Bioprosthetic mitral valve thrombosis in a patient in sinus rhythm after the radiofrequency maze procedure

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Bioprosthetic valves are the preferred option for mitral valve replacement in the elderly because there is a low risk of thromboembolic complications compared with mechanical valves. Current consensus is that long-term anticoagulation can be avoided in patients with bioprosthetic valve replacement because postoperative thromboembolic risk after 3 months is extremely low.¹ Once anticoagulation is withdrawn, thrombosis causing bioprosthetic valve dysfunction is extremely rare.

Clinical Summary

A 69-year-old man with a history of stroke 14 years before surgical intervention underwent mitral valve replacement with a 31-mm Hancock II porcine bioprosthesis (Medtronic, Inc, Minneapolis, Minn) and the radiofrequency maze procedure for severe mitral regurgitation and chronic atrial fibrillation. He was started on warfarin, and the international normalized ratio was kept within a therapeutic range of 2.0 to 2.5. Postoperative transthoracic echocardiography (TTE) showed normal bioprosthetic valve function with a mean mitral pressure gradient (PG) of 4.0 mm Hg.

Warfarin was discontinued at 1½ years postoperatively because there was no recurrence of atrial fibrillation on follow-up electrocardiography and 24-hour Holter tests. Aspirin was continued for minor coronary artery disease. TTE before cessation of warfarin confirmed normal mitral valve function with a mean PG of 6.0 mm Hg. Ratio of early filling velocity to atrial contraction velocity (E/A ratio) was 1.58, implying active atrial contraction. Left ventricular ejection fraction (LVEF) was 62%. Left atrial diameter was 6.0 cm.

One month after warfarin was stopped, the patient was admitted with fever, shortness of breath, and loss of appetite for 3 days. Blood culture was sterile. Chest radiography revealed acute pulmonary edema. TTE demonstrated severe mitral stenosis with a mean mitral PG of 24 mm Hg and pressure half-time (PHT) of 187 ms. The mitral valve leaflets were thickened, and leaflet motion was restricted. There was no change in LVEF or left atrial diameter. The E/A ratio was 1.4. Transesophageal echocardiography (TEE) confirmed the findings on TTE. No discrete intracardiac

thrombus was visualized. Valve thickness on TEE was 6.5 mm (Figures 1 and 2).

Intravenous unfractionated heparin and diuretics were started, and warfarin was resumed. Heparin was withdrawn when the international normalized ratio reached the therapeutic range. Hematologic work-up did not reveal a hypercoagulable state.

A repeat TTE 1 week later showed a decreased mean mitral PG of 11 mm Hg. PHT had also decreased to 107 ms. Two weeks after treatment was commenced, TTE demonstrated normal mitral bioprosthetic valve function with a mean mitral PG of 6.2 mm Hg and a PHT of 114 ms. Three months after discharge, TEE revealed a mean mitral valve PG of 4.0 mm Hg and a valve thickness of 1.2 mm.

The patient was maintained on warfarin and was well on follow-up 6 months later.

Discussion

Bioprosthetic mitral valve thrombosis is a rare cause of prosthetic valve dysfunction postoperatively. Low cardiac output,¹ large left atrium,¹ prior history of thromboembolic events,¹ low transvalvular flow caused by atrial fibrillation,^{2,3} and abnormality in blood rheology⁴ are most commonly cited as predisposing factors.

In this case there were no obvious risk factors for thrombosis: LVEF was normal, the left atrium was only mildly enlarged, and there was no hypercoagulable diathesis, although the patient did have a history of stroke 14 years before the operation. Surgical intervention had also successfully been used to treat the problem of atrial fibrillation, and he had been in sinus rhythm for 1½ years. His E/A ratio was 1.58 before warfarin was withdrawn. He was also taking aspirin, and it has been reported that aspirin alone can reduce the risk of postoperative thromboembolic complications after

Figure 1. Transesophageal echocardiogram (TEE) before treatment showing grossly thickened mitral valve.

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Figure 2. Doppler scan showing increased mean mitral pressure gradient (mean mitral pressure gradient 24 mm Hg).

bioprosthetic valve implantation in the mitral position.¹ Radiofrequency maze lesion lines were unlikely to be a source leading to propagation onto the valve leaflets because there were no intra-atrial clots and thrombus was laminar along all the valve leaflets.

Intravenous heparin was started in this patient with a plan to commence thrombolytic therapy if heparin alone was ineffective. Although thrombolytic therapy is an established first-line treat-

ment, this patient responded to heparin anticoagulation followed by warfarin. Several studies have reported good resolution of bioprosthetic thrombus after conventional anticoagulation.²⁻⁵ Concurring with these studies, we had a favorable result after 1 week of heparin and subsequent warfarinization.

Clinically significant bioprosthetic valve thrombosis is rare, although the reported incidence in the study by Oliver and colleagues⁵ is 6.2%. It should be considered a possible cause of valve dysfunction when there is increasing transvalvular pressure gradient after anticoagulation is terminated. Treatment with conventional anticoagulation is unavoidable in such a setting.

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A case of mitral valve plasty for Libman–Sacks endocarditis mimicking a cardiac tumor

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We report a patient with multiple cerebral infarcts in whom 2-dimensional echocardiography showed a large mass attached to the anterior mitral leaflet and chordae. An intracardiac tumor was diagnosed, and surgical intervention was performed. The resected mass

proved to be a Libman–Sacks vegetation on microscopy. This is frequently found in patients with systemic lupus erythematosus (SLE). Unique echocardiographic and intracardiac findings are reported here with the technique of mitral valve plasty.

Clinical Summary

A 34-year-old woman was admitted after the sudden onset of aphasia associated with mild hemiplegia of her left arm and leg. She had been receiving treatment for SLE for 9 years, but the disease had been inactive, and she had been well, except for numbness in her left hand, at 7 months before this episode. Her medications were an H₂ blocker and an oral steroid (5 mg/d prednisolone). Admission laboratory tests revealed normal serum chemistry, normal C-reactive protein (CRP) levels, no anemia, and mild thrombocytopenia (the platelet count was $55 \times 10^3/\mu\text{L}$).

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