Aspergillus Prosthetic Valve Endocarditis Causing Functional Aortic Stenosis: Initial Case Report

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Aspergillus Prosthetic Valve Endocarditis Causing Functional Aortic Stenosis: Initial Case Report

Section:
CASE REPORTS


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Despite extensive advances in cardiology over the past 50 years, the definitive treatment for aortic stenosis remains unchanged. Aortic valve replacement, while employing enhanced prostheses, continues as an imperfect therapy with inherent risks and complications. Prosthetic valve endocarditis (PVE), which affects as many as 3.4% of patients in the year following surgery and 1% annually thereafter,¹ is frequently associated with reoperation and significant mortality. Valve thrombosis remains a concern in patients with mechanical prostheses, particularly if adequate anticoagulation is not maintained. While endocarditis and thrombosis can occur at any time, mechanical degeneration of prosthetic valves is generally time-dependent, manifesting 10–20 years (or more) after valve replacement.² We report a case of Aspergillus infection leading to obstructive endocarditis of a bioprosthetic valve.
Case Report. A 59-year-old male with a history of calcific aortic stenosis, coronary artery disease and ischemic cardiomyopathy presented with chest pain, dyspnea and fevers. Nine months earlier he underwent aortic valve (AoV) replacement with a Carpentier-Edwards pericardial bioprosthesis and two-vessel coronary artery bypass graft surgery. Prior to hospital discharge, the estimated left ventricular ejection fraction was 45% with inferoposterior hypokinesis. The velocity across the AoV at that time was not documented, however, the valve appeared morphologically normal with normal leaflet motion.

Four months following valve replacement, the patient developed fevers with redness and drainage at the site of a recently implanted cardiac defibrillator (ICD). Blood cultures documented methicillin-resistant Staphylococcus aureus (MRSA). The ICD and its leads were removed, and the patient received intravenous vancomycin with resolution of his fevers.

Seven months postoperatively, the patient pre-presented with fevers, but no obvious infectious source was discovered and blood cultures remained sterile. He was empirically treated with antibiotics for a presumed respiratory infection with resolution of his fevers.

Nine months after valve replacement, the patient presented with left-sided chest pain and dyspnea. Examination on supplemental oxygen revealed a temperature of 99.7°F, a blood pressure of 114/52, a pulse of 78/minute, respirations of 22/minute, and oxygen saturation of 99%. His jugular veins were not distended. The patient’s breath sounds were diffusely decreased with dullness to percussion at the left base. Cardiac examination revealed a mid-peaking III/VI harsh systolic murmur at the left sternal border. There was mild peripheral edema.

Laboratory studies revealed a white blood cell count of 19,700/microliter without leftward shift. The electrocardiogram revealed sinus rhythm, lateral ST-T wave abnormalities, and inferior Q-waves and was unchanged compared to prior tracings. Chest radiography confirmed the presence of a left pleural effusion with pulmonary edema. Chest, abdominal and pelvic computed tomography scans revealed no other acute pathology. Pleuritic fluid cultures showed no bacterial growth. Blood cultures obtained at presentation also remained sterile.

The patient developed hypotension requiring vasopressors, as well as respiratory failure requiring mechanical ventilation. Given the onset of clinical heart failure, cardiac catheterization was performed and revealed pulmonary hypertension with a peak transaortic gradient of approximately 75 mmHg (Table 1 and Figure 1). Catheter pullback performed under fluoroscopic guidance was notable for the absence of both an intraventricular gradient and a pulse pressure decline following ventricular extrasystoles, thus casting doubt on the diagnosis of functional subaortic stenosis. Native and graft coronary angiography revealed no discrete lesions in the left circumflex artery, with patent bypass grafts to both the LAD and RCA.

Following cardiac catheterization, the differential diagnosis included thrombosis of the bioprosthesis, obstructing vegetation and commissural fusion.
Transthoracic Doppler echocardiography confirmed the invasively determined hemodynamic data, estimating a 65 mmHg peak instantaneous gradient across the bioprosthesis. However, due to poor acoustic windows, direct visualization of the bioprosthesis was limited. Left ventricular function was markedly reduced, with an estimated ejection fraction of 15%. Transesophageal echocardiography (TEE) was performed and revealed a 4 x 2 cm mass on the bioprosthetic AoV, encasing all three leaflets and severely limiting leaflet excursion (Figure 2). Doppler examination revealed a peak gradient of nearly 100 mmHg.

Concern was raised that the findings on TEE could represent either infectious endocarditis or thrombus involving the bioprosthesis. Given the history of MRSA bacteremia and recurrent fevers, PVE was the favored diagnosis. With valvular obstruction leading to heart failure and hemodynamic instability and the ongoing risk of embolization with a mass of this size, the decision was made to operate urgently. At surgery, a 3.5 x 1.5 cm mass was found encasing all three leaflets of the bioprosthesis (Figure 3). Two subannular abscess cavities were debrided and patched with bovine pericardium. A 21 mm Carpentier-Edwards pericardial bioprosthesis was implanted. Microbiologic examination revealed fungus consistent with an Aspergillus species. Follow-up transthoracic echocardiography prior to discharge revealed a gradient of 40 mmHg across the aortic bioprosthesis without evidence of recurrent vegetation.

Discussion

Initially described in 1967, endocarditis leading to valvular obstruction rather than regurgitation is less common, but certainly no less clinically important. Reports have identified endocarditis leading to valvular obstruction in native aortic and mitral valves as well as in prosthetic valves, resulting from numerous pathogens, both bacterial and fungal. Obstruction may result from valvular vegetations or the formation of a perivalvular abscess.

PVE is categorized based on the temporal relationship of infection to surgery. Early infections within 60 days of surgery usually result from perioperative skin or wound infection, residual infected endocarditis tissue, or contaminated intravascular devices such as catheters or pacemaker leads, with most pathogens being gram-positive bacteria. Late PVE after 60 days is caused by a spectrum of pathogens similar to native valve endocarditis. Fungal PVE can occur early or late, but is generally not seen early in the absence of preoperative fungal infection. Most fungal endocarditis is caused by Candida species, although other fungi, including Aspergillus, can cause endocarditis. Prosthetic valve endocarditis due to Aspergillus was first reported involving a Starr-Edwards mitral prosthesis and subsequently reported on mechanical prostheses in both the mitral and aortic positions. Two authors have previously reported Aspergillus endocarditis of bioprosthetic valves in the aortic position, however, these cases resulted in aortic regurgitation without obstruction. This is the first reported case documenting obstruction of a
bioprosthetic valve due to Aspergillus. Like other reported fungal PVE cases, this case documents the difficulty with antemortem diagnosis of fungus as the offending pathogen. Fungal infection should be considered in patients like ours who have been exposed to prolonged antibiotic therapy. Due to the prolonged time required for blood cultures to become positive and limitations of echocardiography in patients with prosthetic valves, fungal PVE is difficult to diagnose, highlighting the importance of clinical suspicion in the febrile patient, especially those with prior antibiotic therapy following valve replacement.

Like native valve endocarditis, most fungal PVE results in valvular destruction and regurgitation, but large fungal vegetations may obstruct flow.\textsuperscript{5,7} Even though most prosthetic valves have a smaller effective orifice area than physiologically expected, yielding a small residual transvalvular gradient, this gradient generally does not result in obstructive symptoms. Normally functioning prosthetic valves should have no significant increase in transvalvular gradient for at least 10–15 years.\textsuperscript{1,2}

As noted in our patient, an abrupt increase in the prosthetic valve gradient, which can usually be documented using Doppler echocardiography, is an important clue to the presence of obstruction in PVE. Prior to echocardiography, evidence of PVE leading to obstruction was limited to clinical signs and symptoms and findings at surgery or autopsy.\textsuperscript{3,4,13–17} Echocardiographic evidence of obstruction due to PVE was initially reported using M-mode\textsuperscript{20} echocardiography. Subsequent reports have demonstrated the utility of two-dimensional echocardiography in visualizing PVE, with obstruction caused either by massive vegetations\textsuperscript{5,7,11} or abscess.\textsuperscript{10,11} Fungal PVE with obstruction has been reported with Candida species\textsuperscript{5,7} but never to our knowledge with Aspergillus.

**Conclusion**

PVE remains a significant complication of prosthetic valve surgery and is associated with excess morbidity and mortality. As the first reported case of Aspergillus infection causing PVE with obstruction of a bioprosthetic valve, this case reiterates the importance of an aggressive diagnostic approach in the febrile patient with a prosthetic heart valve, particularly in the presence of an increased transvalvular gradient.
References

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<tbody>
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<td>RA</td>
<td>(18)</td>
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<tr>
<td>RV</td>
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<tr>
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