Aortic Bioprosthetic Valve Deterioration 8 Months After Implantation

Dominik Wiedemann, Nikolaos Bonaros, Günther Laufer and Alfred Kocher

Ann Thorac Surg 2010;89:277-279
DOI: 10.1016/j.athoracsur.2009.06.081

approached at this time), and the distal anastomosis was completed with reattachment of the celiac axis. The previous carotid-subclavian bypass graft was left in place. To address the fistula, omentum was harvested as a pedicled graft and used to cover the thoracoabdominal graft and seal the left upper lobe fistula.

The patient tolerated the second procedure and awoke neurologically intact, without further complications or respiratory complaints. No more bloody sputum episodes occurred postoperatively. He was discharged with 6 weeks of intravenous antibiotics and life-long oral antibiotics.

Comment

Most cases of ABF are secondary and detected in patients with previous thoracic or cardiac operations, therefore requiring a redo emergency aortic procedure. Despite the improved results with traditional open repair in recent years [1, 2], surgical mortality and morbidity are still a major concern due to the complexity of the procedure, emergency conditions, contaminated field, and the difficulties of exposure in the reoperative setting. Emergency perioperative mortality ranges from 16% to 24%, with a mortality as high as 41% in secondary ABF [1, 3].

Thoracic stent graft repair may be an alternative for aortic rupture into the airways. Although most of the recent studies have reported no perioperative death, stroke, or paraplegia, these primarily have been reports of single patients or small series, with the largest including 8 patients [3, 4]. In a recent review of ABF, perioperative mortality was 0% in the stent graft vs 16% in the open surgical group [1].

Advantages of stent graft repair include the avoidance of the stress of an open repair with anticoagulation for cardiopulmonary bypass, lessening the risk for bleeding. The primary concern remains durability. Stent grafts may migrate, develop endoleaks, or lead to bronchial wall erosion with recurrent ABF. Although these complications may occur less frequently with later-generation devices, recurrences requiring reintervention have been reported in 40% of patients, with an associated 30% mortality.

Recurrence of ABF is likely related to the deployment of a stent graft in a potentially infected field adjacent to a vulnerable aortic wall susceptible even to the small forces generated by endotension. Furthermore, rushed aortic evaluation in the emergency setting may lead to imperfect stent graft sizing [5]. With open repair, extensive débridement, graft replacement of the aorta, and pedicled flap coverage are possible, none of which can be achieved with endovascular repair.

In conclusion, the natural history of ABF is lethal hemorrhage without prompt treatment. The mortality of open surgical repair remains high when performed in the emergency setting. Stent graft repair decreases the perioperative risk, allowing for recovery from the initial insult of bleeding and rupture; however, this is fraught with frequent recurrences associated with significant mortality. A good treatment strategy is the staged approach using a thoracic stent graft to control initial bleeding and prevent respiratory soilage, followed later by a more definitive open thoracic surgical repair.

G. Ken Goodrick is acknowledged for his editing help and Chris Akers for the figures.

References

1. Piciche M, De Paulis R, Fabbri A, et al. Postoperative aortic fistulas into the airways: etiology, pathogenesis, presentation, diagnosis, and management. Ann Thorac Surg 2003;75:1998–2006.
2. Eren E, Keles C, Toker ME, et al. Surgical treatment of aortobronchial and aortoesophageal fistulae due to thoracic aortic aneurysm. Texas Heart Inst J 2005;32:522–8.
3. Wheatley GH 3rd, Nunez A, Preventza O, et al. Have we gone too far? Endovascular stent-graft repair of aortobronchial fistulas. J Thorac Cardiovasc Surg 2007;133:1277–85.
4. Bockler D, Schumacher H, Schwarzbach M, et al. Endoluminal stent-graft repair of aortobronchial fistulas: bridging or definitive long-term solution? J Endovasc Ther 2004;11:41–8.
5. Pitton MB, Herber S, Schmiedt W, et al. Long-term follow-up after endovascular treatment of acute aortic emergencies. Cardiovasc Intervent Radiol 2008;31:23–35.

Aortic Bioprosthetic Valve Deterioration 8 Months After Implantation

Dominik Wiedemann, MD, Nikolaos Bonaros, MD, Günther Laufer, MD, and Alfred Kocher, MD

University Clinic of Cardiac Surgery, Innsbruck Medical University, Innsbruck, Austria

One year after aortic bioprosthetic valve replacement with a 23-mm Epic bioprosthesis (St. Jude Medical, St. Paul, MN) with concomitant coronary artery bypass grafting, early valve degeneration with leaflet calcification and stiffening was documented in a 61-year-old man. A possible reason for this extremely early deterioration could not be identified, although a xenograft reaction could be a plausible hypothesis. Consequently, a successful redo aortic valve replacement with a 21-mm Magna Ease (Carpentier-Edwards, Irvine, CA) was performed.

(Ann Thorac Surg 2010;89:277–9)

The Epic stented porcine prosthesis (St. Jude Medical, St. Paul, MN) in the aortic position has demonstrated excellent long-term durability and hemodynamic stability [1]. Low rates of valve-related events and structural failure prove that the Epic bioprostheses is a good choice for patients with calcified aortic stenosis. Degeneration of bioprostheses usually occurs rather late in an age-dependent manner. St. Jude bioprostheses are at risk

Accepted for publication June 15, 2009.

Address correspondence to Dr Wiedemann, University Clinic of Cardiac Surgery, Innsbruck Medical University, Anichstr. 35, Innsbruck, A-6020, Austria; e-mail: dominik.wiedemann@i-med.ac.at.

0003-4975/10/$36.00
doi:10.1016/j.athoracsur.2009.06.081

of structural valve degeneration beginning as soon as 7 years postoperatively for patients aged younger than 65 years. Therefore, aortic valve replacement with a bioprosthesis is a reliable option, especially for elderly patients. We describe a patient with early valve deterioration of a St. Jude Medical bioprostheses 1 year postoperatively.

A 61-year-old man with coronary artery disease (CAD) and severe aortic stenosis combined with moderate aortic insufficiency was admitted to our hospital. His preoperative echocardiogram confirmed severe aortic stenosis (maximum pressure gradient, 98 mm Hg; mean gradient, 57 mm Hg), moderate aortic regurgitation, and normal left ventricular function. He had 80% stenosis of the left anterior descending artery (LAD) and 60% stenosis of the right coronary artery, after two percutaneous coronary interventions with unsatisfactory long-term results. Both coronary arteries were excellent target vessels for coronary artery bypass grafting (CABG).

The patient opted for a biologic valve because he objected to lifelong anticoagulation. He underwent aortic valve replacement with a 23-mm St. Jude Epic bioprosthesis with concomitant CABG with the left internal mammary artery (LIMA) to the LAD and a saphenous vein graft to the posterior descending artery. His postoperative course was uneventful and he could be discharged after 6 days.

By 8 months after the operation, dyspnea symptoms had already reappeared. An echocardiogram showed reappearance of severe aortic stenosis, with a maximum gradient of 100 mm Hg that rapidly progressed to a maximum gradient of 170 mm Hg. He had mean gradient of 90 mm Hg at 11 months.

A redo aortic replacement was performed with a 21-mm Carpentier-Edwards Magna Ease bioprosthesis (Edwards Lifesciences, Irvine, CA). The St. Jude valve (St. Jude Medical) presented as strongly degenerated, the leaflets were restricted with a fibrous layer on both sides, and the opening surface of the valve was reduced to a minimum (Fig 1). The valve was sent for histologic evaluation, which showed mild infiltration of granulocytes together with signs of calcification.

The patient's postoperative course was again uneventful. A transesophageal echocardiogram revealed a mean gradient over the aortic valve of 28 mm Hg (maximum, 33 mm Hg). This is in the acceptable range for this patient, with a patient prosthesis match of 0.91 cm^2/m^2. No further intervention was necessary, and the patient is doing well 6 months postoperatively.

Comment

This patient presented with leaflet fibrosis within a 1 year. The Epic stented bioprosthesis has been used in Europe for almost 9 years and received United States Food and Drug Administration approval in 2004. It has a proven history of excellent durability and long-term

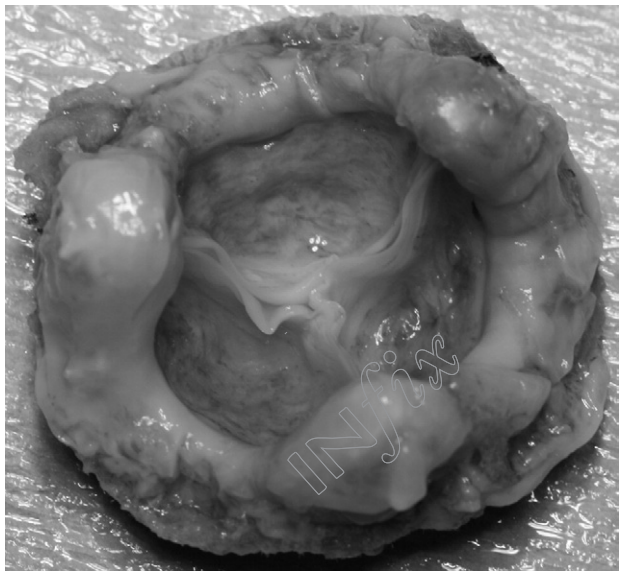

Fig 1. The degenerated St. Jude valve is shown after explantation.

stability of hemodynamic performance in both the aortic and mitral positions. Valve dysfunction rates are low.

Classic reasons for deterioration of bioprosthetic valves are calcification, infection, degeneration, instrument trauma, and leaflet detachment from the valve strut. There were a few reports of early calcification of bioprosthetic valves [2, 3]. The crucial pathologic process of valve degeneration is leaflet calcification [4]. Glutaraldehyde pretreatment-devitalized residual cells often initiate leaflet calcification. The following mechanism leads to reaction of calcium rich extracellular fluid with membrane-bound phosphorus to build calcium phosphate mineral deposits. Calcification of the leaflets leads to leaflet stiffening and finally failing of the valve. The rate of valve deterioration in older patients is lower and the process is accelerated in younger patients. Reasons for this age dependency are unclear [5].

A recent study showed that valve calcification can be induced or inhibited by special factors [6]. Dialysis patients often have early valve degeneration together with vascular calcification. Risk factors for the vascular calcification include calcium or vitamin D supplementation. One may speculate that higher systemic calcium levels can also trigger early bioprosthetic valve degeneration. There is a potential relation between calcium consumption and early leaflet calcification. Reduced kidney function may increase the deposition of calcium.

Our patient had none of these risk factors for early degeneration. His serum calcium levels were normal preoperatively and postoperatively, with no calcium supplementation perioperatively. Renal function was only slightly reduced, with maximum creatinine levels of 1.4 mg/dL. The serum calcium levels were always within the normal reference range in the routine blood samples.

A rather new hypothesis for early bioprosthetic valve degeneration is xenograft reaction [7]. Although glutaraldehyde fixation decreases the antigenicity of biopros-

thesis, it does not eliminate antigens completely. Rejection and as consequence, calcification, may be a reason why bioprosthetic valves fail, especially in young patients. In an experimental study, Manji and colleagues [7] showed that glutaraldehyde-fixed xenografts show cellular and humoral signs of rejection. The histologic evaluation in our patient showed a rather unspecific granulocyte infiltration and only a few signs of inflammation. Therefore, we could not prove xenograft rejection as reason for the early failure of the bioprosthetic valve; nevertheless, this might be a possible explanation.

Despite the early valve degeneration, the patient did not receive a mechanical heart valve, because of his attitude to life-long anticoagulation. In view of the possible mechanisms of deterioration, we switched from a porcine valve to a bovine pericardial bioprosthesis because we believed this change provided the best possible antigenic diversity to preclude a second immune-mediated xenograft rejection.

References

1. Eichinger WB, Hettich IM, Ruzicka DJ, et al. Twenty-year experience with the St. Jude medical Biocor bioprosthesis in the aortic position. Ann Thorac Surg 2008;86:1204–10.
2. Polo ML, Legarra JJ, Vilar M, Cabrera A, Duran D, Pradas G. Early calcification of a Carpentier-Edwards Perimount mitral valve in an elderly woman. J Thorac Cardiovasc Surg 2002; 124:1043–4.
3. Izutani H, Shibukawa T, Kawamoto J, Mochiduki S, Nishikawa D. Early aortic bioprosthetic valve deterioration in an octogenarian. Ann Thorac Surg 2008;86:1369–71.
4. Butany J, Nair V, Leong SW, Soor GS, Feindel C. Carpentier-Edwards Perimount valves—morphological findings in surgical explants. J Card Surg 2007;22:7–12.
5. Schoen FJ, Levy RJ. Calcification of tissue heart valve substitutes: progress toward understanding and prevention. Ann Thorac Surg 2005;79:1072–80.
6. Speer MY, Giachelli CM. Regulation of cardiovascular calcification. Cardiovasc Pathol 2004;13:63–70.
7. Manji RA, Zhu LF, Nijjar NK, et al. Glutaraldehyde-fixed bioprosthetic heart valve conduits calcify and fail from xenograft rejection. Circulation 2006;114:318–27.

Benign Metastasizing Leiomyoma: A Rare Metastatic Lesion in the Right Ventricle

Sean D. Galvin, MBChB, Brecon Wademan, MBChB, John Chu, FRACP, and Richard W. Bunton, FRACS

Departments of Cardiothoracic Surgery and Cardiology, Dunedin Hospital, Dunedin, New Zealand

Cardiac tumors require resection for diagnostic purposes and to avoid complications associated with an intracardiac mass. We present the case of a 41-year-old woman with a known uterine leiomyoma who presented 3 months after elective cesarian section and hysterectomy with a right ventricular mass that was confirmed histologically to be a benign leiomyoma of the same pathologic type as the uterine primary. Benign metastasizing leiomyoma is a rare pathologic entity occurring in women with a history of a uterine leiomyoma. This is the second reported case of cardiac metastasis from a benign uterine leiomyoma.

(Ann Thorac Surg 2010;89:279–81)

Tumors of the heart are uncommon in everyday surgical practice. Primary cardiac tumors are rare and are usually benign. Most cardiac tumors occur secondary to metastatic disease processes and these are 20 to 40 times more common than primary cardiac malignancies [1]. Intracardiac tumors may require resection for diagnostic purposes and to avoid complications, including right heart failure or right ventricular outflow tract obstruction.

A 41-year-old woman was investigated for a systolic murmur. Three months earlier she underwent an elective cesarean section and hysterectomy. Histology showed a benign intrauterine leiomyoma without venous invasion. This was also present at a cesarean section 7 years previously.

A transthoracic echocardiogram suggested a mass in the right ventricular outflow tract, and computed tomography (CT) of the chest (Figs 1A and 1B) showed a mass in the right ventricle without other intrathoracic lesions. Transesophageal echocardiogram (Figs 1C and 1D) confirmed a 35- × 19-mm pedunculated lesion, arising from the anteroseptum of the right ventricle, mobile in the right ventricular outflow tract.

Through median sternotomy and right atriotomy, a firm, highly mobile pedunculated mass (Fig 2A) was found attached to the interventricular septum anteriorly. This was resected (Fig 2B) without need for septal repair. Immunohistochemical staining was positive for both estrogen and progesterone receptors and histology (Figs 2C and D) was consistent with an intracardiac leiomyoma the same histologic type as the uterine primary.

Postoperatively, CT scans of the chest, abdomen, and pelvis showed no metastatic disease at 6 months, and transesophageal echocardiogram performed at 18 months showed no recurrent tumor in the right ventricle.

Comment

Leiomyoma are the most common benign tumour of the uterine myometrium and rarely pose significant clinical problems. On occasions, however, a uterine leiomyoma may directly invade the venous system (intravenous leiomyomatosis) and reach the right atrium through extension in the inferior vena cava [2]. Benign metastasizing leiomyoma, however, describes a different pattern of spread, whereby a histologically benign leiomyoma exhibits metastatic qualities and is found at sites distant to the uterus [3]. It is a unique condition occurring in women with

Accepted for publication June 9, 2009.

Address correspondence to Dr Galvin, Department of Cardiothoracic Surgery, Dunedin Hospital, Great King St, Dunedin, 9001, New Zealand; e-mail: galse@xtra.co.nz.

0003-4975/10/$36.00
doi:10.1016/j.athoracsur.2009.06.050

Aortic Bioprosthetic Valve Deterioration 8 Months After Implantation

Dominik Wiedemann, Nikolaos Bonaros, Günther Laufer and Alfred Kocher

Ann Thorac Surg 2010;89:277-279
DOI: 10.1016/j.athoracsur.2009.06.081