Case 8. Perforation of Interventricular Septum Complicating Posterior Myocardial Infarction

This patient was a European male aged 76 years. Apart from episodes of heartburn related to certain foods, during the previous 6 months, there was no history suggestive of coronary artery disease.

On the day of admission, while dressing himself, he experienced a sudden severe retrosternal pain which radiated into the right shoulder and arm and was followed by loss of consciousness lasting some minutes. He was admitted to hospital about 4 hours later and when examined appeared comfortable, although still aware of a tightness across his chest.

The pulse was of good volume and regular, 70 per minute. Blood pressure 180/110 mm. Hg. The heart was not enlarged clinically and only a soft ejection type of systolic murmur was audible at the base. There were no other physical signs of note. Temperature was normal.

Blood examination: Haemoglobin 14 g.%, white cells 8,600/c. mm., and ESR 15 mm./first hour (Westergren).

Urine. There were no abnormalities on routine examination.

The electrocardiogram confirmed the clinical diagnosis of posterior myocardial infarction (Fig. 1) and the patient was treated accordingly.

X-ray of the chest taken on admission was normal.

Initial progress appeared to be satisfactory, but on the 4th day after admission, the patient complained of precordial and leftshoulder pain, worse on breathing. On examination a pericardial friction was detected.

On the 8th day after admission he complained of a disturbing dry cough associated with shortness of breath. Examination showed that the patient's general condition had deteriorated. He was orthopnoeic, pale and restless and perspired profusely. Auricular fibrillation was now present (Fig. 3) and the blood pressure had fallen to 110/70 mm. Hg. A loud pansystolic murmur was heard, maximal in the 4th left interspace and associated with a systolic thrill. Numerous crepitations were heard in both lung bases and sacral oedema was present. X-ray of the chest now showed an enlarged cardiac shadow with congestion in both lung fields.

In spite of a salt-free diet, digitalis, and diuretic therapy, the patient's condition continued to deteriorate and he died in congestive cardiac failure on the 20th day after admission.

The clinical course favoured a diagnosis of interventricular septal perforation following myocardial infarction, rather than rupture of a papillary muscle.

Autopsy (Prof. H. W. Weber) was confirmatory, revealing a recent infarct in the posterior wall of the left ventricle with perforation of the septum and severe coronary atherosclerosis involving both left and right coronary vessels. A fibrinous pericarditis was also present.

ELECTROCARDIOGRAM

Sinus rhythm 58 per minute. PR-interval normal. Normal axis deviation. Semi-horizontal heart position with transition between V_3 and V_4 . QRS of rather low voltage in standard leads, but of normal duration and pattern. QT=0.44 sec. (QTc=0.42 sec). Slight elevation of ST-segments in II, III and aVF with reciprocal changes in I, aVL and V_4 to V_6 . Symmetrical T-wave inversion in II, III and aVF. T-waves flat in V_4 and V_5 , inverted in V_6 . The u-wave well seen in V_3 has the same polarity as the T-wave in the precordial leads, except for V_6 where polarity is reversed.

Conclusion. These electrocardiographic findings, taken in conjunction with the clinical picture, strongly suggest partial-thickness postero-lateral infarction.

Sinus rhythm 92 per minute. QT=0.48 sec. (QTc=0.58 sec.). The most significant change is the appearance of pathological Q-waves in III and aVF with the ST and T-changes even more



Fig. 1. (Tracing on day of Admission)

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obvious. The T-waves are upright in V_{α} to V_{α} , but abnormal in shape.

Conclusion. This tracing now indicates a recent, through-and through postero-lateral myocardial infarction.

Auricular fibrillation present with a supra-ventricular extrasystole shown in V_{3} . Depression of ST due to digitalis also present.

DISCUSSION

Infarction of the interventricular septum occurs in about 70% of all cases of myocardial infarction.^{1,4} Yet perforation of the septum occurs infrequently, due in part to the good collateral circulation of the septum. I have been able to find 114 references (including the case described here) of cases of perforation of the septum.²

Rupture of the interventricular septum is the result of antero-septal infarction in 75% of cases, and conduction defects, notably right bundle-branch block, are present in 40% of cases with perforation of the septum.³

In this patient septal perforation followed on a posterior infarct, although no electrocardiographic evidence of septal involvement could be found, i.e. qrS or QS over right precordial leads.

The association of septal infarction and perforation occurring without electrocardiographic confirmation has been reported previously in the presence of a posterior myocardial infarction.^{2,4}

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