Constrictive pericarditis after myocardial infarction

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SUMMARY Constrictive pericarditis developed in a 55 year old man two years after transmural myocardial infarction complicated by severe acute pericarditis. Pericardiectomy was successful. Although this very rare late complication of myocardial infarction has been reported as a sequela of frank anticoagulant induced haemopericardium, in this case there was no clinical evidence of such an association.

Pathological studies show that almost all patients with a transmural acute myocardial infarction develop at least a localised fibrinous pericarditis.¹ It may, however, diffusely involve the pericardial surfaces and may occasionally obliterate the pericardial sac.² It is surprising, therefore, that constrictive pericarditis has not been reported more frequently as a late complication among survivors of extensive myocardial infarction. We describe such a case.

Case report

A 55 year old man was admitted because of severe prolonged central chest pain associated with sweating and dyspnoea. Physical examination was normal. The electrocardiogram showed evidence of an acute transmural inferolateral myocardial infarction with Q waves and ST elevation in leads II, III, aVF, V5, and V6. Forty eight hours later he was pyrexial, dyspnoeic, and hypotensive (blood pressure was 95/60 mm Hg). A loud pericardial friction rub was audible and this persisted for eight days. The jugular venous pressure was not elevated clinically, the central venous pressure measured by right heart catheterisation was normal, and the chest radiograph showed no noticeable cardiomegaly—suggesting that the hypotension was due to the extensive myocardial infarction rather than the development of an appreciable haemopericardium.

Four months after discharge superficial and deep venous thrombosis developed in the right leg for which he received anticoagulant treatment for eight weeks. There was never any clinical or radiological evidence, however, to suggest the development of an important haemopericardium.

Twenty three months later he was referred with a two year history of progressive abdominal distension, mild ankle swelling, and a one year history of exertional dyspnoea progressing to New York Heart Association grade II. He said that he did not have symptoms of orthopnoea and paroxysmal nocturnal dyspnoea. On examination he appeared to be abnormally pigmented, and had gynecomastia, ascites, and hepatomegaly. The jugular venous pressure was elevated to 13 cm above the sternal angle and the waveform showed steep “x” and “y” descents and a rapid rise to the “h” point (fig). Blood pressure was 95/75 mm Hg with 15 mm Hg pulsus paradoxus. A clear diastolic knock was audible at the lower left sternal border. Chest radiograph showed a normal cardiothoracic ratio and no pericardial calcification. The electrocardiogram showed decreased QRS voltage with generalised T wave flattening with atrial fibrillation developing during admission. The findings at cardiac catheterisation included: right atrium (mean) 27/20 (25) mm Hg; right ventricle 42/24 mm Hg; pulmonary artery (mean) 42/28 (35) mm Hg; pulmonary artery wedge (mean) 31/23 (27) mm Hg; left ventricle 108/27 mm Hg; aorta (mean) 108/78 (95) mm Hg.

The pressure waveforms in both ventricles showed an early diastolic dip and plateau with equalisation of diastolic pressures in all four chambers. Constrictive pericarditis was diagnosed and an operation was advised. There was no history of tuberculosis and no evidence of any systemic or connective tissue disorder.

At operation a thickened, adherent, but non-calcified pericardium encased the heart, particularly as an oblique band extending from the infero-anterior surface of the right ventricle to the supero-anterior aspect of the left ventricle. The pericardium was successfully resected. Two months after oper-

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Figure Jugular venous pressure tracing (JVP) shows steep "x" and "y" descents. The pericardial knock (K) follows the second heart sound (2) by 0.12 s and coincides with the nadir of the "y" descent of the jugular venous pulse. PCG, phonocardiogram; MF, mid frequency; LF, low frequency.

ation the jugular venous pressure was normal and the ascites, peripheral oedema, and diastolic knock had disappeared.

Discussion

The syndrome of constrictive pericarditis is caused by adhesions and fibrous contracture of the pericardial sac that surrounds the heart. This impairs normal diastolic ventricular filling, causing a fall in stroke volume and elevation of systemic and pulmonary venous pressures. Fatigue, dyspnoea, and signs of congestive cardiac failure result.

Acute pericarditis is common after myocardial infarction.³ The reaction is usually localised over the area of infarction but may be diffuse in up to 20% of patients and associated with a serosanguinous effusion.^{1,4} Occasionally the pericardial space can be obliterated by a diffuse fibrinous exudate. Chronic constrictive pericarditis as a late complication of myocardial infarction is, however, rare having been reported on only three previous occasions.⁵⁻⁷ All had evidence of pericardial involvement early in the course of the myocardial infarction and after prophylactic anticoagulant treatment had developed frank haemopericardium presenting as constrictive

pericarditis six to twelve months later. Our patient never had clinical, radiological, or haemodynamic evidence of haemorrhage into the pericardial space and no symptoms or signs of post-myocardial infarction syndrome.⁸ Although it is possible that subclinical haemopericardium or Dressler's syndrome was aetiologically important, it is more likely that severe fibrinous pericarditis after extensive myocardial infarction was the major initial abnormality. This accords with the development of a loud and persistent pericardial friction rub for eight days without any rise in central venous pressure. The fibrinous pericarditis progressed to chronic pericardial fibrosis and mechanical constriction over the next two years.

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