A new and simple classification for sinus of Valsalva aneurysms and the corresponding surgical procedure

Hong-Wei Guo, Hui Xiong*, Jian-Ping Xu, Xiao-Qi Wang and Sheng-Shou Hu

Department of Surgery, State Key Laboratory of Cardiovascular Disease, Fuwai Hospital, National Center for Cardiovascular Diseases, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China

* Corresponding author. Department of Surgery, State Key Laboratory of Cardiovascular Disease, Fuwai Hospital, National Center for Cardiovascular Diseases, Chinese Academy of Medical Sciences and Peking Union Medical College, 167# Beilishi Road, Beijing 100037, China.

Tel: +86-10-68313012; fax: +86-10-88398063; e-mail: xionghuidr@sina.com (H. Xiong).

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Abstract

OBJECTIVES: The classification system of Sakakibara and Konno for sinus of Valsalva aneurysm (SVA) is highly complex and seldom utilized in clinical practice. In this study, we propose a new and simple classification system; we suggest a novel approach that utilizes four distinct types of SVAs.

METHODS: We retrospectively studied 257 cases of SVAs in which surgical repair was performed between October 1996 and December 2009 and divided these cases into four types: I, rupture or protrusion into the right atrium; II, rupture or protrusion into the right atrium or right ventricle near or at the tricuspid annulus; III, rupture or protrusion into the right ventricular outflow tract under pulmonary valve and IV, others. The surgical results of the different approaches in each respective type were compared as follows: cardiopulmonary bypass time, clamp aorta time, mechanical ventilation time, intensive care unit time and postoperative stay time.

RESULTS: In all the patients, there was no early postoperative death; all the patients recovered and were discharged as expected. There were no significant differences in intensive care unit time and postoperative stay time among different approaches in each type (P > 0.05). Two hundred and thirty-eight (92.61%) patients were followed up.

CONCLUSIONS: Surgical repair of SVAs exhibited good long-term results. Our classification of SVA could be potentially helpful for surgical practice. For Type I, the right atrium approach is advised; for Type II, the transaortic approach with a right atrium incision is advised; for Type III, the transaortic approach with pulmonary incision is advised while for Type IV, repair according to the respective situation is advisable.

Keywords: Sinus of Valsalva • Aneurysm • Repair • Rupture

INTRODUCTION

Sinus of Valsalva aneurysm (SVA) is a rare cardiac abnormality, which is more common in the Eastern population compared with the Western population [1-3]. The ruptured SVA produces left-to-right shunting of blood in the cardiac chamber; extremely rare cases involve shunt to the pericardial cavity. Since the first successful surgical repair by Lillehei in 1957 [4], SVAs have been repaired with low risk and good results in current cardiovascular surgery practice [5]. The classification system of Sakakibara and Konno for SVA is highly complex [6] and seldom utilized in clinical practice. In this study, we retrospectively studied 257 cases of SVA in which surgical repair was performed in our centre over 13 years; we introduce a new and simple classification for SVA and suggest a classification system that corresponds to the type of SVA.

MATERIALS AND METHODS

Clinical presentation

Between October 1996 and December 2009, 257 patients with SVA underwent surgical repair in Fuwai Hospital in Beijing of China. One hundred and seventy-nine patients were male and 78 were female. The mean age at repair was 31.96 ± 10.84 (range, 2-67 years). The mean body weight was 61.58 ± 13.75 (range, 10-113 kg). Preoperative symptoms included palpitation in 71 (27.63%) cases, asymptomatic heart murmur in 61 (23.74%), dyspnoea in 47 (18.29%), congestive heart failure in 38 (14.79%), chest pain in 25 (9.73%), fever in 11 (4.28%) and syncope in 4 (1.56%). The origin and site of rupture in the 257 cases of SVA are shown in Table 1. Coexisting cardiac lesions consisted of ventricular septal defects in 143 (55.64%), aortic

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insufficiency in 111 (43.19%), tricuspid insufficiency in 31 (12.06%), mitral valve regurgitation in 17 (6.61%), right ventricular outflow tract stenosis in 11 (4.28%), discrete subaortic membranous stenosis in 7 (2.72%), persistent left superior vena cava in 5 (1.95%), patent ductus arteriosus in 4 (1.56%), atrial septal defect in 3 (1.17%), coarctation of the aorta in 2 (0.78%) and partial atrioventricular canal defect in 1 (0.78%).

Operative procedure

The surgical procedure was performed under cardiopulmonary bypass with moderate hypothermia through median sternotomy in all the 257 patients. Cardioplegia was infused through the aortic root or directly into the coronary ostia via an aortotomy. The mean aortic cross-clamp time was 76.33 ± 39.99 (range, 13–215 min) and the mean cardiopulmonary bypass time was 107.55 ± 48.05 (range, 39–313 min).

Corresponding procedures included repair of ventricular septal defects in 143 (55.64%) patients, aortic valve replacement in 55 (21.40%) and aortic valvuloplasty in 9 (3.50%) for aortic insufficiency, tricuspid valvuloplasty in 23 (8.95%) for tricuspid insufficiency, mitral valvuloplasty in 9 (3.50%) and mitral valve replacement in 2 (0.78%) for mitral insufficiency, correction of right ventricular outflow tract stenosis in 11 (4.28%), resection of discrete subaortic membrane in 7 (2.72%), patent ductus arteriosus repair in 4 (1.56%), atrial septal defect repair in 3 (1.17%), coarctation of the aorta in 2 (0.78%) and partial atrioventricular canal defect repair in 1 (0.39%).

A new and simple classification for sinus of Valsalva aneurysms

Following a detailed analysis of the 257 SVA cases, we created a new and simple classification system for SVA. We divided cases of SVA into four types according to the rupture or protrusion site: Type I, rupture or protrusion into the right atrium not near the tricuspid annulus (Fig. 1a); Type II, rupture or protrusion into the right atrium or the right ventricle near or at the tricuspid annulus, which means that the SVA contacts the tricuspid annulus or fuses with the tricuspid valve (Fig. 2a); Type III, rupture or protrusion into the right ventricular outflow tract under the pulmonary valve (Fig. 3a) and Type IV, rupture or protrusion into the pulmonary artery, or the pericardial cavity, or the left ventricle, or the ventricular septum or others (Table 2).

<table>
<thead>
<tr>
<th>Origin</th>
<th>Chamber of rupture</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right coronary</td>
<td>Right atrium</td>
<td>44 (31.91)</td>
</tr>
<tr>
<td>sinus</td>
<td>Right ventricle</td>
<td>137 (54.47)</td>
</tr>
<tr>
<td></td>
<td>No rupture</td>
<td>30 (13.62)</td>
</tr>
<tr>
<td>Non-coronary</td>
<td></td>
<td>82 (31.91)</td>
</tr>
<tr>
<td>sinus</td>
<td></td>
<td>140 (54.47)</td>
</tr>
<tr>
<td>Left coronary</td>
<td></td>
<td>35 (13.62)</td>
</tr>
<tr>
<td>sinus</td>
<td>Total (%)</td>
<td>257 (100)</td>
</tr>
</tbody>
</table>

Table 1: Origin and site of rupture in 257 cases of SVA

Figure 1: (A) Rupture to the right atrium not near the tricuspid annulus. (B) Partial excision of aneurysm sac and direct suture. (C) Patch repair after direct suture.

Type I cases were divided into two groups: Group 1, repair via aortic incision (n = 17) and Group 2, repair via right atrium incision (n = 51). We also divided Type II cases into two groups: Group A, repair via aortic incision (n = 19) and Group B, repair via right atrium incision (n = 21). Type III was divided into three groups: Group i, repair via aortic incision (n = 44); Group ii, repair via pulmonary incision (n = 33) and Group iii, repair via right ventricular incision (n = 72). The surgical results of different groups in each type were compared regarding cardiopulmonary bypass time, clamp aorta time, mechanical ventilation time, intensive care unit time and postoperative stay time.

Statistical analysis

Statistical analyses were performed using Statistical Package for Social Sciences (SPSS) 12.0 (SPSS, Inc., Chicago, IL, USA).
Descriptive data were expressed as mean ± standard deviation (SD). Comparisons between two groups were performed using the unpaired Student's t-test. One-way analysis of variance was used for statistical comparisons among the three groups in Type III. A P-value of <0.05 was considered statistically significant.

RESULTS

There were no early postoperative deaths. The time of mechanical ventilation support ranged from 0.5 to 109.7 h, with a mean of 11.45 ± 10.91 h. The length of time in the intensive care unit ranged from 2.4 to 445.5 h, with a mean of 38.57 ± 38.68 h. The length of postoperative hospital stay ranged from 4 to 62 days, with a mean of 8.76 ± 4.99 days. There were no significant differences in intensive care unit time and postoperative stay time among different approaches in each type (P > 0.05) (Table 2).

Figure 2: (A) Rupture to the right atrium near the tricuspid annulus, or rupture at the tricuspid annulus or rupture to the right ventricular inflow near the tricuspid annulus. (B) Partial excision of aneurysm sac and direct suture. (C) Patch repair of the origin lesion through aortotomy.

Figure 3: (A) Rupture to the right ventricular outflow tract. (B) Partial excision of aneurysm sac and direct suture. (C) Patch repair of the ventricular septal defect after direct suture of the SVA. (D) Patch repair of the origin lesion of the SVA through aortotomy.
Two patients exhibited haematuria and acute renal dysfunction 1 day and 3 days after surgery. Echocardiography and operative inspection showed residual right ventricle outflow tract stenosis and aortic paravalvular leak. Emergency operations were performed to repair the aortic paravalvular leak and to correct the residual right ventricle outflow tract stenosis. Haemofiltration and haemodialysis were used to treat acute renal dysfunction. One patient underwent haemodialysis due to acute renal dysfunction 6 days following the surgical procedure. Two patients with operative incision infection underwent debridement and suturing. All the patients recovered and were discharged. The duration of follow-up of 238 (92.61%) patients ranged from 22 to 180 (mean, 86.32 ± 43.04 months). There were no late deaths during the follow-up period. Two patients underwent aortic valve replacement due to severe aortic regurgitation. Two patients underwent radioablation ablation due to atrial flutter. One patient exhibited coronary artery disease and utilized medication. All the 238 patients were in New York Heart Association functional Classes I and II.

**DISCUSSION**

SVA is a rare cardiac anomaly that occurred in only 0.09% of an older large autopsy series [7], but in 0.14–0.23% of Western surgical series [8, 9] and 0.46–3.5% of Eastern surgical series [2, 10]. It was first described in 1839 by Hope, and the following year Thurnam [11] described the clinical features of SVA. The morpholgy of SVA was described by Edward and Burchell [12]; they demonstrated the absence of normal elastic tissue in the media between the aortic sinus and the hinge line of the aortic annulus. SVA is either congenital or acquired. A congenital lack of continuity between the aortic media and annulus fibrosis may initiate aneurysm formation. Acquired SVAs are believed to result from syphils, endocarditis, atherosclerosis, cystic medial necrosis and trauma. The origin of SVA is primarily in the right coronary sinus (65–85%), followed by the non-coronary sinus (1–30%), and it rarely occurs in the left coronary sinus (<5%) [13, 14]. In our study, the incidence of SVA origin was 82.10% in the right coronary sinus, 17.51% in the non-coronary sinus and 0.39% in the left coronary sinus (Table 1). Rupture of SVA most often occurs in the right ventricle, followed by the right atrium [15, 16], and rarely in the left ventricle [17, 18], pulmonary artery [19] or interventricular septum. In our 257 patients, rupture into the right ventricle was identified in 54.47% of cases, rupture into the right atrium in 31.91% and no rupture in 13.62% (Table 1). No cases exhibited rupture into the left ventricle, interventricular septum or pulmonary artery.

A ventricular septal defect is the most common coexisting cardiac anomaly that occurs in 30–50% of ruptured SVA patients [20, 21]. Aortic regurgitation is the second most common associated lesion in patients with SVA and is present in 20–30% of SVA patients [22]. Uncommonly, other cardiac anomalies coexist with SVA, including aortic coarctation, patent ductus arteriosus, atrial septal defect, subaortic stenosis and tetralogy of Fallot [23]. In our study, the incidence of ventricular septal defect was 55.64%, aortic regurgitation 43.19%, tricuspid insufficiency 12.06%, mitral valve regurgitation 6.61%, right ventricular outflow tract stenosis 4.28%, discrete subaortic membranous stenosis 2.72%, persistent left superior vena cava 1.95%, patent ductus arteriosus 1.56%, atrial septal defect 1.17%, coarctation of the aorta 0.78% and partial atrioventricular canal defect 0.78%.

The first formal classification system for SVA was originally proposed by Sakakibara and Konno [6]. This anatomical classification describes four types of congenital SVA arising from either the right sinus or the anterior non-coronary sinus and does not account for all of the possible chambers of penetration (Table 3); this classification system is highly complex and seldom used for clinical practice. Another proposed classification system by Ring is a hierarchical scheme based on location and acuity (ruptured or non-ruptured) with a modifier added for the etiological factor (Table 3); this is a detailed and complex classification system [24]. We introduced a new and simple classification system for non-coronary SVA. Our novel classification system divides non-coronary SVA into two types according to the rupture or protrusion site and suggests a different approach for the corresponding type of non-coronary SVA [25].

On the basis of the 257 SVA patients included in this study, we created a new and simple classification system for SVA and suggested a different approach according to the corresponding type of SVA (Table 3). For Type I, the right atrium approach is advised, using direct suture combined with patch repair (Fig. 1b and c). For Type II, rupture or protrusion site is near or at the tricuspid annulus, and the sac of the SVA is often fused with the tricuspid valve, sometimes combined with the absence of partial tricuspid septal annulus; moreover, the rupture or protrusion site is near the atrioventricular node and bundle of His. Direct suture of the

<table>
<thead>
<tr>
<th>Types</th>
<th>Groups</th>
<th>Cardiopulmonary bypass time (min)</th>
<th>Clamp aorta time (min)</th>
<th>Mechanical ventilation time (h)</th>
<th>Intensive care unit time (h)</th>
<th>Postoperative stay time (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1 (n = 17)</td>
<td>99.65 ± 32.45</td>
<td>72.71 ± 29.60</td>
<td>17.25 ± 22.16</td>
<td>40.63 ± 38.17</td>
<td>7.94 ± 2.59</td>
</tr>
<tr>
<td></td>
<td>2 (n = 51)</td>
<td>83.80 ± 32.66</td>
<td>53.52 ± 27.52*</td>
<td>9.29 ± 4.25</td>
<td>35.17 ± 24.87</td>
<td>7.82 ± 2.63</td>
</tr>
<tr>
<td>II</td>
<td>A (n = 19)</td>
<td>105.56 ± 24.93</td>
<td>79.00 ± 23.88</td>
<td>6.74 ± 2.32</td>
<td>22.38 ± 19.41</td>
<td>8.11 ± 1.69</td>
</tr>
<tr>
<td></td>
<td>B (n = 21)</td>
<td>99.10 ± 39.67</td>
<td>67.40 ± 35.20</td>
<td>13.91 ± 9.15**</td>
<td>45.70 ± 43.25</td>
<td>8.70 ± 2.16</td>
</tr>
<tr>
<td>III</td>
<td>i (n = 44)</td>
<td>134.91 ± 45.66</td>
<td>104.93 ± 37.37</td>
<td>11.30 ± 4.64</td>
<td>34.18 ± 23.57</td>
<td>8.98 ± 2.25</td>
</tr>
<tr>
<td></td>
<td>ii (n = 33)</td>
<td>131.12 ± 52.09</td>
<td>97.76 ± 44.80</td>
<td>13.88 ± 18.22</td>
<td>45.30 ± 76.32</td>
<td>10.00 ± 6.28</td>
</tr>
<tr>
<td></td>
<td>iii (n = 72)</td>
<td>106.42 ± 52.60</td>
<td>73.51 ± 38.63**</td>
<td>11.13 ± 9.93</td>
<td>41.33 ± 32.11</td>
<td>9.18 ± 4.21</td>
</tr>
</tbody>
</table>

*P < 0.05 with Group 1.  
**P < 0.05 with Group A. 
#P < 0.05 with Group II.  
##P < 0.05 with Groups I and II.
Table 3: Comparison of three different classifications of SVA

<table>
<thead>
<tr>
<th>Classification of Sakakibara and Konno</th>
<th>Classification of Ring</th>
<th>Our classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>I: Right sinus to the right ventricular outflow tract below the pulmonary valve</td>
<td>Hierarchy 1: SVA</td>
<td>I: Rupture or protrusion into the RA not near the tricuspid annulus. Surgical approach: through the RA</td>
</tr>
<tr>
<td>II: Right sinus to right ventricular infundibulum in the supraventricularis crest</td>
<td>Hierarchy 2 (location): Left sinus Right sinus Non-coronary sinus</td>
<td>II: Rupture or protrusion into the RA or RV near or at the tricuspid annulus. Surgical approach: through the Ao and RA</td>
</tr>
<tr>
<td>III:</td>
<td>Hierarchy 3 (acuity): Ruptured Non-ruptured</td>
<td>III: Rupture or protrusion into the right ventricular outflow tract under the pulmonary valve. Surgical approach: through the Ao and PA</td>
</tr>
<tr>
<td>IIIa: Right sinus to the right atrium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IIIv: Right sinus to the RV at membranous ventricular septum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV: Non-coronary sinus to the RA</td>
<td>Hierarchy 4 (pathology and chamber of penetration): Left sinus to the RA, RV, LA, LV and pericardium Right sinus to the RA, RV, PA, LV and pericardium Non-coronary sinus to the RA, RV, LA, LV and pericardium</td>
<td>IV: Rupture or protrusion into the PA, or the pericardial cavity, or the LV, or the ventricular septum or the others. Surgical approach: depending on the site of rupture</td>
</tr>
</tbody>
</table>

SVA: sinus of Valsalva aneurysm; RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle; PA: pulmonary artery; Ao: aorta.

orifice of SVA is often not sufficient to resist the pressure of the aorta; hence, patch repair is utilized. Repair via the right atrium exhibits difficulties in three aspects: first, after partial excision of the sac of the SVA, the suture rim is not visualized clearly and the aortic valve may be ruined during patch repair; secondly, the function of the tricuspid valve may be impaired after patch repair; thirdly, the atroventricular node and bundle of His may be impaired during the surgical procedure. Therefore, we suggest a transaortic approach and patch repair for origin lesion, right atrium incision and direct suture for rupture site for Type II SVA (Fig. 2b and c). For Type III, the transaortic approach with a pulmonary incision is advised, using patch repair through the aortic incision and direct suture through the pulmonary approach. During repair via pulmonary incision or right ventricle approach, after partial excision of the sac of SVA, the suture rim cannot be clearly seen, and the aortic valve may be damaged during repair; moreover, right ventricle incision may potentially ruin the function of the right ventricle, resulting in ventricular arrhythmia. A combined subarterial ventricular septal defect can be simultaneously repaired via pulmonary incision. Therefore, we advise a transaortic approach and patch repair for origin lesion, whereas we advise pulmonary incision and direct suture for rupture of the site (Fig. 3b and c).

Different classifications emphasize different aspects: Sakakibara and Konno’s classification system is an anatomical classification with emphasis on the origin and rupture site of the SVA. Ring’s classification system is a system of hierarchy that emphasizes anatomical sites and different methods of surgical procedures. Our classification system links a specific SVA type to a specified surgical procedure. Our classification system is easier and more practical for clinical usage. We offer a comparison in Table 3.

In our study, surgical repair of the SVA exhibited good long-term results. Our classification of SVA is simple and practical for clinical application. For Type I, the right atrium approach is advised, using direct suture combined with patch repair. For Type II, the transaortic approach with a right atrium incision is suggested with patch repair of the origin orifice of the SVA via aortic incision and direct suture of the outlet site via the right atrium approach. For Type III, the transaortic approach with a pulmonary incision is advised, with patch repair of the origin orifice of SVA via aortic incision and direct suture of the outlet site via the pulmonary approach. For Type IV, repair according to the respective situation is advised.

This paper presented a retrospective study, so there are some limitations of its small sample size and the non-randomized nature of the comparisons made.

Conflict of interest: none declared.

REFERENCES


