

Mitral stenosis with free-floating left atrial thrombus and recurrent systemic embolisation

A case report

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Summary

A 64-year-old white woman with moderately severe rheumatic mitral stenosis complicated by atrial fibrillation and recurrent systemic embolisation to the brain was found at operation to have a large 'free-floating' left atrial thrombus, as well as multiple left atrial appendage thrombi. These had not been detected by echocardiography. She also had significantly reduced left ventricular contractility on cine angiography, and right coronary artery atherosclerosis. She underwent successful mitral valve replacement and excision of the left atrial appendage.

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Case report

A 64-year-old white woman was known to have had chronic rheumatic mitral stenosis and episodes of supraventricular arrhythmia since early adulthood, possibly due to acute rheumatic fever during childhood. Apart from infrequent palpitations she was quite asymptomatic up to 1962, when she suffered a left-sided transient ischaemic attack. In November 1979 she was referred to the Cardiac Clinic at Tygerberg Hospital, where she was seen by one of the authors (J.Z.P.); she complained of grade I dyspnoea and had been given digoxin, a diuretic, prazosin and disopyramide. Beta-blockers had been discontinued on account of bronchospasm. She had no overt signs of heart failure, and was in sinus rhythm with a heart rate of 66/min and a blood pressure 170/90 mmHg. There was no cardiomegaly or pulmonary hypertension. A slightly accentuated first heart sound was heard, as was a clear opening snap quite late following the second heart sound and succeeded by a short grade 1/4 intensity mid-diastolic murmur with no presystolic accentuation. A resting ECG showed infrequent unifocal ventricular extrasystoles. Chest radiography demonstrated slight straightening of the left heart border, no obvious mitral valve calcification, and clear lung fields. A diagnosis of mild rheumatic mitral stenosis was made and the patient was discharged on the same drug therapy.

In 1980 she began experiencing mild classic angina pectoris for which nitrates were given with good effect. In December 1984 she suffered an embolus to the right brachial artery necessitating embolectomy. In July 1985 she again suffered an acute embolus, this time to the left popliteal artery, which was successfully managed by embolectomy, and she was then started on oral anticoagulation. Her angina pectoris was becoming unstable and her effort tolerance had decreased (grade II dyspnoea). Because of this deterioration she was admitted to Tygerberg Hospital in early August 1985. Clinical examination revealed controlled atrial fibrillation, a blood pressure of 130/80 mmHg, no cardiomegaly and no evidence of cardiac failure. There was a moderately loud first heart sound, a normal second heart sound and a closely spaced opening snap followed by a short mid-diastolic murmur. The pulmonic component of the second heart sound at the base was slightly accentuated. The physical findings were those of mild-to-moderate and fairly mobile mitral stenosis complicated by controlled atrial fibrillation. A resting ECG showed a borderline left ventricular (LV) enlargement and digitalis effect. A chest radiograph delineated an enlarged cardiac silhouette with an LV configuration but no features suggestive of significant mitral stenosis. The lung fields contained a few Kerley-B lines but nothing else abnormal.

Echocardiographic assessment

All standard M-mode and two-dimensional recordings were obtained. Both M-mode and two-dimensional examination confirmed the presence of severe mitral valve stenosis with patchy calcification of both leaflets and severe involvement of the subvalvular apparatus. The LV end-diastolic dimension was greater than would have been expected in a patient with pure, severe mitral stenosis, and mild ventricular decompensation was present. These findings were thought to represent the presence of mitral valve incompetence and/or myocardial dysfunction. No obvious stationary or mobile thrombi could be detected in the enlarged left atrium by either examination, nor were other cardiac thrombi recorded. The M-mode tracing of the pulmonary valve confirmed the clinical impression of pulmonary hypertension.

Cardiac catheterisation

The intracardiac pressures and cardiac indices demonstrated moderately severe pulmonary hypertension, biventricular cardiac failure and a moderate degree of mitral stenosis (mean diastolic gradient 7 mmHg) (Fig. 1). LV cine angiography in the right anterior oblique (RAO) projection delineated moderately severe generalised hypokinesia with possible LV mural thrombi, and a severely diseased non-calcified mitral valve with mild mitral insufficiency (Fig. 2). Aortic cine angiography in the left anterior oblique (LAO) view showed a normal aortic valve and arch. Selective coronary arteriography demonstrated a non-dominant right coronary artery (RCA) with 95% diameter stenosis in the first part (Fig. 3), and a normal left coronary artery.

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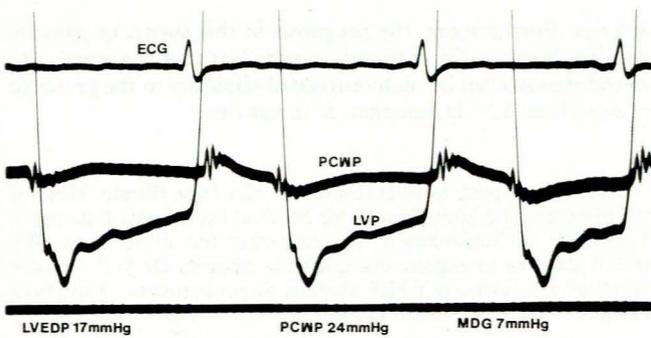


Fig. 1. Simultaneous left ventricular pressure (LVP) and pulmonary capillary wedge pressure (PCWP) measurement. Atrial fibrillation is present. There is a mean diastolic gradient (MDG) of 7 mmHg across the mitral valve (LVEDP = left ventricular end-diastolic pressure).

Further management

In view of the presence of severe mitral stenosis (underestimated clinically, probably on account of the unexpected LV dysfunction) and the history, the patient underwent an emergency operation on 15 August.

On incision of the left atrium, free-floating thrombus (approximately 3 x 3 cm) was seen, too large to traverse the mitral valve opening. The left atrial appendage was full of thrombus loosely attached to the endocardium, but there were no mural thrombi in the left ventricle. The mitral valve was grossly fibrotic, not macroscopically calcified, and severely stenotic. A fresh thrombus was present in the anterosuperior commissure of the mitral valve. The free-floating thrombus in the left atrium was removed and this chamber thoroughly irrigated. The left atrial appendage was excised together with the thrombi *in situ*. The mitral valve was excised *in toto* and replaced by a No. 31 Carpentier-Edwards bioprosthesis.

The patient made an uneventful recovery. On discharge she was still in atrial fibrillation with a slow ventricular response. It was decided to maintain the digoxin and oral anticoagulation therapy for a few months. At her last visit to the Cardiac Clinic she was asymptomatic, with a normally functioning mitral bioprosthesis.

Discussion

The interrelationship between clinically insignificant rheumatic mitral stenosis, recurrent cerebral embolisation, atrial fibrillation, poor LV function on cine angiography, the failure of both M-mode and two-dimensional echocardiography to detect

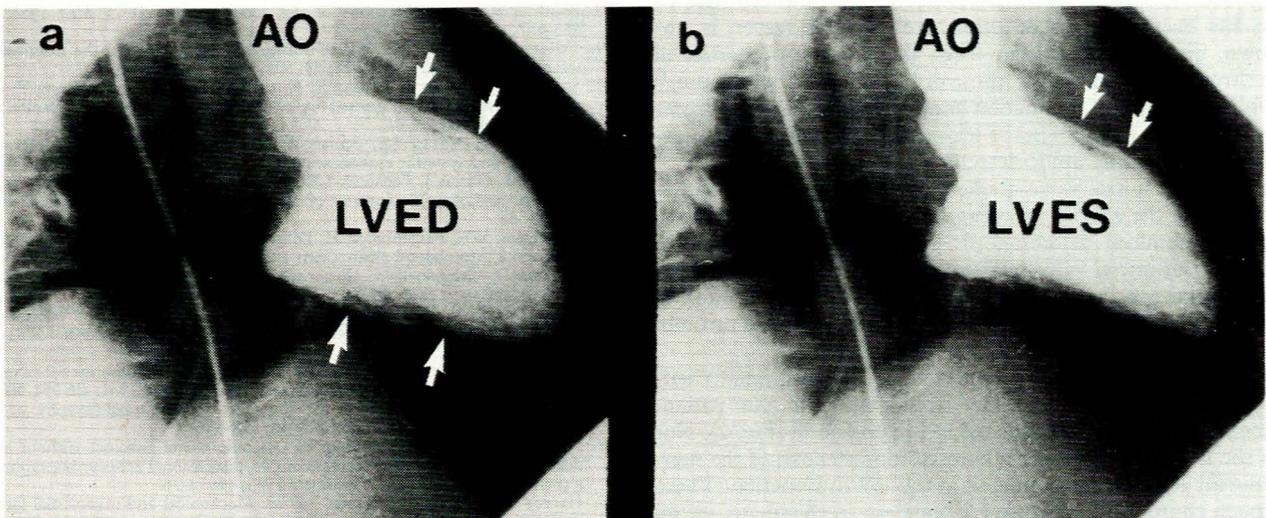


Fig. 2. LV cine angiograms in the RAO projection. The ventricle is moderately dilated and displays generalised hypokinesia. Possible LV mural thrombi (arrowed) are also seen. The mitral valve is not calcified but is severely diseased, and there is mild mitral insufficiency (LVED = left ventricle at end-diastole; LVES = left ventricle at end-systole; AO = ascending aorta).

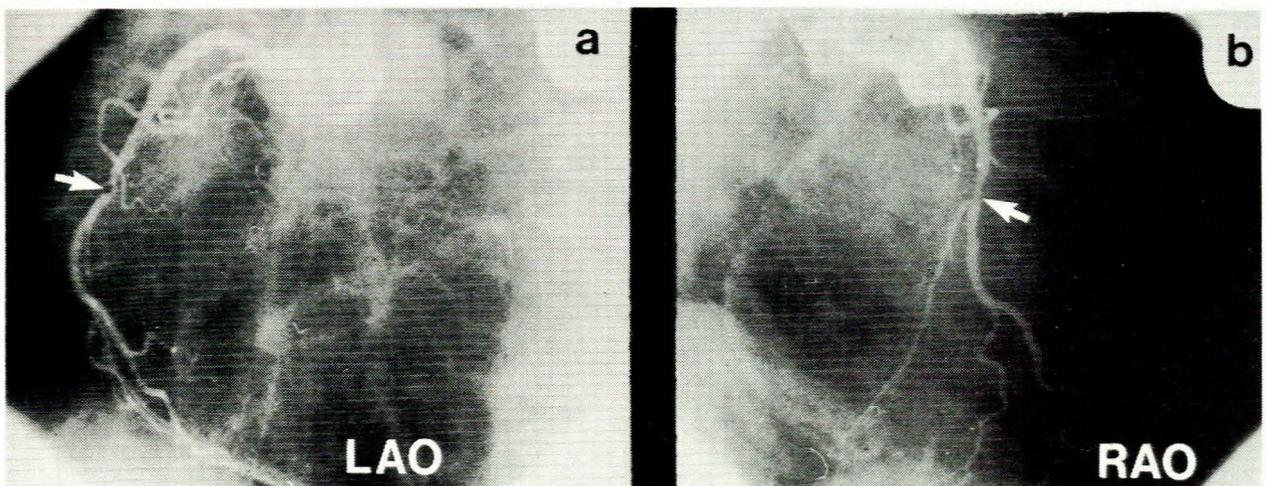


Fig. 3. RCA cine angiograms in the LAO and RAO views. The vessel is non-dominant and has a 95% diameter stenosis (arrowed) in its first part.

the large free-floating thrombus in the left atrial body and the multiple left atrial appendage thrombi, and the concomitant presence of severe single-vessel coronary atherosclerosis and classic angina pectoris is quite intriguing.

Systemic embolisation is a most significant and often quite devastating complication of rheumatic mitral stenosis,^{1,2} and may well be the presenting clinical manifestation. Patients with atrial fibrillation are at risk of developing systemic emboli, particularly if the cardiac output is significantly reduced and there is associated dilatation of the left atrial appendage. Whether increased left atrial dimension is significantly related to the frequency of intra-atrial mural thrombus formation and subsequent systemic embolisation is controversial. The degree of mitral stenosis need not always be haemodynamically severe to be associated with systemic embolisation, and the occurrence of the latter in the presence of sinus rhythm may imply underlying infective endocarditis. However, recurrent systemic embolisation, as in our patient, is an indication for urgent anticoagulation therapy as well as mitral valve surgery (valvotomy or valve replacement).

The most interesting feature of our case was the failure of echocardiography to detect the large 'free-floating' thrombus in the left atrium. Firstly, free-floating left or right atrial thrombi are exceedingly rare.³⁻¹⁴ Secondly, the inability to diagnose this complication of mitral stenosis and estimate its size has been experienced elsewhere.^{15,16} The reason is unknown, but error may occur when the acoustic properties of the thrombus are similar to those of blood and endocardium,¹⁷ or in the absence of lamination and fibrosis within the thrombus; moreover, thrombi in the left atrial appendage are inaccessible to echocardiographic detection.

A diastolic mitral valve gradient recorded on cardiac catheterisation in the absence of mitral valve stenosis may be due to either a submitral membrane or large vegetation attached to the posterior mitral valve leaflet and can be diagnosed by two-dimensional echocardiography,¹⁸ which is superior to the M-mode technique in the diagnosis of free-floating atrial thrombi or other intra-atrial masses.

Poor LV function is not expected in uncomplicated rheumatic mitral stenosis, but cine angiography in our patient demonstrated significant generalised hypokinesia (Fig. 2). Nor can the isolated and severe atherosclerotic stenosis of the non-dominant RCA be incriminated in the LV dysfunction. There is much confusion regarding LV function in rheumatic mitral stenosis.¹⁹⁻²¹ Subramanian *et al.*²² analysed the papillary muscle attached to diseased rheumatic mitral valve excised at the time of mitral valve replacement and demonstrated the presence of a 'micro-arteriopathy' of the intramyocardial coronary arteries in patients with LV dysfunction but normal epicardial coronary

arteries. Furthermore, the prognosis in this subset of patients was much worse. Of importance is the fact that the severity of mitral stenosis can be underestimated clinically in the presence of significant LV dysfunction, as in our case.

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