of the videotapes had inadequate recording for exact measurement of both annular diameters before operation and at latest follow-up. Consequently, 130 patients were excluded from our analysis for this reason. Patients with significant aortic regurgitation (more than trivial, \( n = 11 \)), or residual VSD (\( n = 3 \)) were also excluded. As a result, 98 had complete echocardiographic data available for both preoperative and postoperative (more than 5 years after operation) measurement of the pulmonary and aortic annular diameters (subarterial VSD: 51, perimembranous VSD: 47).

For the assessment of pulmonary annular growth, we used the pulmonary/aortic annular diameter ratio (PV/AV ratio) and pulmonary annular growth rate (mm/year). The growth rate was calculated in the following manner: the difference in the diameter between preoperative (\( D_1 \)) and latest (\( D_2 \)) measurements was divided by the time interval in years (\( T \)) between the two measurements, i.e. growth rate = \( (D_2 - D_1)/T \). The changes in PV/AV ratio and growth rate were determined in both groups and compared with each other. Perioperative data such as age and weight at operation, the size of VSD (absolute and relative sizes to AV diameter), suture numbers at the pulmonary annulus were also collected to identify factors influencing growth of pulmonary annulus after operation.

For controls, we evaluated pulmonary and aortic annular diameters in 25 volunteers aged between 5 and 15 years (without any functional or anatomical cardiac defects) using 2D echocardiography to obtain the normal value of PV/AV ratio in this age group.

2.2. Measurement of the pulmonary and aortic annular diameters

Pulmonary annular diameters were measured at the parasternal short axis view and aortic annular diameters measured at the parasternal long axis view of 2D echocardiography, using ATL HDI5000 (Philips, USA). Aortic annular diameters were measured at their maximal valve opening (end-systolic period), and pulmonary annular diameters were measured at the end-diastolic period. Their largest hinge-to-hinge diameters were adopted for data collection. We adopted diastolic dimension of pulmonary annulus instead of its systolic dimension because the diastolic dimension was more available for accurate measurement of pulmonary anulus and there was no significant difference between the systolic and diastolic dimensions in our measurements for normal volunteers.

2.3. Measurement of the VSD size and its relative size

The size of both subarterial and perimembranous VSD was measured at the parasternal short axis view of 2D echocardiography. The relative size indicates the VSD size relative to the aortic annular diameter (VSD size/AV diameter).

2.4. Operative technique

All operations were performed by one surgeon (Dr Sung). The subarterial VSD was exposed through a longitudinal incision at the main pulmonary artery. All subarterial VSDs were repaired with expanded polytetrafluoroethylene patch (Gore-Tex, W.L. Gore & Associates Inc., USA) using non-absorbable interrupted mattress sutures reinforced by spaghetti shaped Teflon pledgets. At the upper margin of the defect where there was scarce muscular rim, the sutures were inserted through the pulmonary annulus from within the pulmonary sinuses (Fig. 1). Two to five sutures were placed within the pulmonary sinuses depending on the size of the conal defect.

2.5. Statistical analysis

All data are shown as the mean values ± standard deviation. Continuous variables were analyzed by the Student’s t-test. The difference between the preoperative and latest PV/AV ratio was determined, and changes of PV/AV ratios in SAVSD group were evaluated by paired t-test. To establish the factors influencing preoperative PV/AV ratio and the change of it, correlation and regression analyses were used. PV/AV ratios in normal volunteer were compared with the latest PV/AV ratio in the two groups using analysis of variance with post-hoc range tests (Tukey’s honestly significant difference test). A p-value less than 0.05 was considered significant. Statistical analysis was carried out using SPSS software (SPSS Inc, Chicago, IL).

3. Results

3.1. Perioperative variables and follow-up duration

Age at operation ranged from 19 days to 9 years (median, 9.9 months). Fifty-three patients (54.1%) were operated on within their first year of life. Weight ranged from 3.2 to 47.5 kg (median, 7.9 kg). Follow-up period was from 5.0 to 12.8 years (median, 6.8 years). The perioperative variables are described in Table 1. The mean age and weight at operation were higher in subarterial VSD group. PV/AV ratio and VSD size (absolute and relative sizes) were smaller in subarterial VSD group. Of these variables, statistically different variables were weight at operation (10.9 ± 7.1 kg in subarterial VSD vs 8.1 ± 4.0 kg in perimembranous VSD, \( p = 0.029 \)) and VSD size (9.0 ± 2.4 mm in perimembranous VSD vs 7.0 ± 2.0 mm in subarterial VSD, \( p = 0.000 \)). There was no difference in follow-up durations of both groups.
3.2. Growth of pulmonary annulus

3.2.1. Changes in PV/AV ratio

Because of left to right shunt through the VSD, preoperative mean diameter of pulmonary annulus was larger than aortic annulus (16.3 ± 3.2 mm in pulmonary annulus vs 11.2 ± 2.9 mm in aortic annulus) and preoperative mean PV/AV ratio was 1.5 ± 0.3. The preoperative PV/AV ratio was correlated with younger age (r = −0.349, p < 0.01) and lower body weight (r = −0.325, p < 0.01) at operation and VSD size (r = 0.345, p < 0.01). Although the mean preoperative ratio of PV/AV was slightly higher in the perimembranous VSD group, there was no statistical difference between the two groups [subarterial VSD: 1.45 (range, 0.94–2.31) vs perimembranous VSD: 1.57 (range, 1.15–2.51), p = 0.059]. During the follow-up period, the difference of the PV/AV ratio between the two groups exhibited a clear distinction, so that the mean latest PV/AV ratio was notably lower in the subarterial VSD group [subarterial VSD: 1.02 (range, 0.77–1.41) vs perimembranous VSD: 1.36 (range, 1.11–1.75), p < 0.01] (Fig. 2).

Twenty-three patients (45%) in subarterial VSD group had postoperative PV/AV ratio less than 1. The decrease in PV/AV ratio in subarterial VSD group ranged from 33.6 to 51.5% [mean decrease: 42.5%, p < 0.01 (21.4% in perimembranous VSD)]. It was also correlated with preoperative higher PV/AV ratio (r = 0.885, p < 0.01), younger age at operation (r = −0.417, p < 0.01), lower body weight at operation (r = −0.357, p < 0.05), and larger size of VSD [absolute size (r = 0.298, p < 0.05), relative size (r = 0.587, p < 0.01)] (Table 2, Fig. 3A–D). Multiple regression analysis showed good correlation between the decrease in PV/AV ratio and relative size of VSD in the subarterial VSD group (r = 0.619, p = 0.004).

3.2.2. Growth rate of pulmonary annulus

Preoperative and latest diameter of pulmonary annulus ranged from 11.3 to 30.5 mm (mean, 16.2 ± 3.2 mm) and from 14.2 to 30.7 mm (mean, 21.3 ± 4.1 mm), respectively. Growth rate was calculated using the equation of latest pulmonary annular diameter—preoperative pulmonary annular diameter (mm)/follow-up period (years). Overall growth rate of pulmonary annulus ranged from −1.45 to 2.29 mm/year (mean, 0.66 ± 0.67 mm/year). The growth rate was significantly lower in subarterial VSD group than perimembranous VSD (subarterial VSD: 0.34 ± 0.52 mm/year, perimembranous VSD: 1.03 ± 0.65 mm/year, p < 0.01) (Fig. 4).

3.2.3. Comparison of latest PV/AV ratio with normal volunteers

The ages and weights of the normal volunteers ranged from 5 to 14.4 years (mean, 10.0 ± 3.1 years) and from

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**Table 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>All (n = 98)</th>
<th>Subarterial VSD (n = 51)</th>
<th>Perimembranous VSD (n = 47)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months) [median]</td>
<td>17.7 ± 20.5 [9.9]</td>
<td>21.5 ± 22.6 [13.4]</td>
<td>13.6 ± 17.2 [7.2]</td>
<td>0.057</td>
</tr>
<tr>
<td>Weight (kg) [median]</td>
<td>9.5 ± 5.9 [7.9]</td>
<td>10.9 ± 7.1 [8.9]</td>
<td>8.1 ± 4.0 [6.6]</td>
<td>0.029</td>
</tr>
<tr>
<td>Diameter of aortic annulus (mm)</td>
<td>11.2 ± 2.9</td>
<td>11.6 ± 2.8</td>
<td>10.7 ± 3.0</td>
<td>0.128</td>
</tr>
<tr>
<td>Diameter of pulmonary annulus (mm)</td>
<td>16.3 ± 3.2</td>
<td>16.3 ± 2.9</td>
<td>16.2 ± 3.6</td>
<td>0.955</td>
</tr>
<tr>
<td>PV/AV ratio</td>
<td>1.5 ± 0.3</td>
<td>1.4 ± 0.3</td>
<td>1.6 ± 0.3</td>
<td>0.059</td>
</tr>
<tr>
<td>VSD size</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute (mm)</td>
<td>8.0 ± 2.4</td>
<td>7.0 ± 2.0</td>
<td>9.0 ± 2.4</td>
<td>0.000</td>
</tr>
<tr>
<td>Relative (VSD/AV)</td>
<td>0.84 ± 0.48</td>
<td>0.65 ± 0.28</td>
<td>0.95 ± 0.45</td>
<td>0.000</td>
</tr>
<tr>
<td>Follow-up (years)</td>
<td>7.2 ± 2.0</td>
<td>7.1 ± 2.2</td>
<td>7.4 ± 1.8</td>
<td>0.447</td>
</tr>
</tbody>
</table>

VSD, ventricular septal defect; AV, aortic valve; PV, pulmonary valve. All variables are presented as mean ± SD.

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**Table 2**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Decrease of PV/AV ratio in subarterial VSD group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
</tr>
<tr>
<td>Preoperative PV/AV ratio</td>
<td>0.885</td>
</tr>
<tr>
<td>Weight at operation</td>
<td>−0.357</td>
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<tr>
<td>Age at operation</td>
<td>−0.417</td>
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<tr>
<td>VSD size</td>
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<tr>
<td>Absolute size</td>
<td>0.298</td>
</tr>
<tr>
<td>Relative (VSD/AV)</td>
<td>0.587</td>
</tr>
<tr>
<td>Number of suture</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>0.084</td>
</tr>
<tr>
<td>Hinge</td>
<td>0.126</td>
</tr>
<tr>
<td>Hinge/total sutures</td>
<td>0.008</td>
</tr>
<tr>
<td>Follow-up duration</td>
<td>−0.171</td>
</tr>
</tbody>
</table>

PV, pulmonary valve; AV, aortic valve; VSD, ventricular septal defect.

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Fig. 2. Comparison of PV/AV ratio between groups. (*) p < 0.01. PV, pulmonary valve; AV, aortic valve; VSD, ventricular septal defect.
14.2 to 54.2 kg (mean, 33.4 ± 12.2 kg), respectively. Their mean PV/AV ratio was 1.13 ± 0.06 (range, 0.99–1.21). It was significantly higher than subarterial VSD group and lower than perimembranous VSD. There were significant differences between groups (\(p = 0.000\), ANOVA) (Fig. 5).

### 3.3. Pressure gradient through pulmonary valve

Eighteen patients (35%) with subarterial VSD had low pressure gradient through PV at the latest follow-up. The mean gradient was 10 mmHg with a range of 4–28 mmHg. There was no correlation between the gradient and decreasing PV/AV ratio and no tendency of increasing pressure gradient with time.

### 3.4. Pulmonary valve regurgitation

Thirty-two patients (62.7%) with subarterial VSD had pulmonary regurgitation, which was mostly trivial or mild. No case had pulmonary regurgitation more than a mild degree.
4. Discussion

For the last decades, VSD has been known as one of the curable congenital heart defects. Mortality rate following repair of isolated VSD during early life approaches zero in most specialized centers. Recently, many cardiologists and surgeons have shown growing interests in the late results, such as conduction disturbance, ventricular function, residual shunting, residual pulmonary hypertension, aortic, and tricuspid incompetence, rather than early morbidity or mortality. Reflecting this trend, many monitoring studies were published on the postoperative conditions and progress of repaired VSD patients. However, so far, little attention was paid to the growth of the pulmonary annulus after patch repair of subarterial VSD. After experiencing a number of VSD repair cases, we suspect that pulmonary annular growth may be adversely affected by placement of the nonabsorbable sutures at the pulmonary annulus in patch repair of subarterial VSD. In order to verify this hypothesis, we reviewed subarterial VSD cases in comparison with perimembranous VSD and found that patch repair of subarterial VSD hampered the growth of pulmonary annulus.

The subarterial VSD, which has various synonyms, such as conal defect, subpulmonary defect, type I defect, supra-cristal defect, infundibular defect, and doubly committed juxtaarterial defect, is located within the infundibular portion of the right ventricular outflow tract [2]. The subarterial VSD exhibits some particular features of their clinical presentation and surgical treatment. Higher incidences are reported in Asian than in Caucasian or Black [6]. Our data also showed that subarterial VSD comprised of 40.4% of all VSD. Small or moderately large subarterial VSD may cause AV prolapse with or without aortic insufficiency. The main cause of AV prolapse was known to be due to Venturi effect of the shunt and/or a deficiency of the tissues supporting the valve [7—9]. The actual defect size or shunt amount is frequently underestimated to some extent by the prolapsing AV into the defect [10]. An important indication for repair of this type of defect is the presence of AV prolapse or insufficiency as well as shunt amount [9,11—13].

The defect also has some important anatomic features that must be considered thoroughly for surgery. It lies immediately caudal to the PV, and the superior rim may have a direct relationship with the right coronary cusp of the AV [1]. The superior portion of the defect usually has no muscular tissue, being formed by only a thin fibrous strip between the rings of the PV and AV [1—3]. Most of the defects are closed with a patch to avoid injury or distortion of the AV [3]. Supporting the AV without injury of the AV or PV is a crucial point of the repair as well as complete interruption of the shunt. Precise sizing of the defect and the patch is absolutely necessary in order to prevent the patch from becoming redundant in which case the AV may not be supported or right ventricular outflow obstruction may occur. In placing sutures around the defect, the only dangerous part is the superior margin where there is no muscular tissue between the AV and PV. One does not need to worry about heart block because the conduction system is not in surgical proximity to the defect. If the fibrous tissue between the AV and PV is strong and large enough to anchor the sutures, it can then be used for suture placement. Such cases, however, are rare. Owing to this distinctive anatomic feature, many surgeons routinely place sutures at the base of the PV or pulmonary annulus [3—5]. Various techniques and suture materials have been used in this area. We inserted interrupted mattress sutures through the pulmonary annulus from within the pulmonary sinuses using nonabsorbable suture material reinforced by spaghetti shaped Teflon pledgets. The pledget looks like a small segment of a tiny tube made of thin Teflon membrane (Fig. 6). The main reason we use the pledget is to prevent pulmonary valve tissue crumpling while tying the sutures. We believe that this technique can minimize the distortion of the AV and PV. The suture without pledget can be used in repair of the defect. However, we think that the growth of pulmonary annulus can be impaired as long as nonabsorbable suture is used regardless of use of the pledget. Moreover, attention must be paid not to tie the sutures too hard to prevent the valve tissue crumpling, which can make the valve annulus even smaller. We do not think that pledgets used in our patching of the defects are so bulky that they could affect growth of the pulmonary annulus or pulmonary valve function adversely.

Our data show that suture placement at pulmonary annulus can impair the growth of the PV. We believe that the preoperative size discrepancy of the AV and PV is normalized through a remodeling process developed by the hemodynamic change from VSD closure and through somatic growth after closure of the VSD. Thus, we compared the data of subarterial VSD with those of perimembranous VSD in which patch repair does not involve the pulmonary annulus. The growth rate of the pulmonary annulus in the subarterial VSD group was lower than that of the perimembranous VSD group. When we compare the degree of the reduction of PV/AV ratio in the subarterial VSD group with those of perimembranous VSD, in which only remodeling process is contributed to reduction of the PV/AV ratio, it becomes clear that the suture placement at the pulmonary annulus in the subarterial VSD group contributes to further reduction of PV/AV ratio. We also measured the aortic and pulmonary diameters using 2D echocardiography.
in 25 healthy volunteers to elucidate their normal dimensions. The mean PV/AV ratio from the normal volunteers was 1.13 ± 0.06, which was well compatible with previously data published by others [14–19]. The PV/AV ratio of the subarterial VSD group also could not reach those of the normal volunteers. The implication of these findings is that the growth of pulmonary annulus can be adversely affected by patch repair in the subarterial VSD.

The decrease in PV/AV ratio in the subarterial VSD group was correlated with the preoperative higher PV/AV ratio (r = 0.885, p < 0.01), younger age at operation (r = 0.0417, p < 0.01), lighter weight at operation (r = –0.357, p < 0.05), and larger size of VSD [absolute size (r = 0.298, p < 0.05) and relative sizes (r = 0.587, p < 0.05)] (Table 2, Fig. 2). These findings clearly indicate that pulmonary annular growth is more adversely affected when the large subarterial VSD is repaired in early life. This may be due to more suture involvement of the pulmonary annulus which is required and more aggressive remodeling process follows discontinuation of a large shunt in large subarterial VSD. Undoubtedly, the reduction of PV/AV ratio is greater in large defects. Therefore, we do believe that minimal involvement of the pulmonary annulus in repair of the small subarterial VSD would not affect the growth of the pulmonary annulus but more than minimal involvement in larger defects or elliptical shape defects may produce a suboptimal growth of pulmonary annulus later in life.

Many of our subarterial VSD patients actually have no significant hemodynamic abnormality so far even though PV/AV ratio is abnormally reduced. However, careful attention is required to detect possible late occurrence of the pulmonary stenosis caused by suboptimal pulmonary annular growth in subarterial VSD. Also, further prospective study covering a larger sample size should be performed in order to confirm the negative effect on the pulmonary annular growth due to suture placement at the pulmonary annulus in subarterial VSD.

The major limitation of our study lies in retrospective data collection in which many of the pulmonary and aortic annular dimensions were not intentionally measured. Thus, the whole patient population could not be represented and there might be some inevitable errors in measurement of the dimensions. In order to overcome this limitation to a certain degree, we compared the data from subarterial VSD with those of perimembranous VSD which were collected using the same technique in the same period of time.

In conclusion, pulmonary annular growth after patch repair of subarterial VSD is found to be suboptimal as compared with perimembranous VSD. Careful attention must be paid to the possibility of late clinical impact caused by impaired pulmonary annular growth in subarterial VSD, especially in early repair and in cases with large defect.

Acknowledgement

We appreciate the efforts of Dr Hon Chi Suen in the preparation of this article.

References


Appendix A. Conference discussion

Dr A. Takriti (Damascus, Syria): I would like to ask you what about the subaortic and aortic annulus with subaortic VSD when you close?

Dr Sung: If the subaortic VSD was perimembranous, it was included in PM group, but if it was muscular outlet, it was excluded.
**Dr Takriti**: When you have subaortic VSD, you have to pass your stitches in the annulus of the aortic ring.

**Dr Sung**: I think there are some different opinions in definition, but we don’t use the term ‘subaortic VSD’.

**Dr D. Metras (Marseille, France)**: It is impressive to see your series, we know that, in Korea and Japan, about half of the VSD are infundibular. But when you say that you are going to pay attention, do you have any thought about modifying your technique because of that?

**Dr Sung**: Actually, I have no idea about that. But I think it is very important to make minimal involvement of the pulmonary annulus during suture placement as possible as we can. I’m not so sure if we can use absorbable suture, but I’m not sure of the future of the patch. I cannot answer that.