Postinfarction ventricular septal defect closure with Amplatzer occluders

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Abstract

Objective: Postinfarction ventricular septal defect (PIVSD) is a rare and life-threatening complication with high risk of both surgical and medical treatment. Another option available now is transcatheter closure. The purpose of this paper is to report the results of such treatment with Amplatzer occluders.

Method: Seven patients aged from 51 to 71 years were included. The procedure was performed between 2 and 10 weeks after myocardial infarction. One patient had double residual VSD (2 months after previous surgery) and another, coexisting critical stenosis of right coronary artery (RCA). All patients were in III/IV NYHA class, on intropes, one patient on aortic balloon counterpulsation. Venous jugular approach was used to close the VSD in six patients, venous transfemoral in one patient. Implantation of six Ampaltzer atrial septal occluders (ASO) and one muscular Amplatzer VSD occluder (VSO) were performed.

Results: All procedures but two were finished successfully. In one patient, the defect could not be entered neither from the venous nor the arterial side due to unusual oblique course (which was confirmed during subsequent operation). In the second patient (2 weeks after MI), the reason was unstable position of 24 mm ASO (probably due to necrotic borders of VSD). Immediate significant clinical improvement was achieved in all patients, in whom PIVSD was closed with Amplatzer occluders. In one postsurgical patient, two ASO were used; in another patient, prior to VSD closure, PTCA and stent implantation to RCA was performed. The stretched diameter of PIVSD ranged from 8 to 22 mm, the size of implanted Amplatzer occluders from 12 to 24 mm. Fluoroscopy time was 60 min (18–120). During the procedure, ventricular fibrillation requiring defibrillation was observed in three patients. One patient died 1 week after the procedure because of multiorgan failure and increasing mitral incompetence (MI).

Conclusions: Despite some technical problems, implantation of Amplatzer occluders, is an attractive option of treatment of patients with subacute PIVSD.

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Keywords: Postinfarction ventricular septal defect; Transcatheter closure; Amplatzer

1. Introduction

In this era of thrombolysis and early intervention, the incidence of postinfarction ventricular septal defect (PIVSD) has decreased from the historically reported 1–3% to 0.2% [1,2]. The risk factors usually associated with this severe complication are: advanced age, anterior infarction, female gender and smoking history (lack of collateral circulation). The 30-day mortality in the GUSTO-1 trial for patients treated surgically was 47 vs. 94% for those treated medically [1]. Another option is transcatheter closure of PIVSD, which is challenging because the patients are severely ill. Furthermore, patients referred for transcatheter closure have usually been turned down for surgical closure because of factors such as cardiogenic shock or advanced age. It is the purpose of this communication to report one center experience in transcatheter closure of PIVSDs.

2. Material and methods

Transcatheter closure of PIVSDs was attempted in seven patients from 51 to 71 (mean 60) years of age. Their clinical data are presented in Table 1. All patients were in III/IV NYHA class, on intropes, one patient on aortic balloon counterpulsation. Two possibilities of treatment – surgery and attempt of transcatheter closure were presented to them and all preferred interventional catheterization. Patient 4 had two residual defects 10 weeks after previous surgical closure.

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closure of PIVSD. The second communication was diagnosed after closure of the first defect with the device. In all but one patient, the procedure was performed at least 6 weeks after myocardial infarction. In patient 7, emergency catheterization was performed 2 weeks after myocardial infarction because of the deteriorating clinical condition in spite of prolonged aortic balloon counterpulsation.

Fluoroscopic and echocardiographic guidance were used during the procedure. Transesophageal echocardiography was especially useful to localize the defect in cases of inferior wall infarction. With apical defects due to anterior wall infarction, transthoracic echocardiography was sufficient and general anesthesia could be avoided (patient 7). Cardiac catheterization was carried out starting with puncture of right jugular vein (11F sheath) and right femoral vein and artery (6F sheaths). After the pressure measurements in aorta and both ventricles, a left ventriculogram was performed in the left anterior oblique position (no more measurements with Swan–Ganz catheters were performed).

In most cases, a right-sided approach via the right internal jugular vein proved advantageous because the majority of the communications were in the basilar portion of the septum. Once the defect was crossed by the catheter, a 0.035 inches £ 2.6 m Amplatz extrastiff exchange guidewire (Cook Inc.), was advanced to form a loop in the LV or ascending aorta. This provided a stable position of the wire for subsequent sizing with an OBW (Meditech) or Amplatzer (AGA Medical, Golden Valley, MN) calibrating balloon. The ‘stretched diameter’ of the VSD was determined on fluoroscopy as the diameter of the balloon that could be withdrawn across the defect with slight resistance resulting in some deformity (OBW balloons). With the static technique the waist of the AGA balloon was measured. After sizing, the balloon catheter was exchanged for an introduction delivery sheath (AGA Medical Corp. or Cook Inc.). The left sided disc was opened under echocardiographic and fluoroscopic guidance to ensure that the device did not open in the mitral chordal apparatus. If the device was not correctly positioned, it was recaptured and redeployed. Echocardiography and angiography were used to check its final position prior to release. After release, residual shunting through or around the device was recorded by echocardiography and angiography and a coexisting additional defect was excluded. If the defect could not be crossed from the venous side (patient 6), an arteriovenous guidewire loop was established by the snaring technique (Amplatz Gooseneck snare, Microvena, Corp.) previously described. [3]

3. Results

Results are presented in Table 2. In patient 5, we could not cross the defect either by venous or arterial access. This man

### Table 1
Clinical data of patients

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Sex</th>
<th>Infarct Location of VSD</th>
<th>Size of VSD (mm)</th>
<th>Time after infarction (weeks)</th>
<th>RV/LV pressure ratio</th>
<th>NYHA class</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66</td>
<td>62</td>
<td>M</td>
<td>Anterior Apical</td>
<td>18</td>
<td>6</td>
<td>0.82</td>
<td>IV</td>
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<tr>
<td>2</td>
<td>63</td>
<td>60</td>
<td>M</td>
<td>Inferior Basal</td>
<td>10</td>
<td>10</td>
<td>0.44</td>
<td>IV</td>
</tr>
<tr>
<td>3</td>
<td>65</td>
<td>73</td>
<td>M</td>
<td>Inferior Basal</td>
<td>11</td>
<td>8</td>
<td>0.48</td>
<td>IV</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>72</td>
<td>M</td>
<td>Inferior 2*</td>
<td>9 and 8</td>
<td>10</td>
<td>0.5</td>
<td>IV</td>
</tr>
<tr>
<td>5</td>
<td>71</td>
<td>75</td>
<td>M</td>
<td>Inferior Oblique</td>
<td>–</td>
<td>–</td>
<td>0.6</td>
<td>IV</td>
</tr>
<tr>
<td>6</td>
<td>53</td>
<td>70</td>
<td>F</td>
<td>Inferior Basal</td>
<td>10</td>
<td>10</td>
<td>0.54</td>
<td>IV</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>84</td>
<td>M</td>
<td>Anterior Apical</td>
<td>15</td>
<td>2</td>
<td>0.50</td>
<td>IV</td>
</tr>
</tbody>
</table>

* Two residual defects 10 weeks after previous surgical closure.

### Table 2
Results of the procedures

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Access</th>
<th>Sizing (mm)</th>
<th>Device</th>
<th>Procedural success</th>
<th>Fluoroscopy time (min)</th>
<th>RV/LV pressure ratio</th>
<th>Residual shunt</th>
<th>Outcome</th>
<th>Follow up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RIJV</td>
<td>19</td>
<td>ASO 20</td>
<td>Yes</td>
<td>18</td>
<td>0.44</td>
<td>Small</td>
<td>Death</td>
<td>1 week</td>
</tr>
<tr>
<td>2</td>
<td>RIJV</td>
<td>15</td>
<td>ASO 18</td>
<td>Yes</td>
<td>33</td>
<td>0.24</td>
<td>Trivial</td>
<td>Good NYHA II</td>
<td>18</td>
</tr>
<tr>
<td>3</td>
<td>RIJV</td>
<td>18</td>
<td>ASO 19</td>
<td>Yes</td>
<td>35</td>
<td>0.29</td>
<td>Small</td>
<td>Good NYHA II</td>
<td>13</td>
</tr>
<tr>
<td>4</td>
<td>RIJV</td>
<td>15</td>
<td>ASO 16</td>
<td>Yes</td>
<td>87</td>
<td>0.29</td>
<td>Significant</td>
<td>NYHA II</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>RIJV</td>
<td>8</td>
<td>ASO 15</td>
<td>Yes</td>
<td>116</td>
<td>0.22</td>
<td>No</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>6</td>
<td>Different</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>120</td>
<td>0.6</td>
<td>–</td>
<td>Surgery – death</td>
<td>–</td>
</tr>
<tr>
<td>7</td>
<td>RIJV</td>
<td>8</td>
<td>VSO 10</td>
<td>No</td>
<td>59</td>
<td>0.54</td>
<td>No</td>
<td>NYHA II</td>
<td>4.5</td>
</tr>
<tr>
<td>8</td>
<td>RFV</td>
<td>8</td>
<td>VSO 12</td>
<td>Yes</td>
<td>45</td>
<td>0.31</td>
<td>–</td>
<td>Surgery – good</td>
<td>2</td>
</tr>
</tbody>
</table>

* RIJV, right internal jugular vein; RFV, right femoral vein; ASO, Amplatzer atrial septal occluder; VSO, Amplatzer ventricular septal occluder.
was operated on and the defect proved to be an intramural tunnel-like oblique canal from the apical portion of the septum on the left side to the basal portion entering the right ventricle under the septal leaflet of the tricuspid valve. This patient died 6 weeks after surgery because of severe pancytopenia.

The procedure was also unsuccessful in patient 7 – with a large apical VSD 2 weeks after myocardial infarction. In this patient, a 24 mm Amplatzer ASD occluder always pulled through into the right ventricle probably because of undersizing and the necrotic margin of the communication. Two efforts to reposition the device also failed. He was successfully operated on. During open-heart surgery, the defect was found to be 30 mm in diameter with irregular necrotic borders. This patient survived surgery.

The procedure was successful in the remaining five patients. The first patient (catheterization performed with Professor J. Masura, Bratislava Children University Hospital) died 1 week after catheterization with multiorgan failure and increasing mitral incompetence (MI) present before the procedure. In the second patient, we stented a dissected distal right coronary artery (RCA) prior to PIVSD closure (Fig. 1a–c). The patient is doing well 18 months after the procedure (NYHA class II). The third patient experienced transient hemolysis due to a residual shunt. Patient 4 had two residual shunts after surgery, which were successfully closed in two procedures with a 15 and 16 mm Amplatzer atrial septal occluders (ASO) devices (Fig. 2a,b). In the sixth patient, implantation of a 10 mm Amplatzer muscular VSD device via right internal jugular vein failed. The procedure was successfully repeated from a right femoral vein approach (after creation of arteriovenous loop) with a 12 mm muscular VSD device. She is in NYHA class II, 4.5 months after the procedure. Five out of seven patients with PIVSDs survived and were discharged from the hospital doing well.

4. Discussion

In 1988, Lock et al. reported transcatheter closure of post-myocardial infarction VSD with the Rashkind double umbrella [4]. However, all three patients died soon after the procedure. Since that time, only sporadic reports have appeared in the literature using the Amplatzer ASO [3,5–7]
or CardioSeal septal occluder [8]. The procedure is one of the most challenging in interventional cardiology because margins of the defect may have necrotic borders and the usually very poor clinical condition of these patients. Amplatzer devices, in contrast to patch-like devices [4], are self-centering and stent the defect. Occlusion is achieved mainly by in situ thrombosis of its waist. By slightly oversizing Amplatzer devices, it is possible to eliminate defect mainly by in situ thrombosis of its waist. By slightly oversizing Amplatzer devices, it is possible to eliminate defect caused by tissue necrosis. The left ventricular retention skirt of the ASO device (7 mm) can also cover adjacent fenestrations. The most advantageous and unique feature of the Amplatzer devices is the possibility of repositioning and redeployment in case of unsatisfactory implantation. ASD devices were not designed to close PIVSDs and are, therefore, not ideal because they have only a 4 mm waist. The Amplatzer muscular VSD device has a smaller retention skirt with a 7 mm waist conforming better to the adult muscular septum which measures on the average 10 mm in thickness. The smaller retention skirts may result in displacement of the device during positioning (as in patient 6). The best closure device for this condition is likely the new Amplatzer PIVSD prosthesis which has a 10 mm waist and a larger retention skirt and is specifically designed for this purpose. The procedure was successful in five out of six in the subacute phase, when scar tissue had developed or started to develop (after 6 weeks). In the acute phase of myocardial infarction, however, the defect may enlarge, as a result of continuing resorption of necrotic tissue (as was confirmed during subsequent surgery in patient 7). In such a situation, additional theoretic risk factor during the procedure may result from sending emboli of necrotic tissue to the systemic circulation. Hopefully, we did not observe such complication in our patient. The best results can probably be expected in cases of residual postsurgical VSDs, when the patient is stable and a firm scar has formed giving support to the implant [5,6]. Another point of concern is possible interference of implanted device with mitral valve apparatus with further increase of MI. We tried to avoid such complication with continuous echocardiographic guidance during the deployment procedure. The incidence of residual shunts after surgery is about 20% [9,10] and the mortality rate close to 50%. Judging by our limited experience, interventional closure of these defects can easily surpass the surgical results.

5. Conclusions

Catheter closure of PIVSDs is believed to be at least as safe as surgical closure. More patients are needed to confirm this statement. Best results are obtained in infaracts at least 6 weeks old, or shunts after surgical patch closure when a firm scar has been formed. If at all possible, intervention should be delayed until that time – our experience in a single patient with acute myocardial infarction was discouraging. We conclude that device closure should only be limited to poor surgical candidates but could become the procedure of choice.

Acknowledgements

The authors would like to thank Professor Amplatz for his permanent help and consultations of the patients.

References


Appendix A. Conference discussion

Dr R. Dion (Leiden, The Netherlands): Before asking the audience whether somebody has experience with such a procedure, can you confirm that the patients were treated 6 weeks after the infarction?

Dr Zembala: Yes.

Dr Dion: Were they only apical or central VSDs, not posterior ones with involvement of the tricuspid valve and posterior wall?

Dr Zembala: Well, in an interventional fashion, we recognize them as apical or basal. The basal ones can have lower location that can be best approached from the upper part, through the upper right jugular vein. So we also have had the patients with basal defects. There were three of them here.

Dr Dion: Have you treated the coronary disease at the same time?

Dr Zembala: Yes. These patients all had their coronary artery fluoro-
scopic examination the night before the procedure. And in one case we had the critical stenosis in the right coronary artery and it was treated before the procedure.

And you asked about the acute patient. It was the one that we haven’t got the consent for surgery before the procedure. We tried to do it by intervention and it failed. After all he agreed to surgery and we had good result with it. So it’s a small group of patients, but we recognize the role of surgery in the acute phase, definitely.

Dr. Dion: How did you measure the size of the defect and what was the ratio between the size of your Amplatzer and the size of the defect?

Dr. Zembala: The measurement of the defect was made in three stages. First, it was measured by transesophageal or transthoracic echocardiography. And this measurement tended to be the smallest one we got. Then we performed the measurement by balloon technique. We used two techniques. And we have very repeatable results comparing both techniques. One is with OBW balloon. We put the balloon into the left ventricle, then make the inflation with dilute contrast material to a diameter that we measured before with T8 plus a little bit. Then we retrieve the balloon throughout hole. We look at the change of the shape of the balloon at the moment of crossing the VSD, measure it, then we retrieve the balloon and we measure it out of the patient on a special plate with holes.

Another balloon technique is the aesthetic one, when we have the long balloon, one end in the left ventricle, the closer end in the right ventricle, we make inflation and we measure the waist that happens in the VSD. I think that it’s actually the problem in the acute phase patient the way we measure it with balloons. I’m afraid that making the inflation – applying pressure in a recent VSD, we can actually enlarge it. In those cases I think we should rely just on TEE measurements. That is risky and we can face the situation of device displacement after the operation.

Dr. Dion: And how much do you oversize the Amplatzer?

Dr. Zembala: Generally we oversize it by 4 to 7 mm. It’s a short, short series. We had no displacements. So maybe we need less of it, but we are afraid of further ongoing necrosis or the presence of some small adjacent holes. So we hope that the bigger disk can cover the adjacent holes, too, and will make some insurance for enlargement of the defect.

When we did the same with a atrial septal defects, at the beginning we had had the tendency to use bigger implants. Now we employ smaller and smaller sizes and still the displacements are very unusual.

Dr. L. Balacumaraswami (Oxford, United Kingdom): Can I just ask you what your anticoagulation protocol was, or did you have any anticoagulation at all?

Dr. Zembala: Our protocol was the full heparinization during the procedure and next 24 hours, another day on Choloxin, followed only by antiplatelet treatment for 6 months. We have just copied the same protocol that we used for ASD or the congenital VSD closure.

Dr. Balacumaraswami: The Teflon within the nitinol mesh needs to clot off before you commence anticoagulation, so do you leave at least 48 hours?

Dr. Zembala: Yes. We don’t use warfarin later on, just antiplatelets, aspirin.

Dr. Balacumaraswami: Did you routinely use temporary pacing wires during the procedure?

Dr. Zembala: No. We have the intravenous access all the time, so if we face the complete heart block, we can make the temporary pacing with the electrodes with ease.

Dr. P. Bhatnagar (Hyderabad, India): Did you do a concomitant revascularization also in these patients, consider the dye load in this unstable group of patients?

Dr. Zembala: No, these patients all were previously treated as an acute phase and we had them several weeks after. We only performed the PTCA in one patient in this group.

Dr. Bhatnagar: They had just a primary VSD closure and PTCA later on?

Dr. Zembala: In one case it was the secondary closure. One patient had 2 VSDs that were arrested postsurgically and all the others our intervention was the primary approach to the closure of the defect. One patient had surgery after our approach. And it was unsuccessful in our approach, successful in surgical approach. So it was 1-to-1 in this case.