A 20-year-old woman developed late thrombosis of a 22 mm Lillehei-Kaster mitral valve prosthesis. Despite adequate anticoagulation measures, clinical detection of significant dysfunction of this particular prosthesis is difficult because of the absence of distinctive opening and closing sounds and the radiolucent character of the disc. Early diagnostic catheterization may be warranted.

Although the majority of late complications and deaths after valve replacement are attributable to myocardial decompensation rather than to prosthetic valve dysfunction, the abrupt recurrence of symptoms after several months or years of improvement following mitral valve replacement may indicate prosthetic valve dysfunction. This is especially true for younger patients, since left ventricular decompensation secondary to coronary artery disease is unlikely in this age group. In addition, primary myocardial failure is rare after a prolonged period of improvement, unless the history suggests the onset of a new disease.

The present report describes a young patient with thrombosis of a Lillehei-Kaster mitral valve prosthesis occurring in the late postoperative period, details the rationale behind low-profile prosthetic heart valves, discusses the clinical evaluation of Lillehei-Kaster valve function, and reviews possible causes of thrombosis in this valve.

CASE REPORT

A 20-year-old woman, who had had acute rheumatic fever at ages seven and 14, underwent mitral valve replacement with a 22-mm Lillehei-Kaster prosthesis for rapidly progressive mitral stenosis at age 16. She remained asymptomatic, receiving Coumadin, until four weeks prior to her admission to University Hospital. At that time, symptoms of dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea recurred.

Physical examination revealed an obese young woman in no acute distress. The blood pressure was 110/80 mmHg, the pulse was regular at 96 beats/min, and the respiratory rate was 20 per minute. There was no jugular venous distension at 30°. A parasternal lift and prominent pulmonary artery pulsation were palpable. On auscultation, a crisp prosthetic closing sound was heard. A grade 2/6, high-pitched holosystolic murmur was heard at the apex and radiated to the axilla and the left sternal border. A grade 1/6 apical diastolic rumble was also present. The lung fields were clear. There was no hepatosplenomegaly or peripheral edema.

The electrocardiogram demonstrated normal sinus rhythm with a prolonged PR interval (0.22 second) and left atrial enlargement. Chest roentgenography showed right ventricular enlargement, left atrial enlargement, and pulmonary vascular redistribution to the upper lobes.

M-mode echocardiography confirmed right ventricular and left atrial enlargement as well as pulmonary hypertension. The left ventricular internal wall thickness and calculated ejection fraction were both normal. Fluoroscopy disclosed apparently normal motion of the prosthetic valve cage.

Cardiac catheterization data are summarized in
HEART VALVE FAILURE

Figure 1. Cardiac catheterization demonstrated a striking left ventricular-pulmonary capillary wedge pressure gradient and normal left ventricular hemodynamics. Cardiac index, 2.2 L/min/m²; systemic vascular resistance, 1899 dynes·sec/cm²; pulmonary arteriolar resistance, 169 dynes·sec/cm²; ejection fraction, 54 percent; prosthetic mitral valve area, 0.6 cm²; LV-pulmonary capillary pressure gradient, 22 mm. (From Sterling R. Reoperation for late thrombosis of Lillehei-Kaster mitral valve prosthesis. Bull Texas Heart Inst 8(2):226)

Figure 1. Minimal mitral regurgitation was seen. The levophase of the pulmonary artery injection revealed an enlarged left atrium, but no radio-lucencies suggestive of thrombus were identified in the area of the prosthesis.

The rapid onset of symptoms in this patient, combined with a high left ventricular–pulmonary capillary wedge pressure gradient, in the absence of significant mitral regurgitation, was thought to represent prosthetic valve dysfunction. Surgical exploration of the prosthetic valve was recommended.

At operation, a thrombus of recent origin was seen to extend from the posterior aspect of the prosthetic ring to the posterior left atrial wall. The sewing skirt was covered with neoendothelium, and the valve orifice was not compromised. The hinge mechanism on the ventricular surface was overgrown with a dense fibrotic pannus that limited the normal 80° excursion of the tilting disc to 15°. This marked limitation of disc motion produced the equivalence of severe mitral stenosis. The Lillehei-Kaster valve was excised and replaced with a #27 Bjork-Shiley prosthesis.

The postoperative course was uncomplicated; on the tenth postoperative day, the patient was discharged on a regimen of Coumadin. She remains asymptomatic one year after surgery.

DISCUSSION

Low-profile prosthetic heart valves, such as the Lillehei-Kaster valve3 (Figure 2), have been designed to eliminate several of the problems associated with caged-ball prostheses—difficulty of insertion in patients with small aortic roots, impedence of left ventricular ejection, and arrhythmias in patients with small ventricular cavities who undergo mitral valve insertion. Although satisfactory hemodynamic performance and a low incidence of hemolysis have been confirmed by several investigators,4-5 long-term follow-up of patients with Lillehei-Kaster valves has revealed a relatively high (up to 10 percent) incidence of valve thrombosis in the mitral position, despite adequate anticoagulation.6

Left ventricular decompensation secondary to endocardial fibrosis following mitral valve replacement has been reported as a cause of unexplained cardiac failure during the late postoperative period.7 In our patient, however, the lack of left ventricular enlargement and the normal ejection fraction made this diagnosis unlikely, even before cardiac catheterization confirmed normal left ventricular hemodynamics.

Clinical evaluation of the Lillehei-Kaster prosthesis is difficult for several reasons, and valve dysfunction is not easily detected by auscultation. In contrast to caged-ball or nontilting disc mitral valves, which have distinctive opening and closing sounds, the opening click of the Lillehei-Kaster prosthesis is heard in as few as four percent of patients and is phonocardiographically recorded in as few as 15 percent of patients with normally functioning mitral prostheses.8 Apical middiastolic murmurs have been identified in 62 percent of patients without valve dysfunction. This murmur is presumed to be related to turbulence around the disc. A single closing sound has been reported in most patients.9
Because both a middiastolic murmur and the absence of an opening click are described in normally functioning prostheses, early detection of significant Lillehei-Kaster prosthetic valve dysfunction on physical examination is difficult. In our patient, the apical holosystolic murmur suggested initially that mitral regurgitation secondary to a paravalvular leak might be responsible for the recurrence of her symptoms. However, the absence of left ventricular enlargement on physical examination, chest roentgenography, or M-mode echocardiography was inconsistent with longstanding mitral regurgitation. In addition, there was no history of endocarditis or any other entity to explain the development of an acute paravalvular leak four years after uncomplicated valve replacement. The radiographic and echocardiographic findings were, in fact, more compatible with a diagnosis of mitral stenosis than mitral regurgitation.

Although fluoroscopy documented normal motion of the prosthetic cage, adequate assessment of the Lillehei-Kaster valve disc was not possible because of its radiolucency. A definitive diagnosis in this patient could be made only by means of cardiac catheterization. In the absence of significant mitral regurgitation, the striking left ventricular-pulmonary capillary wedge pressure gradient seen at catheterization indicated prosthetic valve dysfunction, despite the absence of visible thrombus or pannus formation on angiography.

The etiology of pannus formation leading to valve thrombosis and disc malfunction has been attributed to a disproportion in size of the prosthetic valve relative to the left ventricle. Although the prosthesis may be the correct size for the mitral annulus, if the valve is too large in relation to the left ventricle, one or more of the struts can become embedded in the adjacent endocardium, thereby preventing complete opening of the disc and eventually provoking tissue ingrowth and thrombosis that further impede disc motion. In our patient, this thrombus extended into the left atrium.

Prosthetic disproportion also can occur postoperatively when an initially dilated, enlarged left ventricle becomes smaller after normal hemodynamics have been restored. To prevent this complication, the largest possible prosthesis need not necessarily be used. In the mitral position, the low diastolic gradients and large functional cross-sectional areas of the Lillehei-Kaster prostheses...
HYDATIDIFORM MOLE WITH LIVE FETUS

The incidence of a live fetus associated with a hydatidiform mole is extremely rare.1 While hydatidiform mole without a fetus in the United States has a reported incidence of 1 in 2000 pregnancies, the incidence of hydatidiform mole with a coexisting fetus varies from 1:10,000 to 1:100,000 pregnancies.2

This report describes a patient who underwent cesarean section due to painless bleeding, premature rupture of her membranes, and a transverse lie. On cesarean section a 1,200 g premature normal-appearing infant was delivered in association with unexpected molar tissue. The placenta was entirely separate from this cystic molar tissue.

Requests for reprints should be addressed to Dr. Leslie L. Alexander, Department of Radiology, Queens Hospital Center Affiliation of Long Island Jewish Hillside Medical Center, 82-88 184th Street, Jamaica, NY 11432.

CASE REPORT

A 22-year-old woman, gravida 1, para 1, was admitted to Queens Hospital Center on October 22, 1981 with premature rupture of membranes and giving a history of 28 weeks in utero gestation. Past personal and family history were noncontributory. Except for the presenting complaint, the general physical examination was otherwise unremarkable. Laboratory data revealed a hemoglobin of 12 g, a fibrinogen level of 740 mg/dL, and urinary proteins of P1: 10.3/11 and P2: 28.3/25.4.

Investigation of her pregnancy status revealed a transverse lie of the fetus which had a fetal heart rate of 169 to 180 beats per minute. Ultrasonography confirmed the presence of a single fetus in transverse lie with a biparietal diameter of 6.8 cm (about 28 weeks' gestation). The placenta was posteriorly and distally placed and extended into the

do not change significantly when the largest (25 mm) and the medium-sized (20 mm) prostheses are compared.9 There is no evidence that the accumulation of pannus around a prosthetic valve will decrease with time. The natural history is one of progressive deterioration. The onset of symptoms, months or years after placement of a prosthetic valve, should immediately suggest that mechanical dysfunction has occurred and that urgent reoperation may be necessary. Clinical and noninvasive diagnosis of such dysfunction of the Lillehei-Kaster valve prosthesis may be difficult, and early diagnostic catheterization is warranted.

Literature Cited


HYDATIDIFORM MOLE WITH A LIVE FETUS

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