

Closure of postinfarction ventricular septal defect, left ventricular aneurysmectomy, and coronary artery bypass graft

Case report

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Davis, Z., Borman, J. B., Freund, U., and Stern, S. (1973). Thorax, 28, 650–652. Closure of postinfarction ventricular septal defect, left ventricular aneurysmectomy, and coronary artery bypass graft: case report. A 59-year-old patient underwent successful concomitant closure of a ventricular septal defect, left ventricular aneurysmectomy, and coronary artery bypass of the obstructed left anterior descending vessel. The operation was carried out electively three months after the myocardial infarction. Postoperative studies showed normal contractility of the left ventricle and complete closure of the ventricular septal defect. Nine months postoperatively the patient is completely well.

Rupture of the ventricular septum is one of the serious, albeit rare, complications of acute myocardial infarction. Eighty-five per cent of patients die within two months (Oyamada and Queen, 1961). In postinfarction ventricular septal defect (VSD) compounded by left ventricular aneurysm an even higher mortality may be expected. Recently, reports have appeared of combined surgical closure of the VSD and aneurysmectomy (Daggett, Burwell, Lawson and Austen, 1970; Limsuwan, Glass and Jacobs, 1970; Moss, DeWeese, Lipchick, and Olsan, 1970; Freeny *et al.*, 1971; Schlesinger, Lieberman, Landesberg and Neufeld, 1971; Skagseth *et al.*, 1971). However, we have been unable to trace a single report of concomitant closure of a postinfarction VSD, left ventricular aneurysmectomy, and revascularization of the myocardium using the saphenous vein bypass technique. Such a case is presented below.

CASE REPORT

A 58-year-old white woman was transferred to our hospital for evaluation of postinfarction congestive heart failure and a systolic precordial murmur. Three weeks earlier the patient had sustained an anteroseptal myocardial infarction and was admitted to another hospital. On admission ventricular fibrillation occurred, and successful resuscitation was carried out. Four days

later a harsh systolic murmur was first noted over the left precordium. Symptoms of congestive heart failure with recurrent episodes of atrial fibrillation developed. The patient responded well to conservative management including diuretics, digitalis, and heparin.

Examination on admission revealed a pale obese patient. Her blood pressure was 140/90 mmHg and the pulse rate was 100/minute, regular. There was moderate neck vein congestion. Moist basilar rales were audible bilaterally. There was grade 1 pitting oedema peripherally. A harsh holosystolic grade 3/4 murmur was audible over the precordium and was maximal along the left sternal border. Routine laboratory investigations were within normal limits. An electrocardiogram revealed an anteroseptal infarction. Cardiac catheterization and coronary angiography were performed. Normal pressures were measured in the right heart and pulmonary circulation. There was an elevated left ventricular end diastolic pressure (16 mmHg). A left ventriculogram showed reduced movements of the anterolateral border (aneurysm?) with good contractility of other areas of the left ventricle. There was immediate filling of the right ventricle and pulmonary artery following the left ventricular injection. A left-to-right shunt was demonstrated with a pulmonary/systemic flow ratio of 2.2:1. A grade 1 out of 4 mitral regurgitation was also noted. Selective coronary cineangiography demonstrated a 95% obstruction at the proximal third of the left anterior descending artery and a second area of severe obstruction immediately distal to the first.

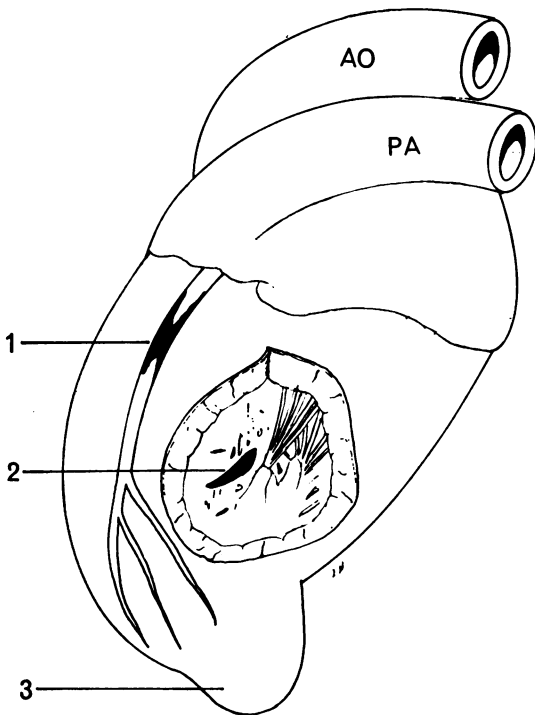


FIG. 1. Preoperative findings: 1 complete obstruction of the left anterior descending artery; 2 ventricular septal defect; 3 apical aneurysm of the left ventricle. AO = aorta, PA = pulmonary artery.

There was good distal runoff. The right coronary and the circumflex arteries were essentially normal.

In the light of the patient's stable haemodynamic condition, surgical intervention was delayed. In the interim there were periods of supraventricular arrhythmias and aberrant conduction disturbances, which responded to lignocaine and maintenance procainamide. During the sixth week in hospital the patient developed a dry cough and tachypnoea associated with right leg thrombophlebitis. Lung scanning demonstrated pulmonary embolization in the right lung and multiple small emboli to the left lung. Her pulmonary symptoms improved markedly following the administration of heparin.

Three months following the acute myocardial infarction open-heart surgery was performed. Through a median sternotomy the pericardium was opened and multiple, dense, apical adhesions were separated. A paradoxically contracting fibrotic area was seen in the apex of the left ventricle. A systolic thrill was palpable over the moderately enlarged right ventricle. The left ventricle was entered through the apical aneurysm. There was an 8 mm diameter VSD with fibrotic margins in the apical part of the septum. The defect was closed with silk mattress sutures tied over Teflon felt pledgets. The left ventricular aneurysm was excised,

and the left ventricle closed with interrupted silk sutures over Teflon felt. The proximal left anterior descending artery was bypassed, anastomosing a saphenous vein graft from the ascending aorta to a coronary vessel 1.25 mm in diameter. Total bypass time was 100 minutes.

The patient's postoperative course was essentially uneventful. On the thirteenth postoperative day repeat left ventricular injection demonstrated good ventricular contraction and complete closure of the ventricular septal defect. There was now no evidence of mitral regurgitation. The bypass graft could not be entered.

Nine months after surgery the patient is completely symptom free with no signs of congestive cardiac failure.

DISCUSSION

The majority of patients presenting with an acute VSD following myocardial infarction progress rapidly into congestive heart failure which is intractable to intensive medical management. These patients present a therapeutic and diagnostic challenge. Ideally, a complete right and left heart catheterization and coronary cineangiography should be performed immediately following clinical diagnosis. However, in practice the majority of patients are in such severe haemo-

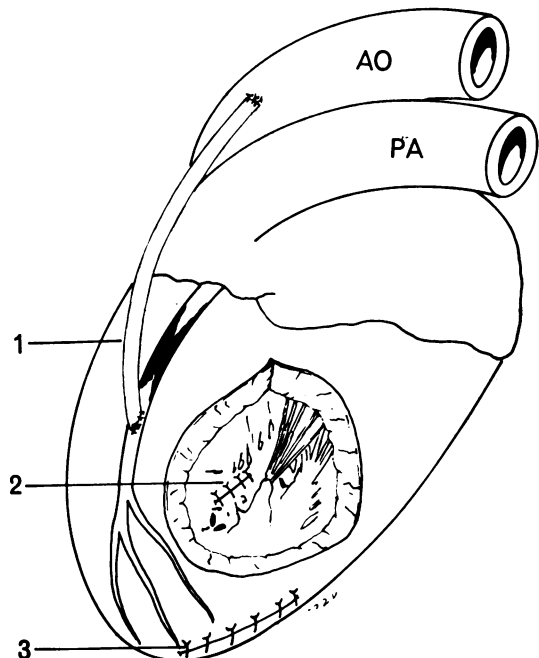


FIG. 2. Postoperative state: 1 saphenous vein bypass graft; 2 ventricular septal defect closed; 3 aneurysm excised and left ventricle reconstituted.

dynamic deficit that their condition permits the performance of limited diagnostic procedures only—right heart catheterization with the possible addition of left ventricular injection. Coronary arteriograms and subsequent coronary artery surgery are not feasible in these extremely ill patients, and emergency surgical closure of the VSD is indicated as a life-saving procedure.

The small group of patients with postinfarction VSD who respond favourably to medical treatment during the acute episode should undergo elective surgery about two months later. At this time the surgical repair of the VSD is technically much simpler due to the development of fibrosis around the defect. If coronary arteriograms are available the underlying coronary artery obstruction may now be dealt with and the appropriate bypass procedure carried out to revascularize the ischaemic area of myocardium. This should diminish the reported late mortality due to recurrent infarction in those patients who have undergone successful closure of a postinfarction VSD (Cleland, Goodwin, McDonald, and Ross, 1969).

The patient reported above is one of six patients with postinfarction VSD seen at the Hadassah University Hospital during the last year. We have had only one more survival, that of a 71-year-old woman who underwent emergency right and left heart catheterization during intractable cardiac failure followed by immediate successful closure of the VSD. All the other patients in intractable heart failure deteriorated rapidly and died before surgery could be carried out.

It should be emphasized that the patient reported above is the only one in our series who responded to conservative therapeutic measures, permitting delay of the surgical procedure until the optimal time has elapsed. In this patient three months following the infarction the aneurysm of the left ventricular apex could be resected, the VSD closed, and the severely obstructed left anterior descending artery bypassed with a saphenous vein graft. Complete repair of all

diagnosed pathologies facilitated, in our opinion, the smooth postoperative course and complete clinical recovery.

The disappearance of the mitral regurgitation following cardiac surgery, as demonstrated by left ventricular injection, may be ascribed to the improved left ventricular function following myocardial revascularization by the bypass technique and left ventricular aneurysmectomy.

We thank Dr. D. Brunner, of the Donolo Hospital, Jaffa, for referring this patient to us.

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