Percutaneous drainage and successful treatment of pericardial tamponade due to Dressler syndrome

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Introduction

Patients with Dressler syndrome generally present with malaise, fever, chest pain, leukocytosis, an elevated erythrocyte sedimentation rate and pericardial effusion.[1] To the best of our knowledge, presentation of Dressler syndrome with pericardial tamponade is very rare. An investigation on Medline revealed that no cases had been reported in the last 10 years. We reported this case because of its rare presentation pattern and its successful treatment with percutaneous catheter drainage.

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Case report

An 80-year-old man was admitted to hospital with a two-week history of malaise, fever, cough and shortness of breath. Two months previously, he had been conservatively treated for acute anterior wall myocardial infarction (MI). He was comfortable when lying flat in bed; his blood pressure was 110/70 mmHg, and there was 30 mmHg of paradox; his pulse was 110/minute; his respiratory rate was 20/minute with venous pressure elevated 7 cm above the sternal angle and not increased with inspiration; temperature was 37.9ºC. Further investigation showed his heart rhythm was regular, with the apical impulse not easily palpated; S1 and S2 was normal; and no S3, S4, murmur or pericardial friction rub was heard. There was also no peripheral oedema, clubbing, or cyanosis.

Telecardiography showed a large heart, and no pulmonary congestion (figure 1). An electrocardiogram showed prominent QS wave in leads V1 through V6 and minimal elevated ST segments. Enzymatic studies gave no evidence of myocardial necrosis.

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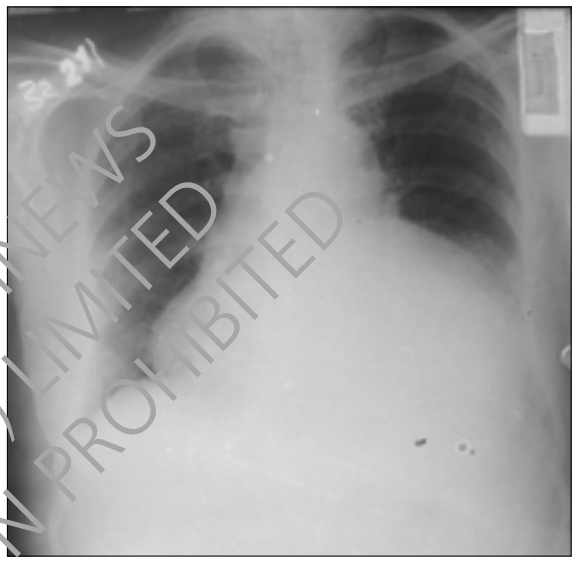
Figure 1. Telecardiography before pericardiocentesis showing a large heart, and no pulmonary congestion

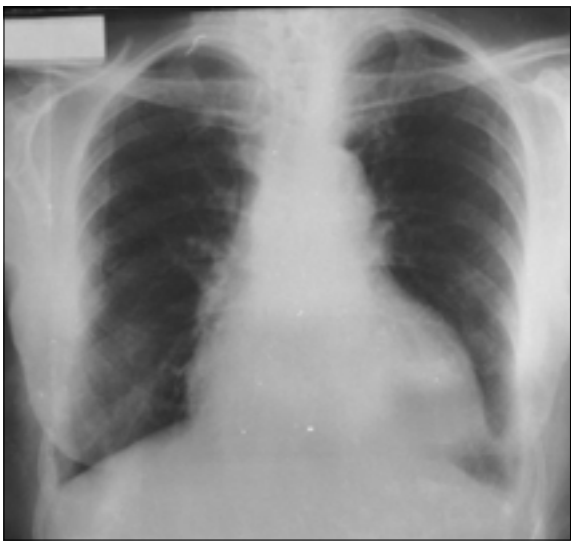


Echocardiography showed a large pericardial effusion with diastolic collapse of the right ventricle. Pericardiocentesis was performed via the subxiphoid route, and a percutaneous 7Fr pigtail catheter was advanced into the pericardial space using the technique described by Lorell and Grossman.[2] Some 2,000 ml of exudate was freely aspirated over two days.

Cytology examination revealed no malignant cells. Thoracoabdominal computed tomography showed minimal pericardial and pleural effusions. There were no parancimal lesions in the lung or liver. Control telecardiography after pericardiocentesis showed regression of cardiomegaly (figure 2). Microscopic examination of the fluid, did not detect any acid resistant bacteria using the Ziehl Nielson technique. Tubercule analysis was negative.

Aspirin 2 g daily and prednisolone 40 mg daily was begun. Echocardiography two weeks later showed no pericardial effusion. Aspirin and prednisolone was tapered in two weeks and two months after the treatment, no pericardial effusion was determined on echocardiography.

Figure 2. Telecardiography after pericardiocentesis



Key messages

- Dressler syndrome is rare and should be considered in any patient with acute myocardial infarction who has not received thrombolysis
- Since Dressler syndrome can cause complications, patients should be admitted to hospital in case cardiac tamponade develops
- Pigtail catheter drainage with anti-inflammatory therapy should be sufficient treatment for those patients with Dressler syndrome complicated by pericardial tamponade

Discussion

Development of cardiac tamponade in patients with MI may be related to pericardial haemorrhage within the first three days after the infarction. Dressler syndrome usually occurs one to eight weeks after infarction. The exact cause of this syndrome is not known but an immunological process is generally implicated.[1] The advent of thrombolysis and the widespread use of heparin has led to a decrease in the incidence of this syndrome.[3] Our patient did not have thrombolytic treatment, however, due to the late presentation of his acute MI to hospital. It may be that this has a role in the genesis of pericardial effusion.

In an old man, the most common cause of cardiac tamponade is malignant disease.[4] This led to our investigations for malignant cells and thoracic and abdominal computerised tomography. But no evidence of malignancy was detected. Although tuberculosis is common in our country, no evidence of this was detected either.

Post-infarction pericarditis generally produces mild symptoms and requires no specific therapy.[1] Differentiation from a second MI or post-infarction angina is very important. Absence of a marked increase in CK-MB and its clinical presentation pattern discriminates these patients from those with a new MI. Pigtail catheter drainage is sufficient for patients with post-infarction pericarditis because it permits a complete drainage of the effusion. They may also require anti-inflammatory therapy to relieve the pericardial pain and this therapy was adequate in our patient.

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