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Case Report

Transient acute left ventricular dysfunction post-pericardiocentesis for cardiac tamponade

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ABSTRACT

A rare but serious complication of pericardiocentesis is the development of transient left ventricular dysfunction. In this report, we present a case of a 65-year-old male patient with cardiac tamponade who suffered from acute left ventricular heart failure post-pericardiocentesis.

<**Learning objective:** Acute left ventricular dysfunction is a rare but serious complication of pericardiocentesis. However, there is lack of existing guidelines on pericardial fluid drainage and monitoring post-pericardiocentesis. To minimize the risk of the development of acute left ventricular dysfunction, the authors propose that every pericardiocentesis should be guided by predetermined parameters, e.g. rate of drainage, while taking into account each patient's physical profile.>

Introduction

Acute left ventricular heart failure is an uncommon but serious complication of pericardiocentesis. However, its exact mechanism remains unknown. In this report, we present a case of a 65-year-old male patient with hemopericardium and cardiac tamponade who developed acute left ventricular heart failure after pericardiocentesis.

Case report

A 65-year-old male patient was admitted to our institution with a 1-month history of intermittent lower chest discomfort and dyspnea that was worse on exertion. His past medical history was unremarkable except for mild gastritis, diverticulosis, and a gradual loss of weight of 4 kg over the past 2 months. He was a chronic smoker of 50 pack years but did not have diabetes, hypertension, or dyslipidemia.

On physical examination, blood pressure, heart rate, and respiratory rate were within normal ranges. Cardiovascular examination was unremarkable. Significantly, the patient did not have muffled heart sounds or raised jugular venous pulsations. Electrocardiogram showed sinus rhythm with low voltage QRS complexes. Chest X-ray (Fig. 1A) revealed an enlarged cardiac silhouette with mild pulmonary venous congestion. Routine blood tests were largely unremarkable except serum sodium of 128 mmol/L and hemoglobin of 11.5 g/dl. Serial cardiac enzymes were within normal ranges.

Transthoracic echocardiogram (Fig. 1B) demonstrated a large circumferential pericardial effusion with right atrial diastolic collapse. The echocardiographic measurements of the pericardial effusion were posterior to left ventricle = 2.1 cm; adjacent to left ventricle apex = 2.8 cm; anterior to right ventricle = 2.8 cm; adjacent to right ventricle = 1.7 cm. The inferior vena cava was plethoric with diminished respiratory variation. Left ventricular systolic function was preserved, and there was no observable regional wall motion abnormality. The ejection fraction was estimated at 60–65%. Uncomplicated therapeutic pericardiocentesis under fluoroscopic guidance was performed and 700 ml of hemoserous fluid was drained. A pericardial pigtail catheter was left to drain the remaining pericardial fluid.

Immediately post-pericardiocentesis, blood pressure was 136/82 mmHg, compared to 120/60 mmHg pre-procedure. Eight hours post-pericardiocentesis, the patient developed hypotension with a blood pressure of 80/60 mmHg, pulse rate of 85 beats per minute, respiratory rate of 21 breaths per minute and SpO_2 100% on 2 L/min of intranasal oxygen. He was resuscitated with intravenous crystalloids and started on intravenous dopamine infusion. Electrocardiogram (Fig. 2C) that was repeated showed sinus rhythm with new-onset hyperacute T waves in the antero-lateral leads, compared to baseline (Fig. 1C). Serum cardiac

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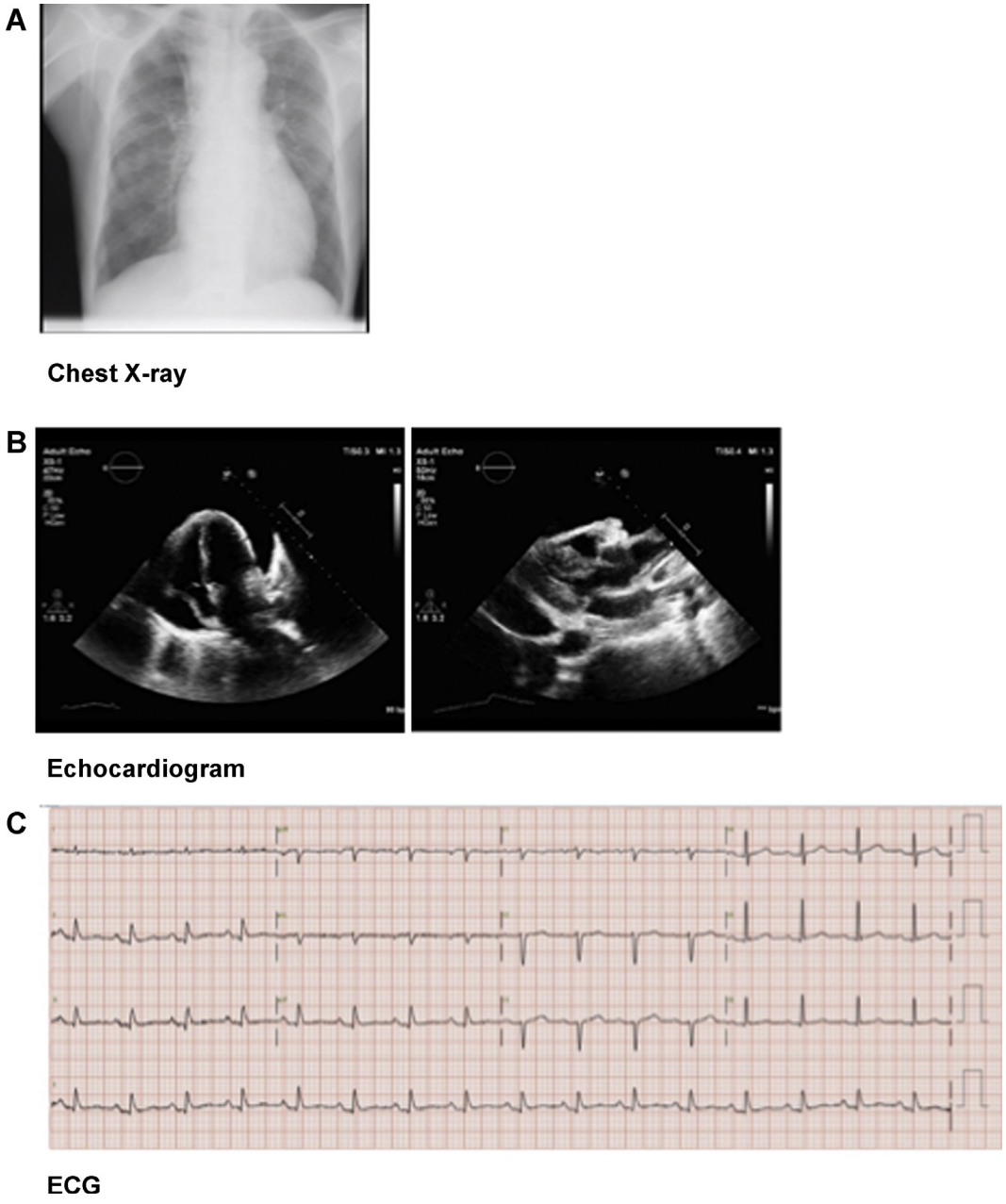

Fig. 1. (A) Chest X-ray on admission. (B) Left – apical 4-chamber view showing circumferential large pericardial effusion causing right atrial collapse; right – parasternal long-axis view. Large pericardial effusion and normal wall motion and contractility of the left ventricle as noted by the good septal and posterior wall thickening. (C) Baseline electrocardiogram. Sinus rhythm with low-voltage QRS complexes.

enzymes were performed and showed a creatine kinase-MB level of 40.1 μg/L (14.0 μg/L pre-procedure) and a troponin T level of 0.48 U/L (0.01 U/L pre-procedure). Repeat renal panel did not show any renal impairment. Liver panel and brain natriuretic peptide were not repeated. Repeat chest X-ray (Fig. 2A) showed airspace shadowing in bilateral lung bases.

Transthoracic echocardiogram was repeated at 15 h (Fig. 2B) after the first echocardiogram showed reduced left ventricular systolic function with an ejection fraction estimated at 35–40% and new-onset akinesia of the mid to apical anterior, apical lateral, and midanteroseptal regions. There was minimal residual pericardial effusion and no evidence of myocardial perforation. Cardiogenic shock secondary to acute myocardial infarction was considered. Emergent coronary angiography performed on the same day showed normal coronary arteries and dyskinesia of the anterolateral wall on ventriculogram.

Just 4 days after the acute left ventricular dysfunction, a repeat transthoracic echocardiogram (Fig. 3A) showed normalization of left ventricular systolic function to 61% with no regional wall motion abnormality and a small residual circumferential pericardial effusion. Repeat electrocardiogram (Fig. 3B) at this point showed persistent inverted T waves in the anterolateral leads. Cytology and immunochemistry staining of the pericardial fluid were consistent with that of a metastatic adenocarcinoma, suggestive of an upper gastrointestinal or pancreatobiliary primary.

Discussion

Case series demonstrated that malignancy accounted for between 13% and 23% of etiologies of pericardial effusion [1]. Cardiac tamponade occurred in approximately 60% of those with