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PERFORATION OF THE INTERVENTRICULAR SEPTUM FOLLOWING CARDIAC INFARCTION

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Rupture of the interventricular septum complicating cardiac infarction has attracted much attention recently. Only one such case, diagnosed during life, has so far been reported from Southern Africa,[6] although several others have been recognized.[14,16] The lesion was first described in 1845, but the clinical diagnosis was not made before 1923; the features are now better defined, and it has become possible to recognize a case at the bedside.

The incidence is uncertain, but it appears to be far less common than rupture of the myocardium in other sites. Mitchell and Parish[9] recorded the admission of 3,790 patients with myocardial infarction to 3 British hospitals in the years 1939 - 1959; of 46 ruptures of the heart among these patients, only 9 were septal.

In the case now reported, a confident clinical diagnosis of ruptured interventricular septum was made, but post-mortem examination could not be performed and full confirmation is lacking. However, the clinical picture appears sufficiently characteristic to justify a brief description of the entity.

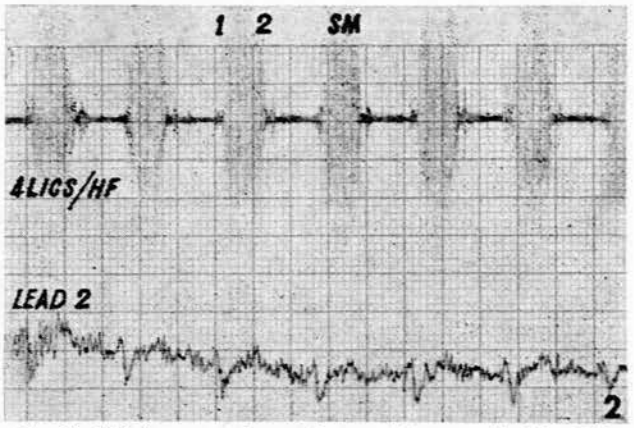

Fig. 2. High-frequency phonocardiogram taken on 25 July 1960. 1 = position of 1st heart sound; 2 = position of 2nd heart sound; SM = systolic murmur; 4LICS/HF = tracing taken at the 4th left intercostal space (at the left lower sternal edge), using the high-frequency setting.

CASE REPORT

A 75-year-old man had suffered from chronic bronchitis for many years. On 16 July 1960 he suddenly experienced severe central chest pain. His family practitioner diagnosed cardiac infarction, an electrocardiogram (ECG) showing gross elevation of the ST segment in leads V_1 - V_4 and a QS wave in V_1. The blood pressure was 150/70 mm.Hg. He was admitted to hospital and treated with phenindione, the prothrombin time being kept at 30 seconds (control 12 seconds). His progress for the first week was satisfactory, with no further pain and no cardiac failure. No murmurs were heard.

Eight days later, on 24 July, his condition suddenly deteriorated and he became shocked, cyanosed, and very dyspnoeic. A systolic murmur was now heard at the sternal edge in the fourth left interspace. This was loud (grade 5/6), unaccompanied by a thrill, pansystolic in type, and conducted towards the left axilla. The blood pressure was 90/60 mm.Hg. There were numerous rhonchi and coarse crepitations, the jugular venous pressure was raised to 5 cm. above the sternal angle, the liver was enlarged to 3 fingerbreadths below the right costal margin, and sacral oedema was present. An ECG showed ST-segment elevation and deep Q waves from V_3R to V_4 (Fig. 1), and the murmur was recorded phonocardiographically; it occupied the whole of systole, engulfing the first and second heart sounds (Fig. 2). On 25 July the serum glutamic oxaloacetic transaminase was 70 Frankel units (normal 12 - 40), and the serum lactic dehydrogenase 720 units (normal 200 - 400).

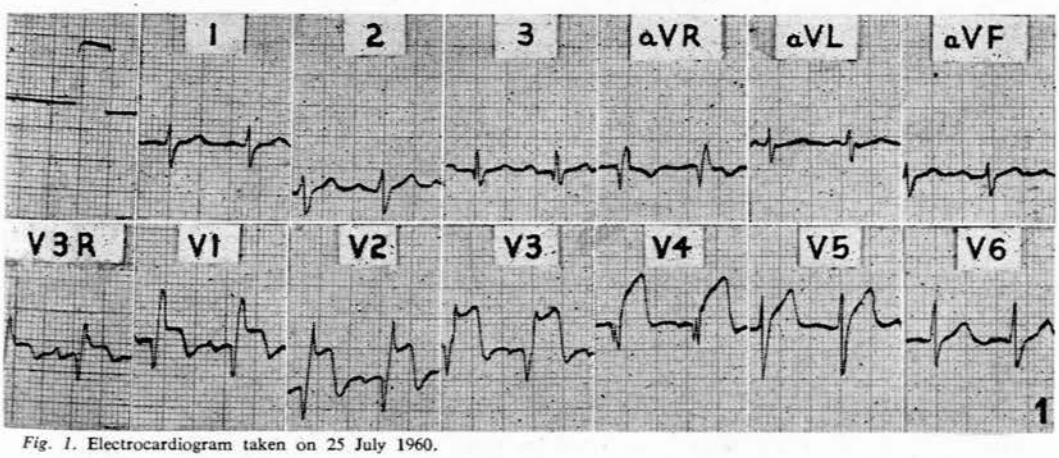

Fig. 1. Electrocardiogram taken on 25 July 1960.

The condition of the patient remained extremely poor, although the blood pressure rose spontaneously and averaged 105/70 mm.Hg. Despite treatment with digitalis, mersalyl, and aminophylline, the symptoms and signs of cardiac failure persisted, and he died on 29 July.

DISCUSSION

This patient showed the typical symptoms and signs of

perforation of the interventricular septum following myocardial infarction.[5,15,17] Most sufferers are elderly and the complication generally occurs a few days after the initial infarction, often at the end of the first week.

Among predisposing factors incriminated or suggested is systemic hypertension; however, the present patient was known to have been normotensive before his infarct. While several workers report that myocardial rupture is 2 or 3 times as common in those receiving effective anticoagulant therapy as in control groups,[2,7] this is not an invariable experience;[9] even if this is true, this complication is relatively so rare that it cannot be a good reason for withholding such treatment from patients with cardiac infarction. It has been suggested that the splinting effect of the right ventricular pressure tends to reduce the incidence of septal rupture,[8] but the present patient may even have had a raised right ventricular pressure because of his chronic bronchitis and emphysema. Most authors agree that specially severe coronary atherosclerosis is an important predisposing factor; the infrequency of septal rupture has been ascribed to the unusually rich anastomotic blood supply of this area.

In about two-thirds of cases, the perforation is situated in the lower part of the septum; 5 cases of multiple perforation occurred in 82 patients reviewed, linear tears tending to be larger than round ruptures.[15] Actual rupture is probably due to further occlusion of a coronary artery. The anterior situation of the infarct in this case suggested that perforation occurred in the area supplied by the left anterior descending artery; posterior rupture follows involvement of the right coronary artery.[5] Evidence for further infarction in the present case was the worsening ECG pattern and the elevated serum-transaminase and lactic-dehydrogenase levels.

The diagnosis can usually be made clinically when the condition of a patient who has recently suffered myocardial infarction suddenly deteriorates, and he develops a loud, harsh, pansystolic murmur at the left lower sternal edge. Among conditions that require differentiation are pre-existing ventricular septal defect, mitral incompetence, pericardial friction rub, and rupture of a papillary muscle.

It is unusual, but not unknown, to find congenital ventricular septal defect in the elderly; the problem will only arise if the patient is seen for the first time after the septum has perforated. In acquired perforation the murmur is said to radiate into the left axilla to a greater extent than in congenital defect, because of the lower anatomical situation and the acuteness of the haemodynamic changes.[5]

Differentiation from organic mitral incompetence can be made on the sudden development of right-sided failure in someone who has not only the murmur, but also evidence of recent cardiac infarction. Auscultation and the clinical features should enable a pericardial friction rub to be excluded.[12] Rupture of a papillary muscle usually leads to left-sided failure (which may not be immediate[8]); the murmur tends to be maximal at the apex, is usually not pansystolic and is often heard in diastole.[15] Pollock[11] found that 96% of patients with a ruptured septum had a murmur, although a murmur was detected in less than half of those with rupture of a papillary muscle. A thrill is present in more than half the cases of septal perforation, generally when the hole is small.[4]

The usefulness of phonocardiography in demonstrating the auscultatory signs is indicated in the present case and in the report by Harrison *et al.*[5] Confirmation of the presence of a left-to-right shunt can be made by cardiac catheterization, which is potentially hazardous, and by dye-dilution studies.[5]

Intraventricular conduction defects are comparatively rare, probably because of the tendency for rupture to occur towards the apical end of the septum.[10] Of 56 cases analysed, complete heart block was found in 2, varying degrees of AV block in 3, and right bundle-branch block in 5.[15] Bohan and Stansbury[1] have reported a case of perforated septum with changing ECG pattern indicating intermittent left bundle-branch block. The ECG of the patient presented in this paper (Fig. 1) shows the '$S_1S_2S_3$ syndrome'; since there were no S waves in lead III of the initial ECG, these appearances are probably due to acute myocardial infarction rather than chronic cor pulmonale.

Swithinbank[15] mentioned a patient who survived for 4 years and 10 months, and Harrison *et al.*[5] quoted a patient of Schlappi who lived even longer (the precise survival time is not given, but was more than $6\frac{1}{2}$ years). Long survival appears compatible with a reasonably active life in a small number of patients.

Conservative therapy can only be supportive, and treatment of cardiac failure is obviously required in those suffering from this complication. When a patient survives perforation for several weeks, but remains in failure, it is possible nowadays to consider surgical closure of the defect. There have already been several attempts at correction, but success so far has been limited. Poor vascularity of the affected area, owing to extensive atherosclerosis, is likely to prove a handicap, but 2 reported instances are at least promising. A 61-year-old man appeared to recover well after surgery, but died suddenly on the seventh day, apparently from a new anteroseptal infarct.[13] The sutures had held well, and there was no mural thrombus at the site of closure. In another case, survival was longer; the patient died 45 days postoperatively, also from another cardiac infarct, but the repair was intact.[3]

SUMMARY

Perforation of the ventricular septum is a recognized complication of cardiac infarction. The characteristic clinical picture is of sudden deterioration 1 or 2 weeks after the infarct, with predominantly right-sided cardiac failure, and the development of a loud, pansystolic murmur between the lower left sternal edge and the cardiac apex. It is compatible with survival for many years, and, in some cases, surgical closure of the defect may be possible.

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