PEDIATRIC AND CONGENITAL HEART DISEASE

Original Studies

Percutaneous Closure of Perimembranous Ventricular Septal Defects With the Amplatzer Device: Technical and Morphological Considerations

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Percutaneous closure of perimembranous ventricular septal defects (VSDs) has been feasible, safe, and effective with the new Amplatzer membranous septal occluder. We report further experience with this device with emphasis on morphological aspects of the VSDs and technical issues. Ten patients (median age and weight, 14 years and 34.5 kg, respectively) with volume-overloaded left ventricles underwent closure under general anesthesia and transesophageal guidance (TEE). The VSD diameter was 7.1 ± 4.0 mm by angiography and 7.8 ± 3.7 mm by TEE. Three patients had defects associated with aneurysm-like formations (two with multiple exit holes), four had defects shrouded by extensive tricuspid valve tissue, two had defects with little or no tricuspid valve involvement, and one had a right aortic cusp prolapse with trivial aortic regurgitation. Implantation was successful in all patients, although in two the initial device had to be changed for a larger one. Kinking in the delivery sheath, inability to position the sheath near the left ventricular apex, and device prolapse through the VSD prompted modifications in the standard technique of implantation. Device orientation was excellent except in one case. Nine patients had complete occlusion within 1–3 months. Device-related aortic or tricuspid insufficiency, arrhythmias, and embolization were not observed. Two patients had slight gradients across the left ventricular outflow tract, normalizing after 3 months. The Amplatzer membranous septal occluder was suitable to close a wide range of perimembranous VSD sizes and morphologies with good short-term outcomes. Longer follow-up is required. Catheter Cardiovasc Interv 2004;61:403–410. © 2004 Wiley-Liss, Inc.

Key words: perimembranous ventricular septal defects; Amplatzer; interventional cardiology

INTRODUCTION

Isolated ventricular septal defect (VSD) is the most common congenital cardiac malformation, accounting for 20% of all congenital heart disease [1–3]. Eighty percent of these defects are located in the area of the membranous septum, with variable extension to the adjacent muscular inlet, trabecular or outlet septum, hence the term “perimembranous (PM) defects” [1–3]. Treatment has been classically indicated in the presence of significant left-to-right shunt resulting in left ventricular overload [3]. Surgery has been performed safely at very low rates of mortality, although complications such as residual leaks, atrioventricular block, postpericardiotomy syndrome, and arrhythmias have been well described [3]. Results of the occasional percutaneous attempts to close

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such defects have been generally poor due to the use of improper devices designed for atrial septal defect (ASD) or patent ductus arteriosus (PDA) occlusion [4–6]. Recently, a device designed specifically for PM VSD closure has been developed from the muscular VSD device by AGA Medical (Golden Valley, MN) [7–10]. It addresses the issue of the peculiar anatomical characteristics of the membranous ventricular septum, including its thickness and the vicinity to the tricuspid and the aortic valve. Recent publications have reported the feasibility, safety, and efficacy with the use of the Amplatzer membranous VSD occluder [11,12]. In this article, we report further experience with this new device, with emphasis on its suitability for different PM VSD morphologies and some technical aspects of the procedure.

**MATERIALS AND METHODS**

**Device and Delivery System**

The Amplatzer membranous VSD occluder and its delivery system have been described in detail in previous reports [11,12]. The device consists of two disks and a short cylindrical connecting waist made from a self-expandable nitinol wire mesh, with the left ventricular disk having an eccentric configuration. The delivery system consists of a delivery cable, a pusher, a rotational plastic vise, a plastic loader with a detachable hemostatic dilator [11,12].

**Patient Selection**

Patients with clinical and transthoracic echocardiographic diagnosis of significant isolated PM VSD with left ventricular volume overload and increased left ventricular end-diastolic dimensions indexed for age and body surface area were selected for the procedure. Only those patients weighing over 17–18 kg were included. Patients with other associated surgical anomalies and with severe pulmonary hypertension were excluded. Informed consent was obtained from patients or their guardians.

**Procedure**

It was performed under general anesthesia and transesophageal echocardiography (TEE) guidance. Standard views were used to assess chamber sizes and function, associated abnormalities, and atrioventricular and aortic valve function. The defect was measured at end-diastole. The distance from the aortic valve was also recorded. Left femoral artery and right femoral vein accesses were obtained and a 5 and a 7 Fr sheath were inserted, respectively. Heparin sulfate (150 IU/kg; maximum, 7,500 IU) was given. Standard right and left heart catheterization was carried out and a left ventricular angiogram was routinely obtained in the long-axial view. Angiograms in the aortic root in shallow left anterior oblique view and in the left ventricle in the hepatoclavicular view were obtained as judged necessary. A marked pigtail catheter (Royal-Flush; Cook Cardiology, Bloomington, IN) was left in the inferior vena cava (IVC) or on the chest for calibration purposes. The VSD was crossed in a retrograde fashion using a 5 Fr right coronary Judkins catheter (Cordis, Miami, FL) with the aid of a hydrophilic guide-wire (Road-Runner, Cook Cardiology). After crossing the defect, the catheter was left at the apex of the right ventricle and the hydrophilic wire was exchanged for a soft and flexible 0.035", 300 cm wire (Rope wire, AGA), which was then maneuvered to the left pulmonary artery. This wire was snared and exteriorized via the right femoral vein to establish a stable arteriovenous loop. The retrograde catheter across the VSD followed the exchange wire, being left at the junction of the IVC and the right atrium (RA). To ensure free passage of wires, catheters, and sheaths across the tricuspid valve, a 7 Fr end-hole Berman catheter (Wedge; Arrow, Reading, PA) was initially used to reach the left pulmonary artery, being subsequently exchanged for a 6 Fr therapeutic right coronary Judkins catheter (Medtronic, Minneapolis, MN) for the snaring maneuver. The long sheath and dilator were advanced from the femoral vein until the tip of the dilator touched the tip of the retrograde catheter in the IVC-RA junction (kissing catheter technique). The arteriovenous loop was tightened by pulling the wire on both sides of the system and fixing its position using surgical clamps at the end of the arterial catheter and the venous sheath. The long sheath was advanced from the femoral vein to the ascending aorta across the VSD by gently pulling the arterial catheter and pushing the sheath. Once the long sheath reached the ascending aorta, the surgical clamps were released, the dilator was retracted a few centimeters inside the sheath, and the arterial catheter was exchanged for a stiffer 5 Fr therapeutic right coronary Judkins catheter (Medtronic). After loosening the wire loop, the sheath was pulled slowly until the tip reached the aortic valve. The wire was then pushed from the arterial side to form a curve in the ascending aorta, cross the aortic valve, and enter the left ventricle. The arterial catheter was also pushed to provide more support for the wire loop to cross the valve and reach the body of the left ventricle. This maneuver in turn pushed the sheath toward the left ventricle, just below the aortic valve. The dilator was advanced over the wire, reaching the left ventricular apex, followed by the sheath. The dilator was removed and a repeat left ventricular angiogram was obtained through the side arm of the long sheath to assess the VSD morphology. The exchange wire was withdrawn from either the artery or the vein.
The device was selected to be 1–3 mm larger than the VSD size, estimated by both TEE and angiography using the maximal defect diameter. In patients with aneurysm-like formations, especially in the setting of multiple exit holes, estimation of the defect size was not as straightforward as in the remaining. The selected device was loaded and advanced through the sheath until it reached the tip of the sheath. The left ventricular disk was deployed in the mid ventricular cavity after the sheath was pulled away from the apex by gently pulling the sheath and pushing the pusher. Because of the design of the delivery system [11,12], the device invariably exited the sheath in the correct orientation, i.e., with the platinum marker in the larger part of the left disk pointing downward, toward the left ventricular apex. After deployment of the left disk, the sheath and the pusher were pulled as a unit until the left disk (almost) touched the left ventricular septum on echocardiography. The connecting waist was then deployed followed by the right ventricular disk by retracting the sheath over the pusher, keeping a gentle traction on the pusher. Device release was achieved by counterclock rotation of the delivery cable.

Repeat left ventricular and aortic angiograms and complete TEE study were used to assess final device position and orientation after release, residual shunting, and the functional status of the aortic, mitral, and tricuspid valves. Cephazolin (20 mg/kg) was given during the procedure and at 8-hr intervals (total, three doses). Hemostasis was achieved by manual compression. The patients were awakened in the catheterization laboratory and transferred to the recovery room for routine clinical observation. They were discharged home the following day and instructed to receive aspirin (2–5 mg/kg/day; maximum, 100 mg) for 6 months, avoid contact sports for 1–2 months, and observe the recommendations for endocarditis prophylaxis for 6 months or until complete closure was documented. A chest radiograph, an ECG, and a transthoracic echocardiogram (TTE) were obtained before discharge and scheduled after 1–3 months, 6 months, 12 months, and yearly thereafter.

**Statistical Analysis**

Values are expressed as means ± standard deviations or medians and range as appropriate. Linear regression was applied to assess correlation between VSD dimensions by TTE, TEE, and angiography. The level of significance was set at \( P < 0.05 \).

**RESULTS**

Transcatheter closure of PM VSDs with the Amplatzer membranous VSD occluder was attempted in 10 patients in December 2002. Successful implantation of the device was achieved in all patients. Tables I and II show the

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Pulmonary artery pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Qp/Qs</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>24</td>
<td>1.5</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>22</td>
<td>2.4</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>74</td>
<td>1.9</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>66</td>
<td>1.8</td>
</tr>
<tr>
<td>5</td>
<td>11</td>
<td>28</td>
<td>1.6</td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>19</td>
<td>2.5</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>80</td>
<td>1.9</td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>23</td>
<td>5.5</td>
</tr>
<tr>
<td>9</td>
<td>15</td>
<td>54</td>
<td>1.8</td>
</tr>
<tr>
<td>10</td>
<td>9</td>
<td>27</td>
<td>2.7</td>
</tr>
</tbody>
</table>
adjacent tricuspid valve tissue. Finally, patient 4 had a subaortic defect with prolapse of the right aortic cusp and no adjacent tricuspid valve tissue on the right side. In this patient, it was hard to profile the VSD at best on angiography due to some foreshortening caused by the prolapsing right aortic cusp. Therefore, the selected device was based on the TEE measurements.

**Technical Issues**

In patient 1, the superior portion of the left ventricular disk of an 8 mm device prolapsed into the right ventricle through the defect during device deployment. The device was removed and arbitrarily changed for a 10 mm device. In patient 3, the device was pulled through the defect because too much tension was applied to the pusher while retracting the sheath during deployment. At the second attempt, the same device was implanted appropriately. In these two instances described above, there was no difficulty retracting the device inside the sheath positioned in the right ventricle. In two patients (patients 6 and 10), the sheaths kinked at the tricuspid valve/VSD level, which prompted their removal after advancing a hydrophilic guidewire through the sheath in patient 6 or recrossing the defect in patient 10. In patient 4, the sheath would not fall into the body of the left ventricle from the ascending aorta after the dilator was pulled. Therefore, it was decided to deploy the left ventricular disk partially (assuming a bubble shape) in the ascending aorta. However, just after crossing the aortic valve, the device was inadvertently pulled through the defect. At a second attempt, the Rope wire (AGA) was first looped and left in the body of the left ventricle before advancing the sheath and dilator from the femoral vein. This allowed the sheath/dilator to reach the left ventricular apex for subsequent implantation. In patient 6, after the kinked sheath was replaced, it was felt that the device should be partially deployed in the ascending aorta given the resistance to advance the sheath across the tunnel-like defect. In this patient, the device was loaded and advanced without the pusher theoretically to optimize flexibility and decrease the amount of tension inside the sheath, which could have forced it to go back toward the right ventricle across the defect. After the device was carefully pulled through the aortic valve, it was noted that the platinum marker at the left ventricular disk was pointing upward. This was dealt with by opening the whole device in the left ventricular outflow tract and rotating the sheath and the delivery cable clockwise, achieving an adequate orientation for subsequent successful deployment. In patient 7, only after the left ventricular disk of a 10 mm device was brought close to the septum was it realized that the aneurysm formation had two sacs with one exit hole in each, one superior and the other inferior (Fig. 1). This enabled a better understanding of the underlying anatomy and sizes of the structures. The sheath was in fact crossing the septum through the inferior exit hole, with the defect itself measuring 15–17 mm at the crest of the septum on the left side. Due to this initial misjudgment, the 10 mm device was changed for an 18 mm device. Despite many attempts at rotating the delivery system, this device remained in the opposite orientation (with the marker at 10 o’clock position) after deployment. Nevertheless, the left ventricular disk was nicely covering the superior exit hole, still being distant from the aortic valve, without interfering in valve function. Therefore, the device was released with complete closure.

**TABLE II. VSD Characteristics and Procedure Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>VSD size (mm)</th>
<th>VSD location, morphology, extension</th>
<th>Other features</th>
<th>Device (mm)</th>
<th>Fluoroscopy time (min)</th>
<th>Immediate (TEE) Results 24 hr (TTE)</th>
<th>1–3 months (TTE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.1</td>
<td>6.5 Tricuspid valve tissue</td>
<td></td>
<td>8/10</td>
<td>40</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>2</td>
<td>10.2</td>
<td>10.0 Tricuspid valve tissue, inlet extension</td>
<td></td>
<td>14</td>
<td>35</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>3</td>
<td>8.7</td>
<td>6.0 Aneurysm formation, one exit hole</td>
<td></td>
<td>10</td>
<td>55</td>
<td>Closure</td>
<td>Closure</td>
</tr>
<tr>
<td>4</td>
<td>10.5</td>
<td>7.0 Right aortic cusp prolapse</td>
<td></td>
<td>12</td>
<td>56</td>
<td>Closure</td>
<td>Closure</td>
</tr>
<tr>
<td>5</td>
<td>7.0</td>
<td>6.5 Tricuspid valve tissue</td>
<td></td>
<td>8</td>
<td>24</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>6</td>
<td>4.5</td>
<td>4.5 Tricuspid valve tissue, inlet oblique extension, tunnel-like</td>
<td></td>
<td>6</td>
<td>55</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>7</td>
<td>15.5</td>
<td>17.0 Aneurysm formation, two exit holes, inlet extension</td>
<td></td>
<td>10/18</td>
<td>29</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>8</td>
<td>7.0</td>
<td>7.0 Aneurysm formation, two exit holes, inlet extension</td>
<td></td>
<td>12</td>
<td>26</td>
<td>Trivial residual shunting</td>
<td>Closure</td>
</tr>
<tr>
<td>9</td>
<td>4.0</td>
<td>3.5 No tricuspid valve tissue</td>
<td></td>
<td>6</td>
<td>16</td>
<td>Closure</td>
<td>Closure</td>
</tr>
<tr>
<td>10</td>
<td>2.5</td>
<td>3.0 No tricuspid valve tissue</td>
<td>Bicuspid aortic valve, left ventricular infundibular fold, parachute mitral valve</td>
<td>6</td>
<td>58</td>
<td>Closure</td>
<td>Closure</td>
</tr>
</tbody>
</table>
and no aortic regurgitation. However, some mushrooming appearance toward the right ventricle was seen (Fig. 1). In patient 8, the fenestrated aneurysm was only profiled at best after the injection through the long sheath was performed in the hepatoclavicular view (Fig. 2). In addition, the injection demonstrated that the sheath was positioned across the superior exit hole, with the defect measuring 7–8 mm on the left side. A 12 mm device was implanted, covering both holes. Some mushrooming effect was also seen in this patient after device release (Fig. 2).

Complications

Patient 4 had transient bilateral brachial palsy due to the arms’ position during the procedure. Patient 7 had transient AV block with bradycardia after the sheath crossed the defect. Immediate recovery was observed after atropine administration.

Immediate and Follow-Up Results

In two patients, there was slightly diminished qualitatively left ventricular function by TEE after acute volume unloading of the left ventricle. Complete closure was seen in 6 patients immediately after device release and in 9/10 patients after 1–3 months (Table II). There was no de novo tricuspid or aortic insufficiency immediately after the procedure or during follow-up. In patient 4, the ratio insufficient jet/aortic valve annulus increased from 0.10 to 0.15, which remained unchanged after 3 months.
All patients except one (patient 4) were discharged home the following day. Patients 8 and 10 had mild peak instantaneous gradients (25 and 17 mm Hg, respectively) across the left ventricular outflow tract at discharge, normalizing after 3 months. Left ventricular function was normal in all patients at discharge and during follow-up. Although all patients were in sinus rhythm, a new and incomplete right ventricular bundle branch block pattern was observed at discharge in patient 8, which remained unchanged after 3 months. All patients were clinically well, asymptomatic, and on no medications after 3 months.

**DISCUSSION**

Percutaneous closure of PM VSDs of different sizes and morphologies with the Amplatzer membranous VSD occluder was feasible, safe, and effective in this small series. This new device induces a high rate of complete closure, and the delivery system is simple, requires a relatively low profile sheath for implantation, and allows recapturing and repositioning if needed. In addition, the short waist of the device obviates protrusion into both ventricles [10–12]. Because of the eccentric configuration of the left ventricular disk, it remains distant from the aortic valve, without interfering in valve function [10–12]. The way the delivery system was designed usually forces the device to exit in the correct orientation, facilitating positioning during implantation [11,12].

A wide range of VSD sizes and morphologies was observed in this series. In this initial experience, estimation of VSD size was more difficult when the defect was associated with an aneurysm-like formation, especially in the setting of more than one exit hole on the right side.
Such diagnosis was overlooked by TTE and TEE, and even by standard angiography, with the diagnosis being made only by additional contrast media injections performed through the long sheath or through the retrograde catheter during device implantation. Selection of the size of the device was not straightforward in patients with this morphologic pattern and overestimation did occur in two patients, resulting in some mushrooming effect on the device and a gradient across the left ventricular outflow tract in a patient. Even if one measures the VSD at the crest of the septum and sticks to the rule of thumb to select a device 1–2 mm larger than the defect, mushrooming may still occur because the central waist of the device is likely to be squeezed by the exit hole on the right side. Device elongation may theoretically be avoided if one leaves the waist of the device straddling the crest of the septum and the right disk inside the aneurysm, avoiding pulling it all the way toward the right ventricle across the exit hole. On the other hand, no mushrooming was observed in one of our patients with an aneurysm-like formation and just one exit hole, in which the left ventricular disk was left inside the aneurysm, with the waist straddling the exit hole. More experience is needed to establish solid guidelines for optimal device selection in patients with these aneurysm-like formations. Furthermore, device implantation may be difficult and adequate device orientation may not be achieved in multifenestrated aneurysm, especially when the sheath is crossing the septum through an inferior exit hole, as seen in one of our patients. Although recrossing the defect through the superior exit hole may be required in some cases, releasing the device in the opposite orientation in our patient did not result in aortic valve damage since the defect was still distant from the aortic valve. However, longer follow-up on the aortic valve function is needed before this is generally accepted. A mild gradient across the left ventricular outflow tract was also detected in another patient in this series and may be related to the presence of a left ventricular infundibular fold and a narrower subaortic region, since mushrooming was not seen in that case. The gradient reduction observed in both patients after 3 months may be explained by progressive reduction in the device profile, similar to what happens to the ASDs Amplatzer devices. Although the presence of turbulent flow across the left ventricular outflow tract may theoretically damage the aortic valve leaflets as seen in patients with discrete subaortic stenosis, quick gradient reduction due to adjustments of device profile and the expected somatic growth of these two children minimize this risk.

For patients with no aneurysm-like formations, determination of the VSD size and selection of a device 1–2 mm larger than the defect were more straightforward, especially when there was little or no adjacent tricuspid valve involvement. Additional means to better assess the defect size, helping optimize device selection, such as determination of the stretched diameter by balloon inflation, could be explored in further trials. With increasing number of patients, an angiographic classification of the VSD morphology, similar to the one used for PDA closure, could also be helpful in this regard.

Because of the vicinity of PM VSDs and the tricuspid valve, it has been hypothesized that a device in this region may interfere with valve function. Although the Amplatzer membranous VSD occluder is short, the right ventricular disk does remain close to the tricuspid valve. Even so, tricuspid insufficiency was not an issue in the short-term in this experience. It has even been said that the septal leaflet is not important for valve function and the first surgical closure of VSDs was performed by suturing the septal leaflet to the communication [10]. However, we would agree that special attention should be paid to tricuspid valve function during follow-up, especially because of the somewhat longer microscrew on the right ventricular disk, which may impinge on the septal leaflet.

The decision to close the VSD in the patient with prolapse of the right aortic cusp in this series was arbitrary. Although debatable, we believe that closing the shunt below the aortic valve will eliminate the Venturi effect that causes the prolapsing process. In addition, the device itself may theoretically offer some support for the prolapsing cusp. One may argue that cusp erosion may be an issue in the long-term. However, the Amplatzer PDA occluder has been used to close a ruptured aneurysm of the sinus of valsalva with no untoward effect [13]. We would agree that long-term follow-up is mandatory to assess the adequacy of this strategy. If one proceeds to close a VSD in the setting of a prolapsed cusp, TEE seems to offer better assessment of the VSD size due to some foreshortening on angiography.

Some of the technical difficulties encountered in this series were due to our learning curve, which also explained the relatively high fluoroscopy times. Although the braided sheath was designed to avoid kinking, this happened in two patients with smaller defects. We anticipate that the same problem may also happen in small patients (less than 10–15 kg). Smaller tunnel-like defects may hinder proper progression of the sheath across the defect to reach the left ventricular apex, as seen in one of our patients. Looping the Rope wire (AGA) in the left ventricle before advancing the sheath or partially opening the device in the ascending aorta as a bubble and bringing it gently toward the left ventricular outflow tract are technical options in this circumstance. Also, implanting the device without the pusher to reduce the amount of tension on the system may be an alternative. However, by doing so, the device may not exit the sheath in the correct
orientation. If this should happen, it can be dealt with by deploying the entire device in the left ventricle and rotating the sheath and the delivery cable clockwise to make the platinum marker point downward. Despite all the catheter and wire manipulation within the defect, major injury to the conduction system did not occur in this and other series [11,12].

The rate of complete closure in this series was 90% after 1–3 months, similar to the rates achieved by surgical repair [3]. Ongoing endothelialization of the device should be responsible for the progressive decline in the rate of residual shunting with time. As such, complete closure may still be observed in our patients with a tiny shunt. Similar closure rates have been observed in other reports [11,12].

In summary, this experience showed that the Amplatzer membranous VSD occluder was suitable to close a wide range of PM VSD sizes and morphologies with good short-term outcomes. Longer follow-up is needed to draw definitive conclusions.

ADDENDUM

After revision of this article, complete closure was observed in all patients after 6 months of follow-up. Two additional patients underwent attempts at PM VSD closure with one failure due to development of complete heart block during attempts at advancing the long sheath across a 5 mm defect in an aneurysm-like morphology. In this patient, sinus rhythm was restored after 4 hr. The other patient had a 4 mm defect with little tricuspid valve tissue involvement, which was closed uneventfully after a 6 mm device implantation.

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REFERENCES