A new and simple classification for the non-coronary sinus of Valsalva aneurysm

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Received 28 December 2010; received in revised form 31 January 2011; accepted 2 February 2011; Available online 22 March 2011

Abstract

Objective: We introduce a simple classification of the non-coronary sinus of Valsalva aneurysm, and suggest a different approach for the corresponding type of non-coronary sinus of Valsalva aneurysm. Methods: Between October 1996 and December 2009, 45 patients with non-coronary sinus of Valsalva aneurysm underwent surgical repair. Twenty-three were male and 22 female. The mean age was 32.80 ± 11.77 years (range, 13–67 years). We divided them into two types, type I: rupture or protrusion into right atrium; and type II: rupture or protrusion into right atrium or right ventricle near or at the tricuspid annulus. For type I (n = 32), the right atrium approach was chosen, using direct suture with patch repair. For type II (n = 13), the transaortic approach with right atrium incision was chosen, with patch repair through an aortic incision and direct suture through a right atrium incision. Surgical results between types I and II were compared as regards cardiopulmonary bypass time, clamp aorta time, mechanical ventilation time, and intensive care unit time, and postoperative stay time. Results: There was no early death after operation. There were no significant differences in cardiopulmonary bypass time, mechanical ventilation time, intensive care unit time, and postoperative stay time between two types (p > 0.05). There was significant difference in clamp aorta time, with type II being longer than type I (p < 0.05). Forty-three patients (93.33%) were followed up; one case of coronary artery disease using medication occurred, and there was no late death. Conclusions: Approach through the right atrium or right atrium with aortotomy showed the same early surgical results. Our classification of non-coronary SVA is simple and practical for clinical usage.

Keywords: Non-coronary sinus; Sinus of Valsalva; Aneurysm; Repair; Rupture

1. Introduction

Sinus of Valsalva aneurysm (SVA) is a rare cardiac abnormality, and is more common in the Asian population than the Western population [1–3]. The majority of SVA originates in the right coronary sinus with a minority in the non-coronary sinus [4,5]. The classification system of Sakakibara and Konno for SVA defined non-coronary SVA that ruptured into the right atrium as type IV [6]. We retrospectively studied 45 cases of non-coronary SVA underwent surgical repair at our center over 13 years, and found that non-coronary SVA not only ruptured into the right atrium, but also into the right ventricle near or at the tricuspid annulus. Hence, we introduce a simple classification for non-coronary SVA, and suggest a different approach for the corresponding type of non-coronary SVA.

2. Material and methods

2.1. Clinical presentation

Between October 1996 and December 2009, 45 patients of non-coronary SVA underwent surgical repair, which comprised 17.51% of the 257 SVA operations performed in Fuwai Hospital over 13 years (Table 1). Twenty-three patients were male and 22 female. The mean age at repair was 32.80 ± 11.77 years (range, 13–67 years). The mean body weight was 61.86 ± 12.76 kg (range, 29–90 kg). Preoperative symptoms include palpitation in 12 (26.67%), congestive heart failure in nine (20%), asymptomatic heart murmur in eight (17.78%), chest pain in six (13.33%), fever and endocarditis in four (8.89%), dyspnea in four (8.89%), and syncope in two (4.44%) patients. Eight patients (17.78%) were asymptomatic and 37 patients (82.22%) were in the New York Heart Association (NYHA) functional classes III and IV. Thirty-eight patients ruptured into right atrium (84.44%), three ruptured into right-ventricular inflow tract (6.67%), and four had unruptured SVAs (8.89%) (Table 1). Co-existing cardiac
lesions were aortic valve incompetence in 12 (26.67%), tricuspid insufficiency in eight (17.78%), ventricular septal defect in four (8.8%), mitral insufficiency in three (6.7%), persistent left superior vena cava in three (6.7%), and partial atrioventricular canal defect in one (2.22%).

2.2. Operative procedure

The operation was performed under cardiopulmonary bypass with moderate hypothermia through median sternotomy in all 45 patients. Cardioplegia was infused through aortic root or directly into the coronary ostia via an aortotomy. Surgical approaches to repair SVAs included aortotomy or right atrium incision. The mean aortic cross-clamp time was 62.13 ± 33.57 min (range, 13–147 min), and the mean cardiopulmonary bypass time was 94.09 ± 40.09 min (range, 40–215 min). Simultaneous procedures for co-existing cardiac lesions included aortic valve replacement for irreparable aortic regurgitation in three patients, ventricular septal defect repair in four, tricuspid valvuloplasty in six, mitral valve replacement in one, and partial atrioventricular canal defect correction in one.

2.3. A simple classification for non-coronary SVA

From 45 non-coronary SVA patients, we evolved a new and simple classification of non-coronary SVA. We divided the non-coronary SVA into two types by the rupture or protrusion site: rupture or protrusion into the right atrium not near the tricuspid annulus (type I, n = 32); and rupture or protrusion into the right atrium or the right ventricle near or at the tricuspid annulus (type II, n = 13), which means that the SVA contacts the tricuspid annulus or fuse with the tricuspid valve (Fig. 1). For type I, the right atrium approach was chosen, using direct suture combined with patch repair (Figs. 2 and 3). For type II, the transaortic approach with right atrium incision was chosen, with patch repair through the aortic incision, and direct suture through the right atrium incision (Figs. 3 and 4).

For type I, co-existing cardiac lesions included aortic valve incompetence in five patients, tricuspid insufficiency in six, ventricular septal defect in one, mitral insufficiency in one, and persistent left superior vena cava in two. Simultaneous procedures included tricuspid valvuloplasty in four patients and ventricular septal defect repair in one. For type II, co-existing cardiac lesions included aortic valve incompetence in seven, tricuspid insufficiency in two, ventricular septal defect in three, mitral insufficiency in two, persistent left superior vena cava in one, and partial atrioventricular canal defect in one. Simultaneous procedures included aortic valve replacement in three patients, ventricular septal defect repair in three, tricuspid valvuloplasty in two, mitral valve replacement in one, and partial atrioventricular canal defect repair in one.
replacement in one, and partial atrioventricular canal defect correction in one.

Surgical results between SVA types I and II were compared as regards cardiopulmonary bypass time, clamp aorta time, mechanical ventilation time, intensive care unit time, and postoperative stay time.

The patients were followed up at our outpatient department, or by telephone calls, e-mails, and questionnaires. Follow-up time ranged from 10 to 168 months, with a mean of 71.98 ± 47.00 months. Heart function was appraised according to echocardiograms and reported symptoms.

2.4. 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 types were performed using unpaired Student’s t-test. A p value of 0.05 was used to deem statistical significance.

3. Results

There was no early death after operation. There were no significant differences in cardiopulmonary bypass time, mechanical ventilation time, intensive care unit time, and postoperative stay time between SVA types I and II (p > 0.05). There was significant difference in clamp aorta time, with type II being longer than type I (p < 0.05) (Table 2). Forty-three patients (93.33%) were followed up, one occurred coronary artery disease using medication, and there was no late death. All 43 patients were in the NYHA functional classes I and II.

4. Discussion

SVA is a rare cardiac anomaly, which may be either acquired or congenital. The clinical features of SVAs were first described by Thurnam in 1840 [7]. The morphology of congenital SVA was described by Edward and colleagues in 1957, who showed the absence of normal elastic tissue in the media between the aortic sinus and the hinge line of the aortic annulus [8]. A congenital lack of continuity between the aortic media and annulus fibrosis may initiate aneurysm formation. The congenital weakness in this region gradually gives way under aortic pressure to form an aneurysm. Congenital SVA constitutes nearly 0.14–1.5% of congenital heart repairs [9,10]. Acquired SVAs are believed to result from syphilis, infection, atherosclerosis, or cystic medial necrosis. The incidence of ruptured SVA in Asian patients is about five times more common than in white populations [11,12]. The origin of SVA is mainly in the right coronary sinus, followed by the non-coronary sinus, and rarely in the left coronary sinus [12,13]. In our group, the incidence of SVA origin was 82.10% in the right coronary sinus, 17.51% in the non-coronary sinus, and 0.39% in left coronary sinus (Table 1). Rupture of SVA most often occurs into the right ventricle, followed by the right atrium [14,15], and rarely into the left ventricle [16,17], pulmonary artery [18], or interventricular septum. In our group, rupture into the right ventricle was 54.47%, into the right atrium 31.91%, and unrupture 13.62% (Table 1). No one ruptured into the left ventricle, pulmonary artery, or interventricular septum.

It has been demonstrated that surgical repair of SVA carries low morbidity and mortality [19], since it was first repaired successfully by Lillehei and colleagues in 1957 [20]. Ruptured SVAs indicate surgical repair; unruptured SVAs that produce malignant arrhythmia, coronary ostia obstruction or right-ventricular outflow tract stenosis, and infective endocarditis are also indications for surgery [21–23]. For asymptomatic and unruptured SVAs, some clinicians argue that monitoring is the best approach, but optimal management of an unruptured, asymptomatic aneurysm is uncertain, as no precise natural history is currently available. Unruptured and asymptomatic SVAs, although remaining silent, may expand, causing more severe symptoms and requiring more extensive surgical repair in the future. Silent SVAs may lead to fatal intrapericardial rupture [24], thrombus formation, or bacterial colonization, which may cause serious complications, such as stroke and sepsis. Hence, some authors suggested that early SVA repair is usually best because surgery generally produces excellent outcomes in such cases [3]. There are many ways to repair

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Table 2. Comparisons of types I and II (mean ± SD).

<table>
<thead>
<tr>
<th>Groups</th>
<th>Cardiopulmonary bypass time (min)</th>
<th>Clamp aorta time (min)</th>
<th>Mechanical ventilation time (h)</th>
<th>ICU time (h)</th>
<th>Postoperative stay time (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I (n = 32)</td>
<td>87.19 ± 39.98</td>
<td>53.41 ± 29.94</td>
<td>9.24 ± 5.15</td>
<td>32.05 ± 28.46</td>
<td>8.75 ± 3.64</td>
</tr>
<tr>
<td>Type II (n = 13)</td>
<td>111.08 ± 36.43</td>
<td>83.62 ± 33.37</td>
<td>17.93 ± 24.66</td>
<td>36.74 ± 34.59</td>
<td>8.00 ± 2.74</td>
</tr>
<tr>
<td>p values</td>
<td>0.070</td>
<td>0.005</td>
<td>0.654</td>
<td>0.069</td>
<td>0.507</td>
</tr>
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Fig. 4. Approach for type II non-coronary sinus of Valsalva aneurysm. Direct suture exit site through right atrium approach.
SVAs, but there have been no clinical trials to demonstrate that one surgical technique is superior to another. The primary goals of SVA repair are to close the SVAs securely, remove the aneurysmal sac, and repair any associated defects. The classification system of Sakakibara and Konno for SVA is too complex, and is seldom used for clinical practice. Sakakibara and Konno divided SVA into four types, according to the origin and rupture site. They defined non-coronary SVA ruptured into the right atrium as type IV [6]. 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 etiologic factor. This is a detailed and complex classification system [25]. We found that non-coronary SVA not only ruptured into the right atrium, but also into the right ventricle near or at the tricuspid annulus. The classification of Sakakibara and Konno did not include these patients of non-coronary SVA rupture into the right ventricular near or at the tricuspid annulus. Hence, we introduced a new and simple classification for non-coronary SVA, and divided the non-coronary SVA into two types, type I: rupture or protrusion into the right atrium near the tricuspid annulus; and type II: rupture or protrude into the right atrium or the right ventricle near or at the tricuspid annulus (Fig. 1). For type I, a right atrium incision is advised, and direct suture combined with patch repair is chosen (Figs. 2 and 3). For type II, rupture or protrusion site is near the tricuspid annulus, and the sac of the SVA is often fused with the tricuspid valve, sometimes with partial tricuspid septal annulus absence; moreover, the rupture site is near the atrioventricular node and Bundle of His. Direct suture of the orifice of SVA is often not enough to resist the pressure of the aorta; hence, patch repair is used. Repair simply through the right atrium has difficulties in three aspects. First, after partial excision of the sac of SVA, the suture rim could not be seen clearly, and aortic valve may be ruined during patch repair. Second, the function of the tricuspid valve may be impacted after patch repair. Third, the atrioventricular node and Bundle of His may be impaired during operation. Therefore, we suggest a transaortic approach and patch repair for origin lesion, right atrium incision, and direct suture for rupture site for type II non-coronary SVA (Figs. 4 and 5). We also compared types I and II as regards cardiopulmonary bypass time, clamp aorta time, mechanical ventilation time, intensive care unit time, and postoperative stay time. There were no significant differences regarding cardiopulmonary bypass time, mechanical ventilation time, intensive care unit time, and postoperative stay time between types I and II. There was significant difference in clamp aorta time, type II being longer than type I. Hence, we concluded that repair through the right atrium for type I and repair via a transaortic approach combined with right atrium incision for type II had the same early surgical results.

Surgical repair of non-coronary SVA showed good long-term results. Our classification of non-coronary SVA is simple and practical for clinical usage. 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, patch repairing the origin orifice of the non-coronary SVA through an aortic incision, and direct suture of the outlet site through a right atrium approach.

References


Fig. 5. Approach for type II non-coronary sinus of Valsalva aneurysm. Patch repair the origin lesion through aortotomy.


