Transapical Valve-in-valve Implantation in Failed Mitral Bioprostheses

Implante transapical de prótesis valvular mitral sobre bioprotesis degeneradas

To the Editor,

Reoperation for mitral bioprosthesis dysfunction is a challenging procedure due to patients’ clinical status and the technical difficulties associated with resecting the prosthesis and implanting a new valve in the weakened mitral annulus; in addition, the redo surgery carries the risk of structural damage to the myocardium and perivalvular leakage.

Transcatheter valve-in-valve implantation into a deteriorated mitral bioprosthesis is a little used technique,1–4 but in complex patients it provides an alternative to conventional surgery that avoids cardiac dissection, extracorporeal circulation, and myocardial ischemia. Most prostheses are implanted via the transapical route because this approach provides direct coaxial access to the valve with shorter delivery catheters; however, another access option is the femoral vein, followed by perforation of the interatrial septum and anterograde valve implantation.

From January 2007 to September 2014, the surgical team at our center performed 70 implantations via the transapical route. In 6 patients, a new valve was implanted in a deteriorated prosthesis: 4 in the aortic position and 2 in the mitral position. The clinical characteristics of the 2 patients selected for the mitral procedure are summarized in the Table.

Both mitral valve implantations were carried out under general anesthesia in the cardiac catheterization laboratory by 2 surgeons and a cardiac catheterization specialist. A left anterior minithoracotomy was performed through the fifth or sixth intercostal space, and transapical access through the pericardial opening was secured by 2 U sutures with teflon-supported 3/0 monofilament suture thread. The ventricular apex was punctured with a guidewire, which was advanced through the opening of the mitral prosthesis into the left atrium. To prevent traumatic perforation of the atrial wall, this guidewire was then exchanged for a preformed high-support guidewire. Coaxial alignment with the ring of the deteriorated bioprosthesis was guided by radiology and echocardiography. This procedure was more challenging with the Epic bioprosthesis because its annulus is only faintly radiopaque; in contrast, the Perimount bioprosthesis, in addition to having a larger annulus, also has a radiopaque supporting stent. After balloon predilatation, an Edwards SAPIEN XT Transcatheter Heart Valve (Edwards Lifesciences, Irvine, California, United States) was implanted in the opposite orientation to that of a transapical aortic valve implant, following the standard protocol with rapid endocardial pacing (Figure). The valve sizes used were 23 mm for patient 1 and 26 mm for patient 2. These sizes were chosen according to the manufacturer-specified internal diameters of the original bioprostheses, which were consistent with direct measurements by transesophageal echocardiography (23 mm for the 25-mm Perimount valve and 24.5 mm for the 27-mm Epic valve). In both patients the procedure was completed without technical incident, and correct implantation was confirmed by intraoperative transesophageal echocardiography.

Patient 1 developed cardiogenic shock in the first hours after implantation, with biventricular dysfunction, acute renal failure, and elevated hepatic enzymes; the condition was reversed by amine therapy and the placement of an intra-aortic counterpulsation balloon for 72 hours. The discharge echocardiogram showed normal function of the mitral bioprosthesis, a left ventricular ejection fraction of 35%, and a systolic pulmonary arterial pressure of 70 mmHg.

Patient 2 was extubated 8 hours after device implantation, and did not require amine therapy at any time. During hospitalization,

Table
Baseline Clinical Profile of the 2 Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y</strong></td>
<td>79</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td>Female</td>
</tr>
<tr>
<td><strong>Comorbidities</strong></td>
<td>PVD</td>
</tr>
<tr>
<td><strong>Prosthesis type</strong></td>
<td>25-mm Perimount</td>
</tr>
<tr>
<td><strong>Mitral valve disorder</strong></td>
<td>Moderate combined regurgitation and stenosis MVA: 1.1 cm² MR: grade III</td>
</tr>
<tr>
<td><strong>LVEF, %</strong></td>
<td>45</td>
</tr>
<tr>
<td><strong>SPAP, mmHg</strong></td>
<td>65</td>
</tr>
<tr>
<td><strong>NYHA functional class</strong></td>
<td>III</td>
</tr>
<tr>
<td><strong>Logistic EuroSCORE I</strong></td>
<td>58.12</td>
</tr>
</tbody>
</table>

CB, coronary bypass; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; MVA, mitral valve area; MVR, mitral valve replacement; NYHA: New York Heart Association; OMC, open mitral commissurotomy; PVD, peripheral vascular disease; SPAP, systolic pulmonary arterial pressure; TA, tricuspid annuloplasty.

* a 2014: Severe mitral regurgitation due to perforation of a prosthetic leaflet. Imaging results were consistent with endocarditis, but the patient had no fever, and blood cultures were negative.

* b 2012: Prosthesis replaced due to endocarditis caused by Abiotrophia defectiva.
the patient was treated with intravenous antibiotics for a respiratory infection. The discharge echocardiogram showed normal mitral bioprosthesis function with grade I periprosthetic regurgitation from the posterior commissure, grade II tricuspid regurgitation, and a systolic pulmonary arterial pressure of 65 mmHg.

Both patients showed an improvement in New York Heart Association functional class from class III to class II and improved hemodynamic parameters. Patient 1 at 12 months and patient 2 at the 6-month follow-up:

- Patient 1: mitral valve area > 2 cm²; maximum velocity, 2.1 m/s; left ventricular ejection fraction, 30%; systolic pulmonary arterial pressure, 65 mmHg.
- Patient 2: mitral valve area, 2.2 cm²; mean gradient, 8 mmHg; left ventricular ejection fraction, 50%; systolic pulmonary arterial pressure, 55 mmHg; severe tricuspid regurgitation; no mitral regurgitation.

The first treatment option for a failed biological prosthesis is reoperation, but mitral valve explant surgery carries a number of risks. Transcatheter valve implantation is changing the treatment options for aortic valve disease in patients at high surgical risk, but the potential role of the transcatheter approach remains unclear in the treatment of failed bioprosthetic valves in the mitral position.

In our study, we selected 2 high-risk patients excluded from conventional surgery. Although the early postoperative outcomes differed, the clinical progress of both patients was positive, even in the presence of multiple comorbidities. Left ventricular dysfunction continues to be a risk in mitral valve repair because ventricular function is often overestimated in patients with mitral regurgitation, which might partly explain the initially poor postoperative outcome in patient 1. In our opinion, management options for such patients should include the presurgical deployment of an intra-aortic counterpulsion balloon and therapy with inotropic drugs in the first hours after valve implantation. These measures, once introduced, dramatically improved clinical progress in patient 1.

In conclusion, transcatheter valve-in-valve implantation in a deteriorated mitral bioprosthesis via the transapical route is a feasible procedure that is becoming established as an alternative therapeutic option for patients at high or prohibitive surgical risk.

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Available online 21 July 2015

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http://dx.doi.org/10.1016/j.rec.2015.04.009