Ventricular septal defect and mitral regurgitation secondary to myocardial infarction

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A patient is described who developed both a ventricular septal defect and mitral regurgitation due to papillary muscle dysfunction secondary to myocardial infarction. Only one other case with these features has been described.

Both ventricular septal defects and mitral regurgitation are well-recognized complications of myocardial infarction. The former occurs in 0.5-1.0 per cent of all infarcts (London and London, 1965; Ross and Young, 1963). Mitral regurgitation may be due to either fibrosis or rupture of a papillary muscle (Friedberg, 1966). Its incidence, following infarction, is uncertain.

Case report

A Pakistan salesman, aged 50, was admitted to St. Thomas's Hospital on 4 September 1968 in cardiac failure. He had led a full and active life until 18 months previously when he noticed the gradual onset of exertional dyspnoea and ankle swelling, and one month before admission he had developed paroxysmal nocturnal dyspnoea. He had no history of rheumatic fever and he had had a thorough medical examination in 1963 when no mention was made to him of any heart murmurs. He had never suffered chest pain.

On admission he had signs of gross congestive cardiac failure; the jugular venous pressure was raised 10 cm, there was bilateral pitting ankle oedema, bilateral basal crepitations, and the liver, which was tender and non-pulsatile, extended 4 finger breadths below the right costal margin. The pulse rate was 96/min and he was in sinus rhythm. There was left ventricular hypertrophy (grade 3/4) and an atrial impulse (grade 3/4), together with a systolic thrill at the left sternal edge. On auscultation there was a pansystolic murmur (grade 3/4) maximal at the left sternal edge, which radiated to the left axilla; the second sound was normal, but there was a third sound (grade 2/4) at the apex, which was followed by a soft diastolic murmur and a loud left-sided fourth sound (grade 3/4). There was no clubbing, but there were several splinter nail haemorrhages, a palpable spleen tip, and three small retinal haemorrhages in the left eye. The urine contained

a trace of protein and microscopical haematuria. His temperature, which was 37.8°C on the day of admission, was subsequently normal. The haemoglobin was 18.8 g/100 ml, PCV 54 per cent, erythrocyte sedimentation rate 3 mm in 1 hour, white blood cells 8100/min³, SGOT 16 units, and blood urea 34 mg/100 ml. A chest x-ray showed moderate cardiomegaly. There was enlargement of the ventricular mass (grade 3/4), the pulmonary artery (grade 1/4), and the left atrium (grade 2/4), and prominence of the pulmonary veins of the upper lobes together with patchy consolidation throughout both lung fields compatible with pulmonary oedema. There was no evidence of valvular calcification. The electrocardiogram (Fig. 1) showed left ventricular hypertrophy and previous transmural diaphragmatic myocardial infarction. A clinical diagnosis of either mitral regurgitation or a ventricular septal defect secondary to myocardial infarction was made.

He was treated initially with bed-rest, digitalization, and oral diuretics. Four duplicate blood cultures were negative and there was no serological evidence of infection with *Coxiella burnetii*. However, in view of the clinical manifestations of peripheral embolization, the patient was treated with an eight-week course of penicillin and streptomycin. During this period the signs of cardiac failure regressed and the spleen became impalpable. Further splinter nail haemorrhages were observed up until the end of his antibiotic course, but not subsequently.

Fourteen weeks after admission the patient underwent cardiac catheterization. Pressures throughout the right heart were normal, but there was a 20 per cent step up in oxygen saturation in the right ventricle. There was also a loud pansystolic murmur in the body of the right ventricle which, unlike any ventricular septal defect murmur ever recorded by us, was not conducted up to the pulmonary artery. The left heart was catheterized retrogradely across the aortic valve (which was normal) and the left ventricular end-diastolic pressure was raised (16 mmHg; zero at

FIG. 1 Electrocardiogram shortly after admission showing evidence of an old diaphragmatic infarction and left ventricular hypertrophy.

sternal level). Moderate (grade 2/4) mitral regurgitation and filling of the right ventricle was shown on the left ventriculogram (Fig. 2).

The patient gave no history of chest pain, but the recent development of the murmurs and the electrocardiographic changes led to the conclusion that the defects were ischaemic rather than congenital in origin.

The patient at this stage was out of overt cardiac failure, but was dyspnoeic on mild exertion. He was discharged from hospital on 11 January 1969, but was readmitted two days later, again in heart failure. It was therefore decided to attempt operative repair of his cardiac lesions.

At operation under cardiopulmonary bypass, both ventricles were found to be enlarged, and the left atrium dilated. There was a localized thrill at the apex of the right ventricle, and at inspection through the left atrium the mitral valve and its subvalvular apparatus appeared to be normal, and the valvular incompetence was wrongly attributed solely to the dilatation of the valve ring which was considerable. The ventricular septal defect was occluded with a Teflon felt patch and the mitral ring plicated.

The patient's immediate postoperative condition was satisfactory, but after 48 hours his ventricular rate fell to 40/min despite increasing doses of intravenous isoprenaline, and endocardial pacing became necessary. On 31 January 1969 (3 days after operation) he developed ventricular fibrillation which was treated successfully, but his electrocardiogram subsequently suggested

further diaphragmatic infarction. Over the next three days his rhythm alternated between atrial fibrillation and flutter, with varying degrees of block. On 3 February 1969 (6 days after operation) he again developed ventricular fibrillation. Repeated attempts at resuscitation were unsuccessful.

At necropsy, the heart weighed 790 g. There was biventricular hypertrophy and an aneurysm involving the inferior portion of the posterior half of the interventricular septum. The two chambers communicated via a defect at the base of this aneurysm and the site of the lesion accounts for the unusual localization of the intracardiac murmur. The posteromedial papillary muscle was fibrotic and distorted, and there was moderately severe atheroma of the coronary arteries. The lungs were congested and there were healed areas of infarction in the spleen and the upper pole of the right kidney. There was widespread atheroma of the aorta and both internal carotid arteries.

FIG. 2 Left ventricular cineangiogram showing systolic filling of both the left atrium (LA) and the right ventricle (RV). A = aorta; LV = left ventricle; S = interventricular septum.

Discussion

There can be no doubt that this patient developed a ventricular septal defect and papillary muscle dysfunction after a myocardial infarct. In the absence of any history of cardiac pain, it is impossible to assess exactly how long either defect was present, but it seems likely that the sequence of events was as follows: an extensive 'silent' myocardial infarct was followed by the formation of a septal aneurysm and a ventricular septal defect which at that stage was not of haemodynamic significance; the papillary muscle dysfunction led to increasingly severe mitral regurgitation. Left ventricular function was also reduced and the combination of these three abnormalities gave rise to his progressive

symptoms and signs of cardiac failure. It is difficult to ascribe the cause of this patient's peripheral emboli with certainty. On admission he had splinter nail haemorrhages, a palpable spleen, microscopical haematuria, and retinal haemorrhages. New splinter haemorrhages were seen during his course of antibiotic therapy but none subsequently, and at necropsy healed infarcts were found in the kidney and spleen. These peripheral emboli could either have arisen from subacute bacterial endocarditis or from thrombus in the ventricular aneurysm. On balance, the latter is the most likely, for though the absence of positive blood cultures and apyrexia are well documented in endocarditis (Hampton and Harrison, 1967) the patient was afebrile during his admission and his erythrocyte sedimentation rate was normal. Furthermore, at necropsy, there was no evidence of healed endocarditis.

Honey *et al.* (1967) have emphasized the importance of assessing left ventricular function before deciding on operative repair of an acquired ventricular septal defect. This is an extremely difficult task, particularly in an anaesthetized patient with two different 'leaks' from his left ventricle. Our patient was clearly to remain chairbound, despite a vigorous diuretic regimen and digitalization, unless some other procedure could be performed. In view of the fact that he had two surgically correctable lesions it seemed reasonable to attempt their repair.

We have been able to find only one report

of the development of both a ventricular septal defect and papillary muscle dysfunction after a myocardial infarct (Skoulas and Beier, 1967). In view of the close anatomical association between the area of the septum usually involved (Friedberg, 1966) and the origin of the posteromedial papillary muscle, it may well occur more frequently but be masked by the signs of the septal defect.

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References

- Friedberg, C. K. (1966). *Diseases of the Heart*, 3rd ed. W. B. Saunders, Philadelphia and London.
- Hampton, J. R., and Harrison, M. J. G. (1967). Sterile blood cultures in bacterial endocarditis. *Quarterly Journal of Medicine* (N.S.), **36**, 167.
- Honey, M., Belcher, J. R., Hasan, M., and Gibbons, J. R. P. (1967). Successful early repair of acquired Ventricular septal defect after myocardial infarction. *British Heart Journal*, **29**, 453.
- London, R. E., and London, S. B. (1965). Rupture of the heart: a critical analysis of 47 consecutive autopsy cases. *Circulation*, **31**, 202.
- Ross, R. M., and Young, J. A. (1963). Clinical and necropsy findings in rupture of the myocardium: a review of 43 cases. *Scottish Medical Journal*, **8**, 222.
- Skoulas, A., and Beier, R. L. (1967). Dissecting perforation of infarcted interventricular septum with associated posterior papillary muscle involvement. *American Journal of Medicine*, **43**, 461.

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