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Case Report

Concurrent pericardial and pleural effusions: a double jeopardy☆

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Abstract A 19-year-old man with large malignant pleural and pericardial effusions with tamponade physiology and signs of congestive heart failure presented for emergent subxiphoid pericardial window. Surgical drainage of the pericardium was complicated by a paradoxical cardiovascular collapse that failed to respond to pressors and intravenous fluids. Suspecting a pericardial perforation, a median sternotomy was performed and revealed an intact heart. The arterial pressure was promptly restored after drainage of the pleural effusion. It is proposed that, in patients presenting with tamponading pericardial and pleural effusions, drainage of the pleural effusion be given priority. The pathophysiology of low cardiac output states resulting from pericardial and large pleural effusion is discussed and the literature reviewed.

1. Introduction

Cardiac tamponade is a medical emergency caused by the accumulation of fluid in the pericardium, resulting in compromised cardiac filling. Image-assisted drainage by needle paracentesis or evacuation by subxiphoid pericardial window normally safely ameliorates the condition. Pericardial effusion with neoplastic involvement of the pericardium occurs in 23% of patients with malignancies but may also result from multiple other causes such as inflammatory or infectious disease [1]. Rapid accumulation of only 60 to 100 mL of blood in the pericardium due to trauma to the heart or the coronary vessels can cause compression of the low-pressure myocardial chambers, restrict diastolic filling, and cause cardiogenic shock [2,3]. Considerably larger volumes are needed to cause hemodynamic impairment in subacute accumulations of pericardial fluid. Ongoing accumulation of fluid in the enclosed pericardial space progressively restricts cardiac filling, thus resulting in "tamponade."

Large pleural effusions alone or in association with a small pericardial effusion can also lead to significant compression of the right atrium (RA) [4,5], right ventricle (RV) [4,6,7], or left ventricle (LV) [4,6,8] and interfere with diastolic filling. Similar to pericardial effusions, the degree of hemodynamic impairment caused by pleural effusion is not a discrete occurrence but represents a spectrum that can ultimately present with a clinical picture of tamponade physiology.

We present a case of surgical drainage of pericardial effusion complicated by unexpected cardiovascular collapse

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after evacuation of the pericardial effusion possibly caused by dynamic obstruction of the pulmonary arteries. The circulation was restored after drainage of a large right-sided pleural effusion.

The patient was contacted after discharge from the hospital and gave a written permission to publish this report.

2. Case description

A 19-year-old previously healthy man presented at the urgent care clinic with a 2-week history of cough, chills, and fever. Failing to respond to oral antibiotics and with worsening symptoms of shortness of breath, weight gain, nausea, and vomiting, the patient was taken to the emergency department of the local hospital where the provisional diagnosis of sepsis with pneumonia was made. Because further diagnostic work-up demonstrated lower extremity deep vein thrombosis and a tamponading pericardial effusion, the decision was made to transfer the patient to our institution for further evaluation and management.

Repeat transthoracic echocardiography (TTE) showed a "swinging heart" within a large, circumferential pericardial effusion, abnormal septal motion, RV early diastolic, and RA late diastolic collapse with estimated LV ejection fraction (EF) of greater than 60% (Video still 1, Video clip 1). A 12-lead electrocardiogram demonstrated sinus tachycardia of 134 beats per minute (bpm) with characteristic low-voltage QRS complexes. Chest radiography confirmed a right-sided pleural effusion and an enlarged, globular cardiac silhouette (Fig. 1*A*). Because of lower extremity edema, a venous duplex scan was performed, and a right calf deep venous thrombosis and bilateral saphenous vein thrombus were detected. Heparin was withheld due to the pending surgical procedure.

At the time of preoperative evaluation, the patient was sitting upright and was dyspneic with a respiratory rate in mid-20s but could answer questions appropriately. His oxyhemoglobin saturation (SpO_2) was 93% on 6 L of O_2 via face mask. In addition to difficulty in breathing, the patient reported a weight gain of 7 kg over the past week. (His admission weight was 107 kg; normal weight, 100 kg; height, 185 cm.) The patient's neck veins were distended, and the skin was warm and clammy with a slight pitting edema of the upper and lower extremities. His blood pressure (BP) was 110/90 mm Hg, and his heart rate varied between 130 and 150 bpm. The radial pulse virtually disappeared during inspiration (Fig. 2). Auscultation revealed attenuated heart sounds without rubs or added sounds, and breath sounds were diminished over the right chest. Hematocrit was 43.6%, white cell count was 16.8, and serum electrolytes were within normal limits. His liver function tests showed elevation in aspartate aminotransferase of 331 IU/L (reference range, 5-45), alanine aminotransferase of 368 IU/L (reference range, 5-60), total bilirubin 2.0 mg/dL (reference range, 0.1-1.2) all of which were within the normal limit 2 days prior. The prothrombin time (PT) was 15.3 (reference range, 9.8-11.8), and troponin was less than 0.01 (normal, <0.02 ng/mL).

Because of signs of tamponade physiology and the patient's inability to lie supine, it was agreed with the surgical team that a subxiphoid pericardial window would be performed with local anesthetic infiltration and sedation. An additional large bore peripheral intravenous (IV) and a radial arterial cannula were inserted. The first set of vitals revealed BP of 140/75 mm Hg, heart rate of 128, and SpO_2 of 89% on 6 L of O_2 via face mask. Marked inspiratory attenuation of the arterial pressure trace affirmed our decision that spontaneous ventilation be maintained during the procedure (Fig. 2). After the case "time out" and the appropriate dose of IV antibiotics, 2 mg of midazolam and 0.5 mg of hydromorphone were administered. The incision site was now infiltrated with 10 mL of 0.25% bupivacaine with 1:200,000 of epinephrine. The respiratory rate continued in the low 20s, and the BP was maintained in the 125 to 140 mm Hg systolic range with SpO_2 at 91%. Sedation was maintained with intermittent boluses of ketamine (total 50 mg) and etomidate (20 mg in divided doses). Approximately 20 minutes into the case, a tight pericardial sac was identified, and 800 mL of dark bloody fluid was drained. This was immediately followed by an abrupt fall in arterial pressure to 20 to 30 mm Hg systolic and a drop in SpO_2 to 40% to 50% with marked central cyanosis. The

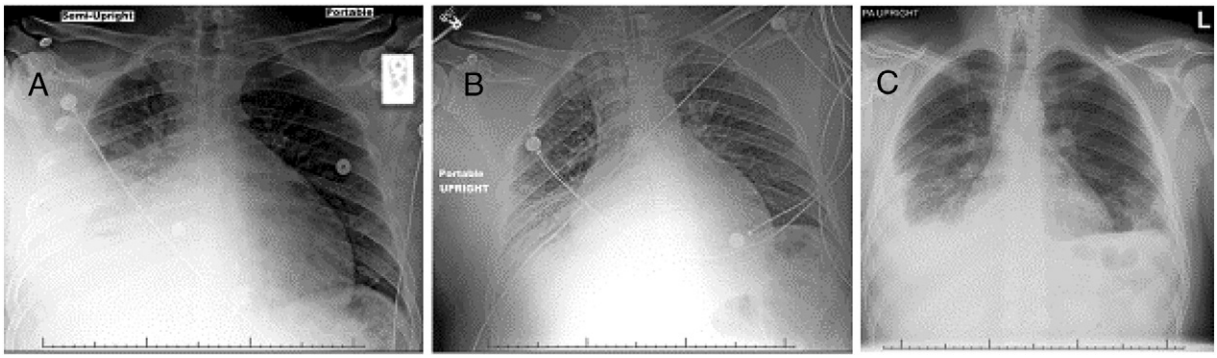

Fig. 1 Preoperative chest radiograph showing markedly increased, globular cardiac silhouette and right pleural effusion (A). Persistent cardiac enlargement on postoperative chest film (B), and for comparison, the normal heart size 17 days after surgery (C).