Concealed post-infarction left ventricular rupture – a diagnostic dilemma

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Summary
We describe a patient with post-infarction left ventricular rupture exhibiting several atypical features. A successful outcome was achieved after serendipitous surgery.

Keywords: acute myocardial infarction, left ventricular rupture, aortic dissection, cardiac tamponade

Left ventricular rupture is a catastrophic complication of acute myocardial infarction and almost invariably fatal. This mechanism accounts for up to 10% of all infarct-related deaths and the prevalence appears to have been unaffected by the widespread introduction of thrombolytic therapy. This complication has assumed increasing importance as the all-cause mortality rate has generally declined in acute myocardial infarction, while the incidence of left ventricular rupture has been constant over the past 16 years.¹ Although left ventricular rupture may be more common in women and in those sustaining their index cardiac event, attempts at predicting and preventing this potentially salvageable condition have been unsuccessful.² The case described below well illustrates the difficulties in establishing a diagnosis.

Case report

A 63-year-old woman presented with near syncope followed by interscapular pain of 30 minutes duration. She gave a three-month history of chest and arm discomfort which had been ascribed to dyspepsia. There was no known cardiovascular disease but she had a long-standing pack-a-day cigarette habit. The patient was peripherally cyanosed with borderline hypotension (systolic blood pressure 90–100 mmHg) and prominent jugular venous distension. She exhibited a resting sinus tachycardia of 110 beats/min and a paradox of 15 mmHg. Left arm pulses were undetectable. Clinical examination of the heart was unremarkable. Electrocardiogram (ECG) revealed borderline ST elevation in I, aVL, V₁ and V₂. The mediastinal shadow on chest X-ray was normal. Echocardiography showed a small pericardial effusion with early right heart diastolic collapse and a non-dilated left ventricle with globally reduced function. Arterial blood gases revealed a mild metabolic acidosis. The clinical diagnosis was thoracic aortic dissection

with cardiac tamponade. Cardiac catheterisation showed a raised mean right atrial pressure of 15 mmHg with equilibration of the right atrial pressure and left and right ventricular end diastolic pressure confirming cardiac tamponade. Single-plane contrast ventriculography (30° right anterior oblique) revealed an abnormality of left ventricular wall motion involving the contiguous antero-basal and antero-lateral segments, but no dye leakage into the pericardial space. Selective coronary angiography demonstrated an isolated occlusion of the first diagonal branch of the left anterior descending coronary artery. Contrast aortography showed chiral asymmetry of ascending aortic flow and a non-regurgitant aortic valve. There was a mobile valve-like structure at the origin of the left common carotid artery and the left subclavian artery was absent.

The diagnosis remained one of thoracic aortic dissection complicated by cardiac tamponade and coronary heart disease. She was transferred to surgery with deteriorating haemodynamics culminating in a bradycardic cardiac arrest during induction of cardiac anaesthesia. Immediate sternotomy confirmed cardiac tamponade and a small haemopericardium was evacuated. Inspection of the heart revealed a recent lateral wall infarct confined to the first diagonal territory. The site of ventricular rupture was seen as an haemorrhagic punctum surmounted by a small epicardial haematoma. There was no active bleeding. The integrity of this spontaneous seal was confirmed by careful inspection and formal repair was not indicated. Exploratory aortotomy showed no evidence of aortic dissection. However, frond-like masses were found at the origins of the left common carotid and subclavian arteries. Excision biopsy revealed complex atheroma with a scanty inflammatory

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Cardiac tamponade: causes
<ul style="list-style-type: none">• chest trauma• aortic dissection• post-cardiac surgery• myocardial infarction with rupture• iatrogenic – cardiac catheterisation• uraemic patients on haemodialysis• malignancy• acute pericarditis – anticoagulation

Box 1

cell infiltrate. No bacterial or fungal pathogens were isolated. The patient made an uncomplicated recovery.

Discussion

Left ventricular free wall rupture after myocardial infarction almost invariably predicates a fatal outcome and represents an increasingly important mechanism of infarct-related death. Successful salvage with surgical therapy has occasionally been reported, but survival after associated cardiac arrest is indeed very rare in acute rupture.³ However, it appears that subacute forms of this condition occur, in which patients undergo a slowly progressive or stuttering disruptive process. This has been well described in the classification of the different forms of left ventricular rupture.⁴ Such cases offer a window of opportunity for stabilisation prior to cardiac surgery.

Establishing the correct diagnosis may be problematic as illustrated in the case described, where there was a paucity of clinical evidence for myocardial infarction, and in which the clinical signs and conflicting data derived from the investigative protocol suggested a complex thoracic aortic dissection. In retrospect, the potentially hazardous left ventriculogram was most suggestive of the underlying pathology, but it is unclear whether transoesophageal echocardiography or thoracic computed tomography would have provided a more accurate diagnosis. Although the specific findings at surgery were not anticipated, this was of little consequence as the patient required emergency thoracotomy for relief of cardiac tamponade.

A high index of suspicion of left ventricular rupture may be central to the initiation of a successful diagnostic protocol. Success even in the setting of a more typical infarct demands a high state of awareness for this complication, in the face of rather non-specific clinical signs. Hypotension is a sensitive marker of subacute ventricular rupture but lacks specificity. Therefore, unexplained hypotension post-infarction should be urgently evaluated by echocardiography. Findings which suggest left ventricular rupture include pericardial effusion, which may exhibit a relative echogenicity or the specular appearance typical of thrombus formation. Confirmation of haemopericardium by pericardiocentesis or the development of cardiac tamponade requires

Cardiac tamponade

Clinical features

- clinical shock
- low output state + fatigue
- tachycardia + raised jugular venous pressure
- muffled/quiet heart sounds
- low voltage ECG with possible electrical alternans
- globular heart on chest X-ray

Investigations

- trans-thoracic/transoesophageal echocardiography
- Swan-Ganz right heart catheterisation
- chest X-ray
- ECG
- pericardiocentesis

Box 2

early transfer for surgical intervention and carries a 50% chance of survival. Evaluation by transoesophageal echocardiography complements trans-thoracic imaging and may have clarified the diagnosis in the case described as the sensitivity and specificity are 99% and 98%, respectively.⁵ The rapid deterioration in our patient precluded this assessment prior to cardiac surgery.

The ECG in left ventricular rupture is usually abnormal. Often the changes are not marked, however, ostensibly rendering the ECG relatively 'normal'. Unexpected alteration in T-wave polarity may be observed and there tends to be a lack of the expected evolutionary pattern typical of acute myocardial infarction.⁶ The site of the infarction electrocardiographically may be important in identifying subsets of patients at increased risk of left ventricular rupture. Lateral or inferoposterior transmural infarction confer the greatest risk.⁶ However, the occurrence of left ventricular rupture with ECG changes confined to V₁ and V₂ appears to be unusual.⁷

Cardiac catheterisation and coronary angiography in isolation add little to the primary diagnosis but are required in the work-up for cardiac surgery. The infarct-related artery most often implicated is the left anterior descending, followed by the circumflex and right coronary arteries. Thus the vessel-disease distribution was another unusual feature of this patient's presentation.

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