Evaluation of Bioprosthetic Mitral Valve Failure

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Porcine bioprosthetic heart valves have been in use for the past 13 years. Although they are superior to mechanical valves in several clinical situations, their long-term durability remains in question. The slight increase in failure rate of the bioprosthetic valve starting about six years after implantation mandates close follow-up of these patients. Since primary care physicians often provide most of the medical care to patients who have undergone heart valve replacement, it is important that they recognize the signs of bioprosthetic valve failure.

Family physicians often provide most medical care to patients who have undergone heart valve replacement. There are currently two types of heart valves in widespread use: the mechanical prosthesis (eg, Starr-Edwards, Bjork-Shiley) and the porcine bioprosthesis (eg, Hancock, Carpentier). The mechanical valve is durable and time-tested, but makes necessary long-term anticoagulation therapy because of its high risk of thromboembolic complications. The porcine bioprosthesis has been shown to be superior to the mechanical heart valve in terms of embolic risk, but its long-term durability remains in question.[1] Since the porcine bioprosthesis has been in widespread use for the past 13 years, primary care physicians, cardiologists, and cardiac surgeons are now seeing more episodes of valvular degeneration. This report presents an illustrative case of mitral bioprosthetic degeneration and provides guidelines for primary care management of the patient with a porcine bioprosthesis based on a review of the recent literature.

Case Report

D.S., a 66-year-old woman with a long history of mitral valve stenosis secondary to rheumatic heart disease, first underwent mitral valve replacement in May 1975 with insertion of a Hancock porcine bioprosthesis. Her postoperative recovery was uncomplicated, and she remained in normal sinus rhythm with no evidence of congestive heart failure. D.S. did well until March 1981, when she developed atrial fibrillation. As attempts at cardioversion were unsuccessful, she was begun on warfarin (Coumadin), and her ventricular rate was controlled with digoxin. In June 1981, D.S. had a cerebrovascular accident thought to be embolic in origin and involving the left middle cerebral artery. She recovered from this event with no residual deficits. The echocardiogram performed in June 1981 showed thickening of the posteriorly oriented cusp of the mitral valve and loss of distinct stint definition. In October 1982, D.S. developed slowly progressive dyspnea on exertion and weight gain. There was no change in the cardiac examination or chest x-ray results; however, there was trace pedal edema. Furosemide was added to the medical regimen with improved exercise tolerance and decrease in the edema. In February 1983, D.S. presented at the office complaining of palpitations and increasing shortness of breath. Cardiac examination now revealed a new grade 4/6 holosystolic murmur heard over the entire precordium and left axilla. The electrocardiogram showed rapid atrial fibrillation. She was admitted to the hospital, where two-dimensional echocardiogram showed striking prosthetic valve thickening and posterior prolapse suggestive of a loose cusp. Cardiac catheterization revealed moderate to severe mitral regurgitation with normal left ventricular function. Multiple blood cultures were negative for bacterial or fungal growth. In

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Figure 1. Deteriorating mitral valve with thickened cusps and tears in upper and lower leaflets

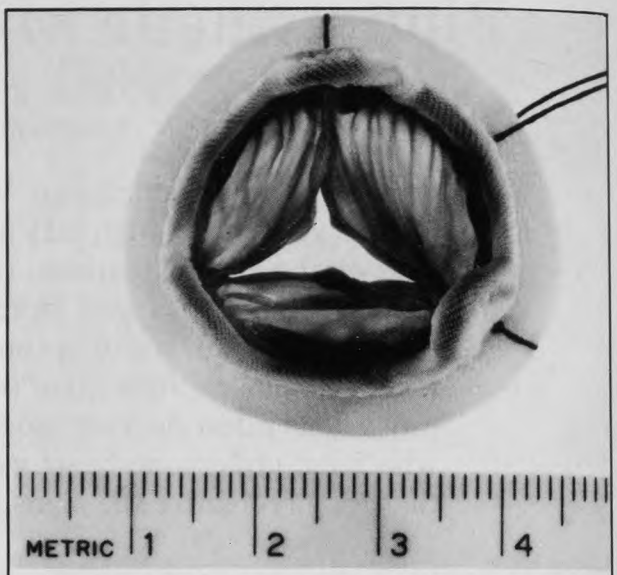

Figure 2. New Carpentier porcine bioprosthesis

March 1983, D.S. underwent replacement of a leaking Hancock mitral prosthesis (Figure 1). The valve used for replacement was a Carpentier porcine bioprosthesis (Figure 2). Her postoperative recovery again was uncomplicated. She has remained in atrial fibrillation, and because of her previous cerebrovascular accident, D.S. will be continued on warfarin indefinitely.

Discussion

This case exhibits many characteristics of the degenerating porcine bioprosthetic heart valve. Oyer et al[2] defined cardiac prosthetic valve failure as (1) the postoperative development of a new regurgitant murmur, (2) thrombotic valvular occlusion, (3) infective endocarditis resulting in reoperation or death, or (4) hemodynamic valvular dysfunction confirmed by catheterization and resulting in reoperation. In retrospect, the first evidence of valvular dysfunction occurred 20 months prior to the actual mitral regurgitation that required operation. This first sign was an embolic cerebrovascular event that occurred while the patient was on anticoagulants. Riddle et al[3] reported that endothelial cell denudation and adherence of activated platelet aggregates were the initial surface alterations that could lead to progressive degeneration of the implanted porcine bioprosthesis. It is likely these processes led to the embolic event even before hemodynamic changes in the valve had occurred. Geha et al[4] state that thromboembolic events occurred primarily in patients with mitral valve bioprosthesis who were in atrial fibrillation and on no anticoagulants. Although some clinicians[5] restrict anticoagulant therapy to patients with mechanical aortic valves or a history of emboli, if a patient develops atrial fibrillation, anticoagulants are an appropriate preventive consideration.

The second sign of porcine valve deterioration was an abnormal M-mode echocardiogram obtained at the time of the patient's cerebrovascular accident. The thickness of the bioprosthetic mitral valve can be assessed by M-mode echocardiography.[6] Alam et al[7] reported that valve thickness increases after 48 months and that the bioprosthetic valves with thickening of 3 mm or more are at a higher risk of developing clinical evidence of dysfunction. The analysis of heart sounds by phonocardiography may also be a valuable tool in detecting the degeneration of porcine bioprosthetic valves. A progression to higher frequency of the first heart sound has been found in porcine valves in the mitral position for longer than four years.[8]

Magilligan et al[8] found that the average time between increasing symptoms and replacement of a deteriorating porcine bioprosthesis ranged from

Table 1. Survival of Bioprosthetic Values

Author	Years Follow-up	Actuarial Survival Free of Bioprosthetic Failure (%)
Magilligan et al[8]	5	96.4 ± 1.3 (aortic and mitral)
	6	90.8 ± 2.4 (aortic and mitral)
	7	84.2 ± 3.7 (aortic and mitral)
Oyer et al[2]	5	98.6 ± 0.7 (aortic and mitral)
	6	94.2 ± 2.3 (aortic and mitral)
Cohn et al[10]	8	90 ± 3 (aortic and mitral)
Borkton et al[5]	5	99 ± 1 (mitral only)
	7	92 ± 4 (mitral only)
	9	70 ± 12 (mitral only)
	10	61 ± 13 (mitral only)

0 to 12 months and averaged 3.7 months. This patient first developed symptoms of congestive heart failure five months prior to her replacement operation. Several investigators[8,9] have shown that porcine bioprosthesis failure almost always causes a gradual worsening of symptoms rather than a catastrophic event. This gradual change contributes to a low mortality rate at reoperation. In contrast, the mechanical prosthesis often deteriorates suddenly with profound hemodynamic compromise.[9]

This patient was carefully evaluated for endocarditis prior to undergoing replacement of the degenerated mitral valve. In the Hancock multicenter study, 6.3 percent of patients with porcine bioprosthesis implanted prior to 1975 developed endocarditis.[10] When endocarditis develops on the bioprosthetic valve, there is greater chance of sterilizing the valve with antibiotics alone and avoiding reoperation. If valve replacement is required because of endocarditis, the operative mortality is much higher.[10]

There have been several recent reviews concerning the long-term durability of the bioprosthetic heart valve. Table 1 summarizes the data on porcine valve degeneration from four of the major studies. At the University of Vermont, there have been 297 tissue valves implanted since 1970. Of this total, only three are replacements of bioprosthetic valves that had become dysfunctional without evidence of endocarditis. In general, the bioprosthetic valve maintains satisfactory functional characteristics for at least six to eight years postoperatively in most patients. When deterioration does occur, one or all of the clinical findings reviewed in this report—thromboembolic events, thickened valve leaflets, arrhythmias, congestive heart failure secondary to mitral regurgitation, or endocarditis—may be present.

Conclusions

Table 2 presents a suggested follow-up scheme for primary care physicians who manage patients with porcine bioprosthetic heart valves. The slight increase in failure rates of these valves starting at six years after implantation mandates close surveillance. Careful evaluation will ensure timely reoperation when required and may allow intervention to reduce the risk of thromboembolic complications, endocarditis, or severe left ventricular dysfunction. Elective replacement of the

Table 2. Follow-up After Bioprosthetic Valve Surgery

Years After Implantation	Suggested Evaluation	Indications of Bioprosthetic Degeneration
6	History, physical examination	Thromboembolic events, mitral regurgitation, congestive heart failure
	Chest roentgenogram (CXR)	Enlarging cardiac silhouette, congestive heart failure
	Electrocardiogram (ECG)	Arrhythmias
	Phonocardiogram	High-frequency first heart sound
	Echocardiogram	Thickened bioprosthetic leaflets
	Office visits every 3-6 months	
7	History, physical examination	
	Phonocardiogram	
	Echocardiogram	
	Office visit every 3-6 months	
8	Cardiology and/or cardiothoracic surgery evaluation	
	Office visits every 3 months	
	Repeat ECG, CXR, echocardiogram, phonocardiogram depending on clinical status	

failing bioprosthetic valve appears to carry a low operative risk.[2,9]

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