A life-saving case of Dressler's syndrome

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Abstract

Myocardial rupture is an uncommon and life threatening event which may occur in the setting of acute myocardial infarction. In this case report we describe a rare instance of a patient presenting with signs of ventricular rupture as a late presentation of myocardial infarction. The findings of cardiac computed tomography are demonstrated as well as intra-operative images. Despite its very high attendant mortality risk, our patient's life was saved by the earlier development of Dressler's syndrome, an intense inflammatory process in the pericardium, resulting in dense adhesions, which effectively tamponaded the leak from the ventricular free wall rupture and prevented extravasation.

Keywords

Myocardial rupture, complications, myocardial infarction, Dressler's syndrome, left ventricular aneurysm, emergency cardiac surgery, pericardial effusion

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A life-saving case of Dressler's syndrome

Myocardial rupture is an uncommon event which frequently presents with sudden death in the aftermath of myocardial infarction. The mortality rate in the hospital setting approaches 60%. In this case report, we present a very rare case of a patient presenting to hospital with cardiogenic shock secondary to cardiac rupture as a late complication of previously undiagnosed myocardial infarction. We present computed tomography (CT) findings, intra-operative photography and video imaging of the cardiac surgery used as definitive treatment. In this case, the occurrence of Dressler's syndrome was life-saving, as the intense inflammatory response in the pericardium effectively sealed the rupture, preventing death.

A 66 year old female presented to her local hospital with a three week history of malaise, severe vomiting and pericarditic chest pain. She had a history of treated hypothyroidism but was otherwise in good health.

On the day of admission she had become increasingly short of breath and collapsed, presenting in a shocked state to her local hospital. She was commenced on inotropic support. A provisional diagnosis of oesophageal rupture was made and emergent CT scanning was undertaken. This excluded oesophageal perforation, but instead showed a

large pericardial effusion measuring 2.7cm in its maximal dimension. It also showed bilateral pleural effusions and some basal consolidation. She was transferred to our tertiary cardiac centre for investigation and management of impending pericardial tamponade.

On arrival she was being treated with noradrenaline. Despite this, blood pressure was 85/60 mmHg. ECG showed low voltage complexes and a Q wave in the inferolateral leads. This raised the possibility that this was a very late presentation of an inferolateral ST elevation myocardial infarction (Figure 1).

Bedside echo showed an aneurysm at the apex of the left ventricle (LV), which contained spontaneous contrast with moderate to severe LV dysfunction. It confirmed the presence of the large effusion but, surprisingly, there were no echocardiographic findings to suggest that this was causing

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Figure 1. ECG performed at time of admission shows low voltage complexes, inferolateral Q wave and T wave inversion.

Figure 2. (a) Four-chamber view of the heart showing large apical and lateral aneurysm with a defect in the free wall of the left ventricle (LV), but no active contrast leak. (b) Three-chamber view showing aneurysm formation and area deficient of myocardium (black arrows).

haemodynamic compromise. This presented a real clinical conundrum: should the effusion be drained to improve the blood pressure? Why was there no echo evidence of tamponade?

In view of the ECG changes and cardiogenic shock emergency coronary angiography was carried out. This

showed severe disease in the left anterior descending artery (LAD), and an occluded obtuse marginal branch of the circumflex (responsible for the ECG changes). The right coronary had moderate disease only. This ruled out the presence of a critical lesion as a potential cause of ongoing hypotension. An intra-aortic balloon pump was placed to augment

Figure 3. (a) The arrow demonstrates the defect in the LV found when the pericardium was dissected away. (b) The arrow shows the localised haemorrhage and irregularity of the heart's surface caused by the intense inflammation of Dressler's syndrome. (c) The thin walled aneurysm is denoted by the black arrows and the pericardial patch sealing the defect in the LV by the blue arrow.

blood pressure and relieve symptoms whilst further investigations were undertaken.

Cardiac CT was carried out. This demonstrated a haemopericardium and a large LV aneurysm with a defect in the free wall of the LV, but no active contrast leak to the pericardium (see Figure 2). These findings are consistent with a contained ventricular rupture.

The patient was referred for urgent cardiac surgery. At operation she was found to have a ruptured LV free wall (Figure 3(a)) and extensive myocardial scar which was completely adherent to the pericardium. The appearance of the external surface of the heart after removal of adhesions

is shown in Figure 3(b). This was treated by aneurysmectomy and placement of a pericardial patch (Figure 3(c) and supplementary material video file 1), with a single vein graft to the left anterior descending coronary artery.

She was discharged 12 days post admission and remains free of symptoms.

Discussion

This case demonstrates two important complications which may occur in the setting of acute myocardial infarction – the life threatening complication of ventricular rupture (VR)

and Dressler's syndrome, the occurrence of which was life-saving for our patient.

VR complicates myocardial infarction with a frequency of 1%. The in-hospital mortality associated with VR is approximately 58%. With rupture of the free wall of the LV, the mortality is 80%.¹ The incidence of this condition is declining with improved reperfusion therapy after myocardial infarction,² and in a recent paper by Figueras et al., the authors recommend diagnostic aspiration of pericardial fluid with haematocrit measurement to confirm the diagnosis.² However, with current imaging modalities, this may be superseded by non-invasive imaging in the form of magnetic resonance imaging or cardiac CT, as in our case.

The red flag symptoms in this case were persistent hypotension, moderate pericardial effusion, ECG changes and the confirmatory test was cardiac CT.

It is very uncommon for a cardiac rupture to be the presenting sign of myocardial infarction. This has, however, been previously reported in the literature,³ and this case demonstrates the clinical, investigative and operative findings associated with subacute rupture of the LV. The diagnosis should be considered in any patient with myocardial infarction, particularly ST elevation MI, and a pericardial effusion. Management and investigation is best carried out in a centre with cardiothoracic facilities and MR or cardiac CT.

Dressler's syndrome, first described in 1956, is characterised by pleuritic chest pain, low grade fever and pericarditis, which may be accompanied by pericardial effusion, and tends to follow a benign clinical course.⁴ It is thought to be immune-mediated⁵ and has been reported to have had a declining incidence in recent years.⁶

The operative findings were the key to how this patient survived an out of hospital ventricular rupture – the intense

inflammation in the pericardium caused by post myocardial infarction syndrome made the pericardium dense and adherent to the ventricle preventing massive extravasation and attendant sudden death. This is a truly life-saving case of Dressler's syndrome.

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Conflict of interest

None declared.

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