SELECTED REPORTS

Successful Correction of All Mechanical Complications of Ischemic Heart Disease in the Same Patient*

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Extensive surgery for all of the mechanical complications of ischemic heart disease is feasible with early diagnosis, catheterization, and aggressive medical and surgical therapy. A patient is reported who, after recovering from cardiogenic shock, required a coronary bypass, closure of ventricular septal defects, mitral valve replacement, aneurysmectomy, and temporary pacemaker wires. The outcome was successful.

Mechanical complications of acute myocardial infarction have become increasingly important to cardiovascular specialists because of the large number of patients surviving the initial sequelae and dysrhythmias in intensive coronary care environment.1-8 Ventricular septal rupture, papillary muscular necrosis, and ventricular aneurysm of ischemic origin remain associated with a high mortality after surgical treatment,4,4 and with prohibitive mortality after medical treatment alone.5-8

The purpose of this communication is to report successful correction of a ventricular septal rupture, posterior ventricular aneurysm, papillary muscular necrosis with mitral insufficiency, complete occlusion of the right coronary artery, and significant narrowing of the left anterior descending artery in the same patient. Prior to surgery, as a consequence of his myocardial infarction, the patient's condition was also complicated by cardiogenic shock, intractable heart failure, and bradyarrhythmias requiring temporary-pacemaker wires.

CASE REPORT

One year prior to surgery, a 65-year-old white man had been admitted to an affiliated hospital with a myocardial infarction confirmed by electrocardiographic and enzymatic changes. After convalescence the patient did well until April 16, 1975, when he was readmitted with ventricular tachycardia. The ECG and enzyme levels were consistent with a new myocardial infarction. Shortly after this discharge, the patient was readmitted on May 10, 1975 with a third myocardial infarction; he was in cardiogenic shock and had pulmonary edema. On May 16, 1975, after six days of aggressive medical treatment, he was transferred to Temple University Hospital for surgical evaluation; the patient was in intractable heart failure, with a diagnosis of acute myocardial infarction.

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Figure 1. Right anterior oblique view of heart, showing large posteroinferior aneurysm (A). LA, Left atrium; and LV, left ventricle.

At transfer the patient was disoriented and had pulmonary edema, with a harsh grade 4 pansystolic murmur at the apex. His ECG showed inferior intraventricular delay and posterior myocardial infarction. After stabilization of the patient's condition, cardiac catheterization and cineangiographic studies were performed, revealing a large infero-posterior aneurysm, multiple ventricular septal defects, mitral regurgitation, complete occlusion of the right coronary artery, a significant lesion of the anterior descending artery, and bradycardia (Fig 1 to 3). The left ventricular end-diastolic pressure was 18 mm Hg, and the pulmonary arterial pressure was 30/18 mm Hg; "v" waves were measured at 18 mm Hg. A left-to-right shunt was calculated at 3.9:1, and the ejection fraction was 0.25. Due to the shunt, the arteriovenous oxygen content difference was 1.4 volumes percent, and by the Fick method the cardiac output was 12.84 L/min.

Because of persistent heart failure, surgical correction was undertaken 30 days after the last acute infarction. The findings at surgery were consistent with the data from catheterization. The anterior descending artery was revascularized using a reversed saphenous vein; then a 10 × 6 cm aneurysm

Figure 2. Left anterior oblique view of heart, showing multiple ventricular septal defects. RV, Right ventricle; and LV, left ventricle.
and fresh thrombus were excised. The mitral valve was replaced through the left ventriculotomy. The ventricular septum had four separate holes which were individually oversewn. Following this, a Teflon patch was placed over the entire surface of the ventricular septal defects and sewn into place. The edges of the left ventricle were reapproximated with a continuous suture, and temporary-pacemaker wires were placed in the myocardium.

The patient's postoperative course was uneventful, except for a minor infection of the wound, and he has been asymptomatic since the time of discharge. At the six-month follow-up, the patient was asymptomatic, with no signs or symptoms of failure, angina, or shunting.

**DISCUSSION**

Mechanical complications of ischemic heart disease are uncommon, with ventricular septal defects occurring in 0.5 to 1 percent of all myocardial infarctions and accounting for 2 percent of the deaths. The diagnosis of postinfarctional septal defects should be entertained when there is a rapid deterioration of the clinical course and sudden onset of a loud systolic murmur.

Ventricular septal defect and papillary muscular rupture causing mitral insufficiency may be accurately diagnosed at the bedside with the use of the Swan-Ganz flow-directed catheter; however, because of the constellation of potential pathophysiologic defects occurring in coronary artery disease, cardiac catheterization and coronary angiographic studies are indicated before surgery.

The incidence of acute rupture of papillary muscle secondary to myocardial infarction has not been established, but once diagnosed, such rupture has a mortality of 70 percent in the first 24 hours.

Few reports of correction of postinfarctional septal defect with mitral valve replacement or ventricular aneurysmectomy are found in the literature. There are no reports of successful surgical correction of a ventricular septal defect with mitral valve replacement, posterior aneurysmectomy, and aortocoronary bypass in the same patient.

Since the first surgical closure of a postinfarctional ventricular septal defect in 1957, experience with the repair of such lesions has been increasing. In a review of the literature by Shumaker, he found that 91 patients underwent 94 operations, with survival in 76 percent of these. Seven operations were performed within seven days or less from the time of infarction, with no survival. Increased survival rates were observed when the operation was delayed for six weeks or more. The poor results of surgery performed within three weeks after acute infarction are primarily due to technical problems when necrotic tissue is found. Buckley and his associates have reported improved results with the use of intra-aortic balloon assist prior to operation.

Many techniques of approaching the defect have been reported. Kitamura et al and Javid et al emphasized the left ventricular approach, whereas Shumacker uses the right ventriculotomy when no aneurysm has to be excised. Several other techniques have been described, but limited experience in these complicated cases makes each surgical procedure unique, depending on the size and anatomic location of the septal defect, the consistency of the tissue, the location of the ventricular aneurysm, the association of mitral insufficiency, and the extent of coronary artery involvement. In this case the ventricular septal defect was approached through an incision in the ventricular aneurysm, which allows for good exposure of the mitral valve. A low-profile prosthesis is used in such cases because of the small size of the remaining left ventricle after excision of the aneurysm. The septal defect was repaired by direct suture with felt pledgets and was reinforced with a Teflon patch prosthesis. Coronary revascularization should be done first, in order to increase the myocardial blood supply before undertaking the rest of the procedure. Obviously, early recognition, catheterization, and aggressive medical management with administration of digitalis, diuretics, oxygen, and vasoactive drugs are required if these patients are to be saved. Repair of all of the lesions should increase survival, since it augments the flow of blood to the remaining myocardium and improves deteriorating hemodynamics and myocardial contractility by closure of the ventricular septal defect, mitral valve replacement, and aneurysmectomy.

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**REFERENCES**

Posterior Leaflet Motion in Mitral Stenosis*

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The echocardiographic features of mitral valvular motion in a patient with classic rheumatic mitral stenosis are presented. Two unusual features were noted, and the importance of careful echocardiographic scanning of the mitral valve is emphasized. The theories for the classic echocardiographic abnormalities of mitral stenosis are briefly considered in light of the findings in this case.

The classic criterion developed for the echocardiographic diagnosis of mitral stenosis has been the reduced rate of early diastolic mitral valvular closure (E-F slope).1 Subsequently, it was learned that conditions other than mitral stenosis resulted in a similar reduction in the rate of closure.2 In 1972, Duchak et al3 demonstrated that in true mitral stenosis the anterior and posterior mitral valvular leaflets moved in the same direction (concordantly) in diastole, whereas in those conditions other than mitral stenosis, the posterior mitral valve leaflet maintained its normal diastolic “mirror image” (discordant) motion. Recently, several reports of discordant mitral valve motion have been documented in cases of true mitral stenosis.4,5

Presented in this report is a case of documented severe and otherwise typical rheumatic mitral stenosis with several interesting echocardiographic features. First, both discordant and concordant motions of the posterior leaflet were noted from the same patient as the mitral valve was echocardiographically scanned. Second, the anterior leaflet motion was classic for mitral stenosis with no “a” wave (in spite of normal sinus rhythm), while the posterior leaflet demonstrated a clear “a” wave.

CASE REPORT

A 30-year-old woman was referred for evaluation because of increasing dyspnea. She had undergone two mitral commissurotomies 12 and 10 years earlier and had done well until the preceding several months. Atrial biopsies taken at the time of her second commissurotomy demonstrated Aschoff’s bodies. Physical examination revealed the findings of pure mitral stenosis, and cardiac catheterization documented this clinical impression with a calculated mitral valvular area of 0.6 sq cm (Fig 1). Moderate mitral valve calcification was detected on fluoroscopic examination. Echocardiograms consistently demonstrated the features seen in Figure 2. Mitral valvular replacement was recommended, and at surgery a typical calcified mitral valve was removed. The cusps were thickened, commissural fusion was present, and shortened chordae tendineae were noted.

DISCUSSION

Echocardiographic studies have been used extensively in the diagnosis and evaluation of mitral stenosis. The major echocardiographic features are reduction in the E-F slope of the anterior leaflet and concordant motion of the anterior and posterior mitral leaflets. The importance of this finding is related to the knowledge that reductions in the E-F slope can be caused by conditions other than true mitral stenosis, such as hypertrophic cardiomyopathy, pulmonary hypertension, and others; however, Duchak et al3 demonstrated that in contrast to true mitral stenosis, these conditions were associated with discordant mitral leaflet motion. Although by no means obviating the importance of these features, several reports recently have documented mirror-image motion of the anterior and posterior mitral leaflets in true mitral stenosis.4,5

The reasons for reduced E-F slopes and for concordant mitral leaflet motion in patients with mitral stenosis remain unclear. A common theory is that the high left atrial pressure and the continuous pressure gradient

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