Aorto-Right Atrial Fistula: A Complication of Prosthetic Aortic Valve Endocarditis. A Case Report

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The case presented is of prosthetic aortic valve endocarditis in a 23-year-old male patient, complicated by an inflammatory periprosthetic valvular leak, and fistula formation between the aorta and the right atrium. The fistulous tract was diagnosed intraoperatively. Closure of the fistula was performed successfully in concordance with re-replacement of the aortic valve.

The Journal of Heart Valve Disease 2006;15:142-145

Prosthetic valve endocarditis (PVE) is defined as an endovascular, microbial infection occurring on parts of a valve prosthesis or on a reconstructed native heart valve. The condition is classified as early PVE when patients present with typical clinical symptoms within one year of the operation, and late PVE if symptoms occur later than 12 months (1). The etiology of early PVE is considered nosocomial, whilst late PVE is generally considered to be community-acquired.

Infections of mechanical prostheses usually originate from the sewing cuff or from a nearby-located thrombus, rather than from the metallic or polymer material which does not allow microbial adherence. The resulting inflammation in the area may lead to periprosthetic leaks, ring abscesses, and invasion of nearby tissues by the infective process. This, in turn, may lead to fistula formation.

Case report

A 23-year-old male patient was referred to the authors' hospital to evaluate a paravalvular leak after prosthetic valve replacement. At eight weeks before presentation to the hospital, the patient had undergone aortic valve replacement for aortic regurgitation, using a Medtronic 23 monotilting disc prosthesis. Both, operative and postoperative periods were uneventful. Six weeks later, the patient developed fever (38.5°C), palpitations and mild dyspnea. The early blood cultures were negative, but a third positive culture was obtained which revealed *Staphylococcus epidermidis*, which was sensitive to vancomycin. In spite of continued medication, the patient's condition continued to worsen, and he became increasingly dyspneic. Echocardiography revealed a significant paravalvular aortic leak with annular vegetations.

Upon admission to the hospital, the patient's vital signs showed a regular pulse of 80 beats/min, a blood pressure of 120/50 mmHg and a temperature of 37.7°C. Laboratory tests were normal and repeated blood cultures were negative. Transthoracic echocardiography (TTE) showed the same findings as previously, with moderate mitral regurgitation. The patient's left ventricular diastolic diameter was 6.2 cm, the left ventricular systolic diameter 5.0 cm, the left atrial dimension 4.3 cm, and the ejection fraction 40% (Fig. 1).

The decision was taken to add gentamicin (80 mg tds) and rifampicin (900 mg bid) to the antibiotic regimen. However, the patient remained dyspneic and his fever did not settle. A decision was subsequently made to undertake surgery.

The heart was exposed via the previous median sternotomy incision. Extensive adhesions were found and dissected, and routine cannulation of the aorta, superior and inferior venae cavae was carried out. Cardiopulmonary bypass was instituted with systemic hypothermia of 30°C. Blood cardioplegia was given initially through the aortic root, with subsequent doses administered via direct coronary ostia. The mitral valve was exposed through a left atriotomy. The valve showed moderate mitral regurgitation, but there was no evidence of endocarditis. The valve was repaired with a commissuroplasty using Teflon pledgets.

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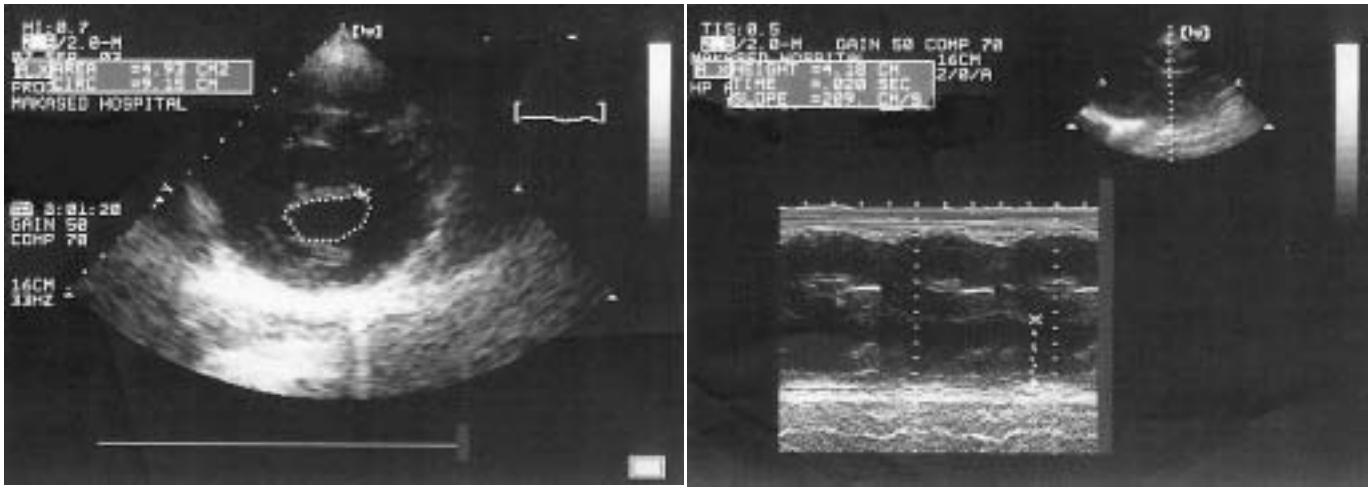

Figure 1: Patient preoperative cardiac parameters.

An oblique aortotomy was used to expose the prosthetic aortic valve. Friable vegetations were found all over the annulus. A 0.5 × 0.5 mm opening was found just above the junction of the non-coronary and left coronary cusps. Likewise, a fistulous tract was found which extended from this opening to the right atrium and was adherent to the aortic wall. The prosthetic valve below the fistula appeared almost completely detached. The entire valve was removed and all vegetations were cleared. Interrupted pledget sutures were used with caution, as most of the annulus area was soft and friable, especially near the site of the fistula. Due to the unavailability of a homograft, a prosthetic St. Jude Medical valve 19 HP was used.

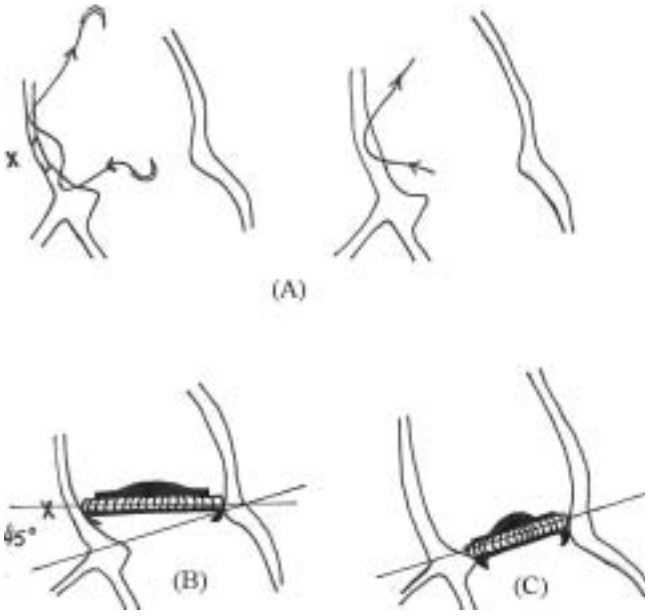

Figure 2: A) The 'X' indicates the site of the aorto-right atrial fistula. Sutures were taken into available healthy tissue above and below the fistula. The suture was anchored to the prosthetic aortic valve. B) The final position of the prosthetic aortic valve, which appeared tilted at 45º. C) The normal position of the prosthetic aortic valve.

The orientation of the newly implanted valve was somewhat different. A combination of both annular and supra-annular implantation was used. The aortic site of the fistula was incorporated within the suture line, using the healthy aortic wall below and above the fistula to anchor the valve. The final orientation of the valve was oblique; that is, supra-annular at the site of fistula and annular in the remaining part of the annulus (Fig. 2). The patient was weaned from the bypass, without complications, and transferred to the cardiac intensive care unit without inotropic support.

Discussion

Today, with improvements in prosthetic valve design and material, valve replacement surgery is generally performed with low mortality and morbidity. Nonetheless, PVE remains a serious complication (1), occurring in up to 3.7% of patients who undergo primary or redo replacement surgeries (2).

A variety of microorganisms are involved in the pathogenesis of PVE. In early cases, *Staphylococcus epidermidis* is the responsible bacterium in most cases, followed by *Staphylococcus aureus*, enterococci and fungi. In contrast, streptococci are involved in the majority of cases of late PVE, together with *Staph. aureus*, enterococci and the HACEK group (*Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikinella*, and *Kingella*) (1,3,10).

In early cases of PVE, the organism reaches the valve prosthesis either directly during surgery or via the blood during the initial days or weeks after surgery. These organisms will gain access to the prosthetic

J Heart Valve Dis
Vol. 15. No. 1
January 2006

annulus interface and to the perivalvular tissue along the sutures, because in early cases the valve sewing ring, cardiac annulus and anchoring sutures are not endothelialized. This is in contrast to late cases of PVE, where these structures are already endothelialized and infection usually will start at the sites of platelet and fibrin aggregates. Extension of the infection beyond the prosthetic valve occurs in 50% of cases (4). Initially, the infection invades the continuous basal myocardium, mitral-aortic intravalvular fibrosa and the conduction system (5).

The infecting organism causes cellulitis, followed by abscess formation. Since the myocardial wall is under considerable pressure the necrotic tissue will disrupt, resulting in a communication with either ventricle, great vessel or pericardium (6). Fistulous communications are commonly seen between the aorta and the left atrium, left ventricle and right atrium and the septum, and this in turn leads to conduction defects (5).

Clinically, patients with PVE present with symptoms and signs similar to those encountered in native valves. However, when infection develops early following surgery, the findings related to surgery or perioperative complications will predominate. The appearance of new or changing murmur, congestive heart failure and new electrocardiographic conduction disturbances occur when the infection begins to invade the surrounding structures. Indeed, 40% of patients may present with arterial emboli, whilst embolic infarcts or hemorrhages affecting the central nervous system are seen in 20-40% of cases (7,11,12).

The diagnosis for the present patient was based on major clinical Dukes' criteria (13), there being a positive blood culture, new regurgitant murmur, and a positive echocardiogram.

The use of TTE to diagnose PVE presents difficulties due to the materials used in the construction of the prosthetic valve, as these will obscure the passage of ultrasonic waves. This problem was solved by using transesophageal echocardiography (TEE), which proved to be highly sensitive compared to TTE (14), and superior in the diagnosis of complications such as ring abscesses, pseudoaneurysms and fistulae (15).

In the present patient the diagnosis was based on both clinical presentation and TTE, which showed clear paravalvular leak and vegetations. The presence of a fistulous tract was missed, despite the use of color-coded Doppler. It appears that the presence of severe regurgitation obscured the site of the fistula. The fistulous tract was found between the aorta and the right atrium. It seems that the vicinity of the right atrial appendage to the aortic wall, as visualized in the operating room, explains such communication.

Treatment of PVE with antimicrobial agents alone frequently fails, as invasion of the infection with subsequent valve dysfunction will occur before or during therapy. Although the present patient received antibiotics and anti-failure therapy (frusemide, captopril, atenolol and spironolactone) for at least four weeks, his temperature and general condition remained abnormal.

The appearance of murmurs suggestive of valve dysfunction, moderate or severe congestive heart failure, and persistent fever for 10 or more days despite antibiotic therapy are indications of an extension of the infection (7). These findings not only suggest abscess formation but also that antibiotics alone cannot cure the condition; consequently, surgical intervention is needed, and this is associated with a morbidity/mortality of 32 to 45% (6).

The extent of the infection in PVE ranges from being localized in the prosthesis, extending to the annulus, or invading the heart. Infection extending beyond the prosthesis represents a difficult challenge in terms of surgical management. However, improvements in myocardial protection, the use of biological materials and the development of new generations of antibiotics has improved the outcome of surgical management of PVE. In the majority of cases, surgery to excise the infected tissue, combined with valve replacement and intensive long-term antibiotic therapy, has been shown to be successful. However, in severe cases it was necessary to use patches and subcoronary valved tubes (8). In cases where the aortic wall is destroyed, or where the aortic annulus is of a small size, a supra-aortic valved conduit has been used (9).

In the present patient, the infective process had extended beyond the annulus to form a fistulous communication with the right atrium. Part of the annulus below the fistula was destroyed. Due to the unavailability of a homograft, the decision was made to remove the infected tissue and to replace the aortic

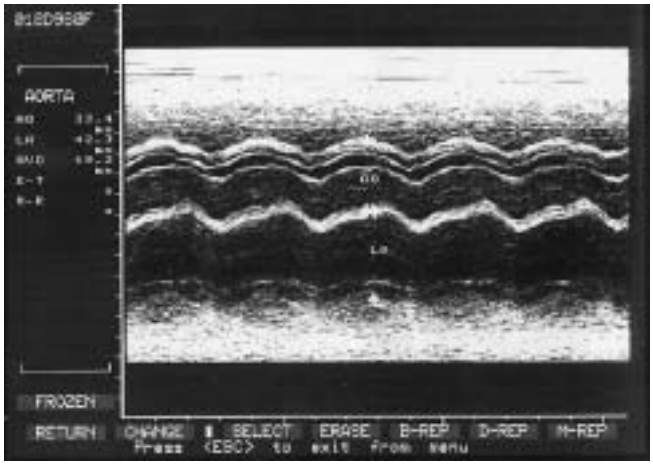

Figure 3: Recent two-year follow up echocardiography of the patient.

valve. The aortic wall above and below the fistula was healthy, and anchoring the prosthetic valve at this level was achieved by using sutures which passed below the fistula, then above it, and then through the prosthetic valve annulus. Attachment of the prosthetic valve to the remainder of the annulus was carried out in the usual manner.

Improvements in the early and late surgical outcome of PVE can only be achieved by the early detection of infection, before annular destruction has occurred, as once the infection has extended beyond the annulus the treatment will be technically difficult. Re-replacement of the prosthetic valve is one method used in this situation. The present patient was followed up for almost two years (Fig. 3), and consequently the technique used proved to be effective.

In conclusion, PVE remains a serious complication of valve replacement. Diagnosis is possible using TTE, but TEE should be used routinely to diagnose complications that might be encountered. Early surgical intervention to debride the infected tissue, extensive antibiotic treatment and replacement of the prosthetic valve can affect both early and late outcome.

References

1. Piper C, Korfer R, Horstkotte D. Prosthetic valve endocarditis. Heart 2001;85:590-593
2. Agnihotri A, McGiffin D, Galbraith A, O'Brien M. The prevalence of infective endocarditis after aortic valve replacement. J Thorac Cardiovasc Surg 1995;110:1708-1724
3. Vlessis AA, Khaki A, Grunkemeier GL, et al. Risk, diagnosis and management of prosthetic valve endocarditis: A review. J Heart Valve Dis 1997;6:443-465
4. Carpenter JL. Perivalvular extension of infection in patients with infective endocarditis. Rev Infect Dis 1991;13:127
5. Karalis DG, Bansol RC, Hauck AJ, et al. Transesophageal echo recognition of subaortic complications in aortic valve endocarditis: Clinical and surgical implications. Circulation 1992;86:353
6. Leung DY, Cranney GB, Hopkins AP, Walsh WF. Role of transesophageal echo in the diagnosis and management of aortic root abscess. Br Heart J 1994;72:175
7. Calderwood SB, Swinski LA, Karchmer AW, et al. Prosthetic valve endocarditis: Analysis of factors affecting outcome of therapy. J Thorac Cardiovasc Surg 1986;92:776
8. Jault F, Gandjbakhch I, Chastre JC, et al. Prosthetic valve endocarditis with ring abscesses, surgical management and long-term results. J Thorac Cardiovasc Surg 1992;105:1106
9. Danielson GK, Titus JL, Dushane JW. Successful treatment of aortic valve endocarditis and aortic root abscesses by insertion of prosthetic valve in ascending aorta and placement of bypass grafts to coronary arteries. J Thorac Cardiovasc Surg 1974:67:443-449
10. Karchmer AW. Infective endocarditis. In: Braunwald E, Zipes DP, Libby P (ed.). Heart Disease: A Textbook of Cardiovascular Medicine. Volume 1, 6th edn. WB Saunders Co, Philadelphia, PA, 2001:1723
11. Keyser DL, Biller J, Coffman TT, Adams HP, Jr. Neurological complications of late prosthetic valve endocarditis. Stroke 1990;21:472
12. Ben Ismail M, Hannachi N, Abid F, et al. Prosthetic valve endocarditis. A survey. Br Heart J 1987;58:72-77
13. Durak DT, Lukes AS, Bright DK. New criteria for the diagnosis of infective endocarditis: Utilization of specific echocardiographic findings. Am J Med 1994;96:200-209
14. Daniel WG, Mügge A, Grote J, et al. Comparison of transthoracic and transesophageal echocardiography for detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic positions. Am J Cardiol 1993;71:210-215
15. San Roman JA, Vilacosta I, Sarria C, et al. Clinical course, microbiologic profile, and diagnosis of perianular complications in prosthetic valve endocarditis. Am J Cardiol 1999;83:1075