

Surgical treatment of postinfarction false aneurysm of the left ventricle

Twelve patients underwent surgery for repair of false aneurysms of the left ventricle. The mean interval between the myocardial infarction and the diagnosis was 19 months (range 2 to 80 months). Congestive heart failure was the most common clinical presentation. Most patients had three-vessel coronary artery disease. The false aneurysm was posterior in 10 patients and anterior in two. Three patients with posterior aneurysm had severe mitral regurgitation. Repair was accomplished by resection of the false aneurysm and primary closure of the defect in four patients and by closure with a patch in eight. Nine patients also had coronary artery bypass. Mitral valve replacement was performed in three patients who had severe mitral regurgitation before the operation and in one patient who had severe mitral regurgitation after repair of the false aneurysm and could not be weaned from cardiopulmonary bypass. There were three operative deaths and one additional death after 2 months. All deaths occurred in patients who had mitral valve replacement. Eight patients survived the operation and remained well after a mean follow-up period of 62 months. Patients with false aneurysms of the left ventricle do well after surgical repair, except when concomitant mitral valve replacement is necessary. (*J THORAC CARDIOVASC SURG* 1993;106:1189-91)

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Left ventricular free wall rupture as a result of myocardial infarction is usually fatal.^{1,2} Occasionally a patient will survive and later have a false aneurysm of the left ventricle.³⁻³² Unlike true aneurysms, false aneurysms can rupture and should be treated surgically.^{3,4}

False aneurysms of the left ventricle are characterized by a narrow neck compared with the width of the fundus. The wall of the false aneurysm is made of fibrous elements and laminated clots; it contains no myocardial tissue.^{3,4} The parietal pericardium is frequently firmly adherent to the outer surface of the false aneurysm.^{3,4} Diagnosis can usually be made before operation by echocardiography, nuclear angiography, contrast ventriculography, and magnetic resonance.^{5-11, 32, 33}

Patients and methods

A review of 412 consecutive patients who underwent repair of left ventricular aneurysms from 1978 to 1992 disclosed 12

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patients who had postinfarction left ventricular false aneurysms. These 12 patients were operated on from November 1978 to February 1992. There were 10 men and two women; mean age was 61 years (range 51 to 78 years). The mean interval between the myocardial infarction and the operation was 19 months (range 2 to 80 months). Nine patients had symptoms of congestive heart failure and three of them were in cardiogenic shock. Seven patients had angina pectoris. One patient had a thromboembolic event to his femoral artery and echocardiography revealed a false aneurysm. Nine patients were in New York Heart Association functional classification IV, one was in class III, one was in class II, and one was in class I.

All patients had coronary angiography before the operation. One had single-vessel coronary artery disease, three had double-vessel disease, and eight had triple-vessel disease. The diagnosis of false aneurysm was made before the operation in 10 patients (by echocardiography in six, radionuclide angiography in two, and contrast ventriculography in four) and during the operation in two. The false aneurysm was posterior in 10 patients and anterior in two. Six patients had mitral regurgitation, which was graded as mild in one, moderate in two, and severe in three.

An intraaortic balloon pump was inserted before the operation in three patients because of cardiogenic shock and after the operation in three because of low cardiac output. The pericardial cavity was found to be totally obliterated by adhesions in all patients. Cardiopulmonary bypass was established after freeing up only the ascending aorta and the right atrium from the pericardial adhesions. The aorta was crossclamped and the heart was arrested with cold crystalloid or blood cardioplegic solution before dissection of the false aneurysm. The adhesions between the pericardium and false aneurysm were extremely fibrous and

vascular. The aneurysm was resected in all cases and the defect in the ventricular wall was closed primarily in four patients and with a Dacron or bovine pericardial patch in eight. One patient with a posterior false aneurysm of the left ventricle was found to have a small perforation in the diaphragmatic wall of the right ventricle, which had caused a left-to-right shunt through the false aneurysm.

Three patients with posterior false aneurysm had severe mitral regurgitation, either from rupture of the posteromedial papillary muscle (two cases) or from extensive fibrosis of the posterior left ventricular wall and papillary muscle (one case). One patient with an anterolateral false aneurysm had moderate mitral regurgitation before the operation and could not be weaned from cardiopulmonary bypass after repair of the false aneurysm. Intraoperative Doppler echocardiography revealed severe mitral regurgitation. Mitral valve repair was attempted, but the valve remained incompetent and replacement was necessary to discontinue cardiopulmonary bypass. Nine patients also underwent myocardial revascularization.

Results

One patient died of low cardiac output syndrome on each of the second, fourth, and fifth postoperative days. Two of these patients were in cardiogenic shock before the operation and the third had intractable congestive heart failure. All three had posterior left ventricular false aneurysm, severe mitral regurgitation, and extensive inferior wall infarction, as well as right ventricular infarction. The patient who could not be weaned from cardiopulmonary bypass and required mitral valve replacement initially did well but later had recurrent congestive heart failure and died 2 months after the operation. The remaining patients did well. They have been followed up from 6 to 139 months (mean 62 months). There have been no late deaths. Seven patients are free of cardiac symptoms and one has stable angina pectoris.

Discussion

Rupture of the free wall of the left ventricle is common in patients who die of acute myocardial infarction.^{1,2} The pathogenesis of myocardial rupture remains uncertain, but infarction expansion after acute transmural myocardial infarction appears to play an important role.^{34,35} Patients who have infarction expansion have an increased risk of ventricular wall rupture, late left ventricular aneurysm, and death.³⁵

After a transmural myocardial infarction, blood may enter the necrotic myocardium and find its way into the pericardial cavity, causing tamponade, shock, and death. This process is frequently rapid; it may occur within hours of the infarction but more often takes place around the fourth or fifth day.² In a small number of patients, the cardiac rupture is more chronic and the epicardium may temporarily contain the hematoma; adhesions between the epicardium and pericardium begin to reinforce the

area.³ If the adhesions are strong enough to contain the rupture for a few weeks, a false aneurysm will develop.^{3,4} On histologic examination, no myocardial cells are found to be present in the wall of a false aneurysm.^{3,4} Rarely, a false aneurysm may develop after rupture of a true aneurysm.¹²⁻¹⁴ Unlike true aneurysms, which rupture only seldom, false aneurysms have a great propensity to rupture and should be surgically treated soon after diagnosis.^{3,4}

The incidence of false aneurysm after acute myocardial infarction is probably very low, considering the number of cases reported in most publications.³⁻³² A review of 28 articles on the diagnosis and treatment of false aneurysms of the left ventricle published during the past two decades disclosed a total of only 55 patients.⁴⁻³² The most common clinical presentation was congestive heart failure; angina pectoris was the second most common mode. Syncope, thromboembolic complications, and ventricular arrhythmias occurred in an extremely small percentage of the patients. Posterior false aneurysms were 3.5 times more common than anterior false aneurysms. Nevertheless, acute rupture of the anterior wall of the left ventricle is more common than rupture of the posterior wall.^{1,2} More than half of the patients had single-vessel coronary artery disease.⁴⁻³²

Our series has three distinct features. It is the largest reported, most patients had triple-vessel coronary artery disease, and four patients needed mitral valve replacement. The four patients who had mitral valve replacement died either in the hospital or early in congestive heart failure. We encountered only one other patient in the literature who had mitral valve replacement.¹⁵ That patient also did not survive operation. The combination of severe mitral regurgitation and false aneurysm of the posterior wall of the left ventricle thus appears to be associated with a high operative mortality rate. The reason is probably related to the extensiveness of the inferior wall infarction.

The other eight patients from our series did well and were alive at a mean follow-up interval of 62 months. Most other reports also indicate that patients do well after repair of the false aneurysm.

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