Acute pericarditis: a presenting manifestation of aortic dissection

Hursit Soyer

Department of Cardiology,
Nisa Hospital, Istanbul, Turkey

Correspondence to
Dr Hursit Soyer,
hursitsoyer@hotmail.com

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DESCRIPTION

A patient presented to the outpatient clinic (non-emergency), with non-exertional retrosternal chest pain of 3 days' duration. The pain was sharp in character and worsened by being in a supine position. The patient had no history of cardiac disease or hypertension and was an active smoker. Phenotypic characteristics of Marfan or Ehlers-Danlos syndrome were absent. On physical examination, the patient had diaphoresis. Vital signs were normal, body temperature was 36.8°C and heart rate was 88 bpm with no dysrhythmia. Blood pressure was 120/70 and 110/70 mm Hg in the right and left arms, respectively. On auscultation, a pericardial friction rub was heard over the left upper sternal border. No cardiac murmurs were noted. Pulses were equal throughout all extremities. Initial ECG (figure 1) showed a normal sinus rhythm. A concave upward ST segment elevation in leads I, II, aVL, aVF and V2–6 was evident

Figure 1 Twelve-lead ECG showing normal sinus rhythm, with concave upward ST segment elevation in leads I, II, aVL, aVF and V2–6, no reciprocal ST segment depression (except in aVR) and PR segment depression in leads I, II, aVL and V3–6.

with no reciprocal ST segment depression (except in aVR). In addition, PR segment depression was seen in leads I, II, aVL and V3–6. A bedside trans-thoracic echocardiogram (TTE) was performed, which revealed normal ventricular function and wall motion. TTE also showed the presence of pericardial effusion (5 mm) and the ascending aorta was dilated (4.8 cm). However, neither aortic flap nor aortic valve regurgitation was seen. Troponin-I level was normal ($<0.01 \mu\text{g/L}$). However, C reactive protein (218 mg/L, reference range $<10 \text{ mg/L}$) and white cell count were high ($10.5 \times 10^3/\mu\text{L}$). After this initial clinical and laboratory work up, aortic dissection was suspected on the basis of the following signs and symptoms: continuing sweating, absence of high fever, severe chest pain disproportionate to the severity of clinically detected acute pericarditis and presence of aortic aneurysm as detected on bedside TTE. D-dimer test and CT angiography were performed. D-dimer was $>10,000 \text{ ng/mL}$ (reference range, $<500 \text{ ng/mL}$). CT angiography of the thorax and abdomen showed the presence of a type I DeBakey aortic dissection, intramural thrombus in the ascending aorta and pericardial effusion (figure 2). The dissection involved the ascending aorta (figure 2B), aortic arch and the descending aorta (figure 2A). In addition, the ascending aorta was dilated. An increase in the volume of pericardial effusion was also noted as compared to the initial TTE findings (figure 2B). Emergency surgery was carried out. During the operation, dissection was found to be starting at the supracoronary level but sparing the aortic valve, with intramural haematoma in the aorta and thrombus in the false lumen. In addition, there was abundant haemorrhagic effusion in the pericardial space. The patient had postoperative mediastinal bleeding and died from irreversible shock 24 hours later.

Figure 2 Contrast-enhanced CT image of the thorax at the level of the descending aorta showing a prominent dissection plane (red arrow) and abundant pericardial effusion (blue arrows) (A). Thoracic view at the level of the ascending aorta showing a prominent dissection plane and thrombus, and dilation of ascending aorta (yellow arrows) (B).

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Learning points

- ▶ Aortic dissection can cause a wide variety of presentations, depending on the aortic segment involved.
- ▶ Aortic dissection is most commonly misdiagnosed as myocardial infarction or other causes of chest pain such as pulmonary embolisation.
- ▶ Acute pericarditis may be the predominant feature in aortic dissection.

Development of pericarditis is not an unexpected condition in cases with proximal aortic dissection.^{1 2} However, as in our

case, pericarditis with its full clinical characteristics may be the presenting feature of aortic dissection. Our case shows that acute pericarditis, particularly its characteristic pericardial chest pain, may dominate the clinical picture by mimicking characteristic chest pain of acute dissection.

Competing interests None declared.

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