Pericardial effusion simulating aortic dissection

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SUMMARY A wide mediastinal shadow on chest x-ray films taken in two patients with acute thoracic pain was interpreted as a sign of aortic dissection. Pericardial effusion with tamponade was present instead. This condition must be suspected in patients with an enlarged base of the heart, and an echocardiogram should be performed to assess the possibility of pericardial effusion.

The rapid widening of the mediastinal shadow is a very common radiological sign of acute aortic dissection and can suggest this condition in the proper clinical setting.

In the presence of pericardial effusion, "bottle shape" configuration of the cardiac silhouette, straightening of its contours, and acute cardiophrenic angles have been emphasised. Broadening of the mediastinal shadow has also been mentioned.

We describe two examples of pericardial effusion, whose radiographic appearance and clinical presentation simulated acute aortic dissection.

Case reports

CASE 1
A 44-year-old man was admitted to the emergency room with a two days history of acute tearing epigastric pain, which was increased by inspiration.

At the age of 20 years he had had tubercular pleurisy: At 40 years the patient began to experience tiredness and fatigue, and two years later he noticed that his skin had become darker. One month before admission, the diagnosis of Addison's disease was made while he was in another hospital, because of a further episode of pleural effusion. A control erect x-ray film of the chest (Fig. 1a) taken 10 days before the present admission showed a slightly enlarged heart, moderately prominent ascending aorta, and absence of pleural effusion.

On admission, the blood pressure was 100/80 mmHg, the pulse was 100/minute and regular, and the skin was cold and clammy. The peripheral arterial pulses were present and equal, but small. The jugular veins were distended. The cardiac impulse could not be felt and the heart sounds were distant. An x-ray film of the chest, taken in the upright position, showed enlargement of the cardiac silhouette, left pleural effusion, and a broad vascular pedicle (Fig. 1b). Diffuse ST segment elevation was seen on the electrocardiogram. The serum creatine kinase (CK) was 432 IU/l and the lactate dehydrogenase (LHD) was 512 IU/l. We suspected a dissecting aneurysm of the aorta with haemopericardium and perhaps an acute myocardial infarction caused by coronary artery dissection.

Cardiac catheterisation was therefore undertaken. Right and left heart pressures were normal. The cardiac index was low (1.6 l/m per m²). The angiographic appearance of the aorta and of the coronary arteries was normal. Pressure measurement repeated five minutes after the injection of contrast material disclosed that the diastolic pressure was almost identical in the left ventricle (23 mmHg), pulmonary artery (20 mmHg), right ventricle (20 mmHg), and right atrium (18 mmHg) (Fig. 1d). A superior vena caval angiogram showed that the vessel was displaced laterally causing the appearance of the broad vascular pedicle (Fig. 1c). Right atrium, right ventricle, and pulmonary artery appeared to be normal. A revised diagnosis of pericardial effusion was made and it was later confirmed by an echocardiogram (Fig. 1e).

On the following day the patient was taken to the operating room, where the pericardial cavity was entered through a xiphoid approach and 600 ml serosanguineous fluid were drained. The heart was covered by a thick fibrin layer of which as much as possible was removed. Loculations were opened and the pericardium was partially resected. Histological examination showed non-specific pericarditis. The pericardial fluid was sterile. The patient recovered uneventfully.

CASE 2
A 54-year-old obese man was admitted to the emergency room because of recurrent epigastric pain radiating to the back. The patient also had a long-
Fig. 1 Case 1. (a) Erect chest x-ray film 10 days before admission. (b) Erect chest x-ray film on admission showing enlarged superior mediastinum and left pleural effusion. (c) The superior vena caval angiogram shows that the vessel is displaced laterally (arterial catheter in the ascending aorta). (d) Left ventricular (LV), pulmonary arterial (PA), right ventricular (RV), and right atrial (RA) diastolic pressures are raised and equal. (e) Echocardiographic sweep from the left ventricle (LV) to the left atrium (LA), showing anterior and posterior echo-free spaces (EFS). AO, aorta; MV, mitral valve; RV, right ventricle.
With the provisional diagnosis of acute dissection of the ascending aorta, the patient was submitted to cardiac catheterisation. The diastolic pressures were equally raised throughout the left and right heart chambers. An ascending aortogram showed no signs of dissection. After a superior vena caval angiogram, a large space between the right external border of the "heart" and the right atrial endocardium was seen (Fig. 2b).

The diagnosis was of cardiac tamponade and the patient was therefore taken to the operating room. Through a xiphoid approach the pericardial cavity was entered. Five hundred millilitres of brown fluid were drained. Fibrin was removed from the anterior surface of the heart and fibrin bridges were disrupted by finger fracture. Cultures of the fluid remained sterile. The patient recovered promptly.

**Discussion**

The two patients described in this paper presented with acute chest pain, hypotension, and a wide superior mediastinum on the chest x-ray film. Other significant features were ST segment elevation and increased serum enzyme levels.

Chest pain and hypotension can be caused by a variety of syndromes, like acute myocardial infarction, aortic dissection, pulmonary embolism, pneumothorax, oesophageal perforation, acute tamponade, and others. Concomitant ST elevation and increased cardiac enzyme levels restrict the diagnostic possibilities to acute myocardial infarction and, less commonly, aortic dissection or acute pericarditis with tamponade. A wide superior mediastinal shadow is one of the most common and specific signs of aortic dissection, whereas it is virtually unknown in the other two conditions. Dissection of the aorta, furthermore, can be complicated by acute myocardial infarction (caused by extension into a coronary artery) and/or by intrapericardial haemorrhage. We felt, therefore, that a dissecting aneurysm of the aorta was the most probable explanation of all the signs and symptoms present in our patients.

The clinical manifestations of acute pericardial effusion with tamponade are mostly related to the ensuing haemodynamic effects. The pulse pressure is diminished, pulsatilis paradoxus is present, and there is inspiratory distension of the veins of the neck. Oppressive chest pain can occur if the effusion accumulates slowly. The electrocardiogram is of limited value in the diagnosis of acute pericardial effusion: most commonly low voltage and alternation of the QRS complexes have been observed. The ST segment has been reported to be either raised or depressed.
The chest x-ray film usually shows a rapidly increasing cardiac silhouette, but there is no diagnostic shape or size, and differentiation from cardiomegaly is difficult. In addition, in patients with pericardial effusion, the change from the erect to the recumbent position may induce some broadening of the base of the heart, better seen when the heart is small. Though this radiographic feature has been mentioned in the medical reports, we have not been able to find published findings of clinical examples supporting this contention. Finally, even when the base of the heart is broadened by accumulation of fluid, the aortic knob, being extrapericardial, should remain visible. In our patients' x-ray films, taken in the upright position, the superior mediastinum appeared greatly and uniformly enlarged and the aortic knob was not discernible.

A possible explanation of such an unusual distribution of the pericardial fluid is that the fibrinous adhesions detected at operation prevented any significant collection in the inferior region of the pericardial space and forced the fluid to accumulate around the vascular pedicle.

Echocardiography is a very sensitive technique for the diagnosis of pericardial effusion and is also useful for detecting dissection of the ascending aorta. In our second patient, the echocardiographic recording was non-contributory because of the poor image. We did not obtain an echocardiogram before heart catheterisation in the first patient, but the ultrasound image taken afterwards was typical of pericardial effusion.

On the basis of this experience, we recommend that the physician should be aware that cardiac tamponade is not excluded by the radiological appearance of an enlarged superior mediastinum and that an echocardiogram should be obtained.

References

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