

Papillary muscle rupture following myocardial infarction

Successful treatment by resection of akinetic left ventricular area, mitral valve replacement, and aorta to coronary artery bypass graft

D. A. MARY, B. C. PAKRASHI, and MARIAN I. IONESCU

Department of Cardiothoracic Surgery, The General Infirmary, Leeds

A patient is reported in whom rupture of the posterior papillary muscle of the left ventricle occurred following myocardial infarction. The intractable pulmonary oedema that followed responded to the combined surgical approach of mitral valve replacement, resection of the akinetic area of the left ventricular wall, and saphenous vein aortocoronary bypass. This combination of surgical procedures has not been reported to date, and was considered to have preserved adequate left ventricular performance.

Papillary muscle rupture following myocardial infarction is rare (Sanders, Neuburger, and Ravin, 1957; Cederqvist and Soderstrom, 1964). The sudden occurrence of mitral incompetence in an already damaged left ventricle might lead to fatal pulmonary oedema (Davison, 1948; Craddock and Mahe, 1953; Sanders *et al.*, 1957; Cederqvist and Soderstrom, 1964; Heikkila, 1967). Survival for a longer time, however, albeit with cardiac failure, is possible when the left ventricular function is compromised to a lesser degree or when rupture of the papillary muscle involves one of its heads only (Breneman and Drake, 1962; Adicoff, Alexander, Ferguson, and Kelly, 1963; Horlick, Merriman and Robinson, 1966; de la Torre, Linhart, and Bartley, 1967; Morrow *et al.*, 1968; Braunwald, 1969). In such patients surgical correction of the ensuing haemodynamic abnormalities by mitral valve replacement with or without resection of the ventricular dyskinetic area has been reported (Austen, Sanders, Averill, and Friedlich, 1965; Holloway, Whalen, and McIntosh, 1965; de la Torre *et al.*, 1967; Morrow *et al.*, 1968; deBusk *et al.*, 1970; Hatcher *et al.*, 1970; Schimert *et al.*, 1970).

This is a report of a case with myocardial infarction and a ruptured left ventricular papillary muscle leading to intractable pulmonary oedema. Resection of an akinetic area of the posterior wall of the left ventricle, mitral valve replacement, and saphenous vein aorto-coronary bypass grafting considerably improved the patient's condition.

CASE REPORT

A 57-year-old labourer, previously fit with no known history of cardiac lesion, was admitted to hospital on 16 January 1971 after sudden retrosternal chest pain and a transient loss of consciousness. A diagnosis of acute myocardial infarction was made. The electrocardiogram (ECG) revealed atrial fibrillation and changes of acute inferolateral myocardial infarction (Fig. 1a). The patient recovered and was discharged after six weeks. No cardiac murmurs were heard.

On 18 March 1971 a localized apical pansystolic murmur was heard and at that time the patient's only symptom was mild dyspnoea. On radiography the cardiothoracic ratio and pulmonary vascularity were within normal limits. The murmur was attributed to a probable papillary muscle dysfunction and mild mitral incompetence. The patient was able to resume work in April 1971.

On 15 December 1971 the patient was readmitted following a sudden pain across the chest and severe dyspnoea. The patient was orthopnoeic and in atrial fibrillation with a ventricular rate of 70 beats/minute, the blood pressure was 130/70 mmHg. A musical apical pansystolic murmur of grade 3/6 radiating to the axilla was heard. There was an audible and palpable gallop. Pulmonary congestion with normal cardiothoracic ratio were found on chest radiography. Serum aspartate aminotransferase was 372 units.

Following admission the patient had repeated episodes of pulmonary oedema which responded only partially and temporarily to medical therapy. Ventricular fibrillation and cardiac arrest occurred twice and sinus rhythm was established by DC shock. The ECG revealed sinus rhythm and changes of old

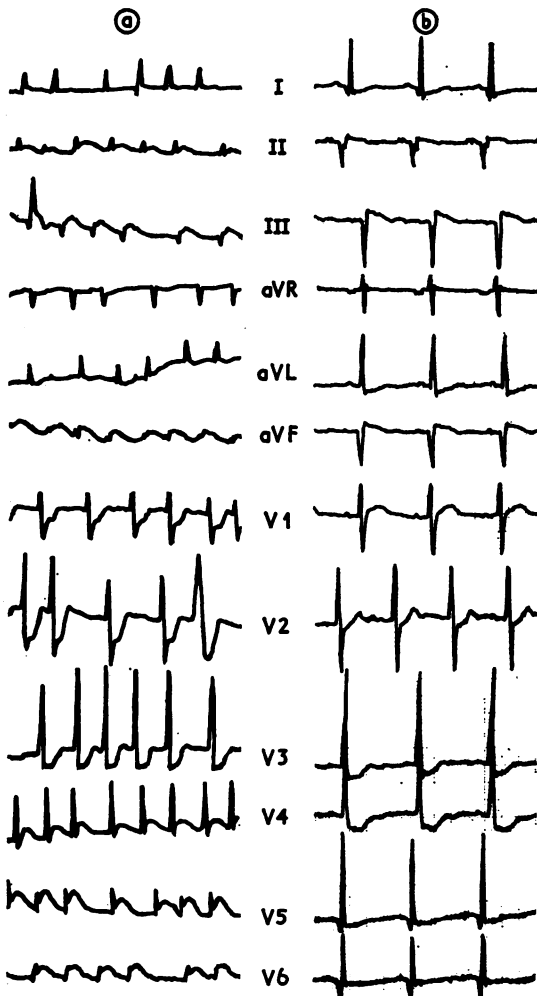


FIG. 1. [†]Electrocardiogram recorded (a) on the day of the first myocardial infarction (Jan. 71) and (b) two days following readmission (Dec. 71).

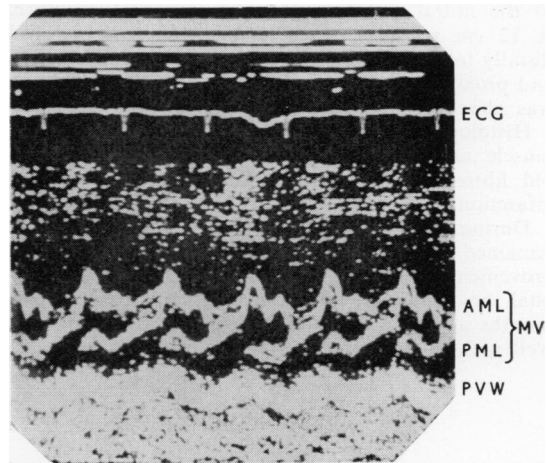


FIG. 2. Mitral echogram obtained from the fourth left intercostal space, 2 cm from the midline. The total amplitude (20 mm) and E-F speed (162 mm/sec) are within normal limits. Both leaflets remain in apposition during ventricular systole. ECG=electrocardiogram; MV=mitral valve; AML=anterior leaflet of the mitral valve; PML=posterior leaflet of the mitral valve; PVW=posterior wall of the left ventricle.

in view of the patient's previous ventricular fibrillation.

The confirmation of mitral incompetence in the presence of refractory pulmonary oedema and a relatively small left ventricle indicated that the mitral regurgitation, presumably due to rupture of a papillary muscle, was the main cause for the patient's symptoms.

On 15 March 1972 the chest was opened through a median sternotomy. The left ventricle was slightly enlarged and its posterior wall was adherent to the pericardium. A fibrosed, thin, and akinetic area was occupying the greater part of the left ventricular posterior wall. The coronary arteries were examined. The right coronary artery was rigid, fibrous, and completely obstructed in its proximal third. The distal part beyond the acute marginal branch was soft and patent. No occlusion was felt in the rest of the main coronary arteries. On full cardiopulmonary bypass the left atrium was opened and the mitral valve was found to be grossly incompetent due to complete rupture of the posterior papillary muscle. The incompetence was mainly at the posteromedial commissure. An area of 8×5 cm (representing about 20% of the left ventricular wall) of scar from the posterior wall of the left ventricle was resected, preserving a rim of fibrous tissue for suturing. The margins of the gap were securely approximated using a double layer of reinforced interrupted and continuous sutures. The mitral valve was removed and a No. 2 Silastic ball Starr prosthesis was attached

inferolateral myocardial infarction (Fig. 1b). Fluoroscopic examination revealed left atrial and left ventricular enlargement and calcification of the acute marginal branch of the right coronary artery. Mitral echography demonstrated normal valve excursions and E-F speed with no evidence of leaflet prolapse into the left atrium during ventricular systole (Fig. 2). On cardiac catheterization the left ventricular end diastolic pressure was 26 mm of mercury. Left ventriculography demonstrated gross mitral incompetence and reduced excursions of left ventricular wall movements. Although selective coronary arteriography is essential to outline coronary artery pathology, it was not considered feasible at the time of catheterization

to the mitral annulus using one continuous suture. A 12 cm segment of saphenous vein was sutured distally to the patent part of the right coronary artery and proximally to the ascending aorta. The operation was concluded without incidents.

Histological examination of the excised papillary muscle and left ventricular scar revealed extensive old fibrosis with no evidence of recent myocardial infarction.

During the first six postoperative days the patient remained in a low cardiac output state. Gradual improvement followed and when discharged from hospital he required only anticoagulant therapy. Six months after the operation the patient has remained well with no relevant cardiac symptoms.

DISCUSSION

Following myocardial infarction, the estimated incidence of rupture of a left ventricular papillary muscle is less than 1% (Cederqvist and Soderstrom, 1964). Involvement of the posterior papillary muscle usually results from occlusion of a branch of a dominant right coronary system (Heikkila, 1967). The anterior papillary muscle is less frequently affected (Craddock and Mahe, 1953; Sanders *et al.*, 1957; Sanders, Armstrong, Willerson, and Dinsmore, 1971).

Reported cases of papillary muscle rupture complicating myocardial infarction have been reviewed (Davison, 1948; Craddock and Mahe, 1953; Sanders *et al.*, 1957). The sudden development of pulmonary oedema and the emergence of an apical systolic murmur during the first two weeks after the onset of myocardial infarction were found to be characteristic features in the diagnosis of mitral incompetence due to rupture of a papillary muscle (Sanders *et al.*, 1957; Breneman and Drake, 1962; Adicoff *et al.*, 1963). Unless further myocardial infarction occurs, and apart from the subsequent development of left atrial or ventricular strain, electrocardiography was not reported to show specific changes (Breneman and Drake, 1962; Phillips, De Pasquale, and Burch, 1963). Chest films might reveal pulmonary vascular congestion with a near normal left atrial size and normal cardiothoracic ratio (DeBusk *et al.*, 1970; Sanders *et al.*, 1971).

Echocardiography was not found useful in the diagnosis or assessment of the severity of mitral incompetence (Effert, Bleifeld, Deupmann, and Karitsiotis, 1964; Winters *et al.*, 1967; Wharton, 1969). Prolapse of the mitral valve leaflets during ventricular systole, or an at random movement of either leaflet, was demonstrated in patients with diseased or ruptured subvalvular anchoring structures (Dillon, Haine, Chang, and Feigenbaum,

1971; Duchak, Chang, and Feigenbaum, 1972). These features were not found useful in the patient reported here, probably because of the localization of the site of incompetence at the postero-medial commissures, which was away from the central part of the valve exposed to the ultrasonic beam. Cardiac catheterization and angiography confirm the presence of mitral incompetence and assess the degree of regurgitation and the movement of the ventricular wall.

Mitral valve repair (Adicoff *et al.*, 1963; Horlick *et al.*, 1966) and mitral valve replacement for such lesions have been reported (Austen *et al.*, 1965; Holloway *et al.*, 1965; de la Torre *et al.*, 1967; Morrow *et al.*, 1968; deBusk *et al.*, 1970; Hatcher *et al.*, 1970; Schimert *et al.*, 1970). The results of valve replacement were shown to depend on the extent of impairment of left ventricular function by the myocardial infarction (Morrow *et al.*, 1968; deBusk *et al.*, 1970).

Aorta to coronary artery saphenous vein bypass grafts in patients with ischaemic heart disease were followed by improvement in cardiac symptoms and left ventricular performance (Favaloro, 1970; Manley, Johnson, Flemma, and Lepley, 1972; Morris *et al.*, 1972). In patients with severe congestive heart failure, particularly when associated with papillary muscle dysfunction and mitral incompetence, no improvement was obtained by myocardial revascularization alone or when combined with mitral valve repair (Spencer *et al.*, 1971). Resection of a left ventricular scar combined with coronary artery bypass did not produce good results in patients with extensive coronary heart disease (Kouchoukos, Doty, Buettner, and Kirklin, 1972; Spencer, 1972). In patients with a lesser degree of myocardial damage, the combination of saphenous vein graft and resection of a left ventricular scar might improve or preserve myocardial performance (Milstein, 1970). Improvement in the clinical state and left ventricular function was observed after resection of an akinetic area in patients with congestive heart failure following myocardial infarction and in patients who had additional mitral valve replacement for papillary muscle dysfunction (Heimbecker, 1969; Schimert *et al.*, 1969; Schimert *et al.*, 1970). Preoperative angiographic assessment of left ventricular wall movement and coronary artery morphology would help to select those patients who may benefit from myocardial revascularization and resection of a non-contracting area of the left ventricle (Spencer, 1972).

In our patient such a combination of procedures might have obviated the postoperative residual left ventricular failure and ischaemia. The

survival for three months after the rupture of a papillary muscle indicated that left ventricular function was not severely compromised. The quality of the coronary arteries was assessed during surgery. One can only presume that the view taken at surgery—that the left coronary arteries were 'normal'—was true in view of the fact that the patient has had neither recurrence of anginal pain nor other cardiac symptoms.

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