Netherlands Journal of Critical Care
Submitted July 2016; Accepted January 2017

CLINICAL IMAGE

Haemorrhagic pericardial effusion

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Keywords - haemorrhagic pericardial effusion, cardiac tamponade, pericarditis

Case

A 59-year-old male with a history of gout, diabetes, hypertension, acute pancreatitis, atrial fibrillation and chronic renal failure presented to the emergency department (ED) with a fever of up to 38.5 °C, abdominal distension, dyspnoea, pain between his shoulder blades and no recent chest trauma. In the ED he developed progressive dyspnoea and shock (blood pressure 81/62 mmHg, heart rate 130/min). Thoracic aortic dissection or cardiac tamponade was suspected. Chest X-ray showed a significant enlargement of the cardiac silhouette, known as the 'water bottle sign'*(figure 1)*.[1] This suggests a large amount of pericardial effusion and is seen in large chronic effusions, which was confirmed by computed tomography scan *(figure 2)*. The patient was admitted directly to the intensive care unit and percutaneous pericardiocentesis was performed. A catheter was inserted which directly drained serosanguineous fluid (haemoglobin 3.8 mmol/l), implying a haemorrhagic pericarditis. A total amount of 1150 ml serosanguineous fluid was drained in the first 24 hours. After drainage and fluid resuscitation with blood transfusions, he showed rapid clinical improvement. Additionally, the patient's INR was above the therapeutic range (5.6) due to phenprocoumon use, which was corrected with 4-factor prothrombin complex concentrates and vitamin K. He developed anuric renal failure for which continuous venovenous haemofiltration was applied. After three days the patient was discharged in a stable condition to the cardiology ward.

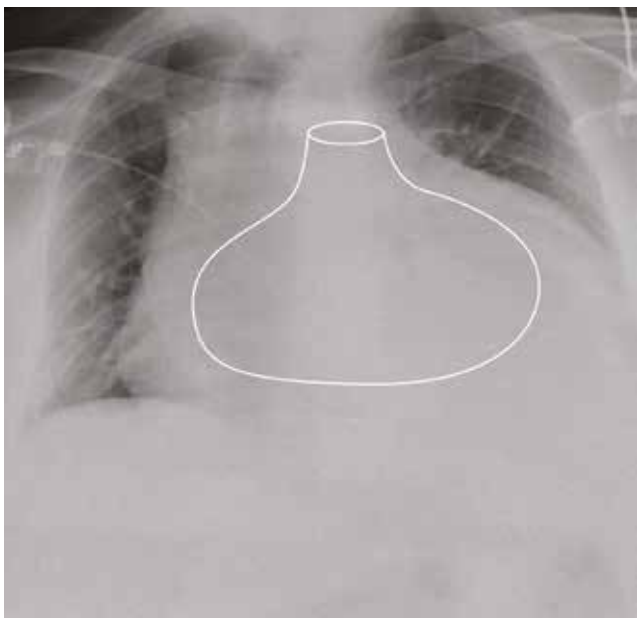

Figure 1. Chest X-ray of the patient demonstrating the 'water bottle sign'

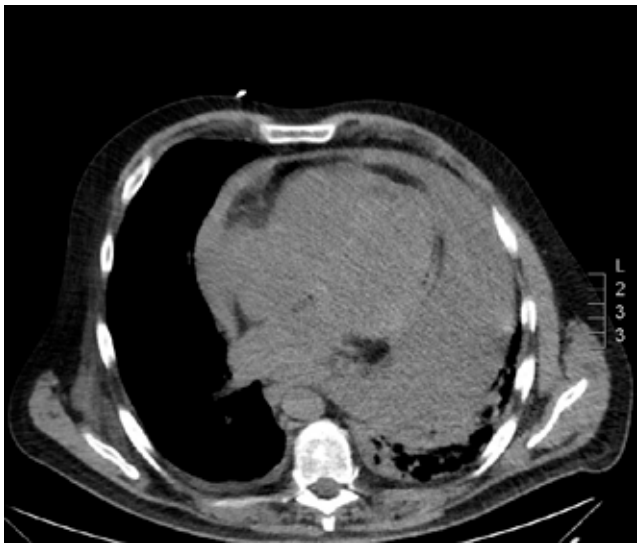

Figure 2. CT scan showing a large pericardial effusion

Diagnosis

Pericardial effusion is classified by its onset, distribution, haemodynamic impact (e.g. cardiac tamponade) and composition (exudate, transudate, blood).[2] The most important causes of pericardial effusion are infectious and idiopathic diseases. Patients with haemorrhagic pericardial effusion have a different aetiology and establishing the cause is essential as it

can reveal a serious underlying condition. The most common causes are iatrogenic, malignancy, post-pericardiotomy syndrome, complication of a myocardial infarction (due to free wall rupture and thrombolysis), uraemia, aortic dissection, pericarditis, trauma and tuberculosis.[3]

Clinical evaluation in patients with (haemorrhagic) pericardial effusion consists of electrocardiography (ECG), a chest X-ray, echocardiography and laboratory analysis of the pericardial fluid to determine the cause of the effusion. In this patient, pericarditis was suspected as the underlying cause of the haemorrhagic pericardial effusion as laboratory tests showed elevated C-reactive protein (114 mg/l) and leucocytes (15.8/nl). Therefore, the first step in the diagnostic process was to culture the pericardial fluid, blood and urine samples and to test for viruses and tuberculosis. All cultures proved to be negative and the serological tests for tuberculosis, HIV and cytomegalovirus also showed negative results. Neoplastic pericarditis was considered but the pericardial fluid showed only benign cells. The ECG and transthoracic echocardiography did not reveal a myocardial infarction. Considering the patient's history of gout, the next step was to consult a rheumatologist. The absence of clinical features and specific tests (ANA, ANCA, rheumatoid factor) ruled out rheumatic diseases. Uraemic pericarditis is observed in patients with advanced renal failure requiring dialysis; uraemia as a cause of pericarditis/pericardial effusion was considered possible (moderately elevated urea level (16.5 mmol/l) on admission).[4] The specific aetiology of this patient's haemorrhagic pericardial effusion was never confirmed. He was treated with colchicine empirically and discharged after one week.

The patient returned for a follow-up visit after two weeks, he was experiencing fatigue and dizziness. Echocardiography examination revealed a persisting small amount of pericardial effusion. After one month he was fully recovered.

Disclosures

All authors declare no conflict of interest. No funding or financial support was received.

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