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Subacute pericardial tamponade presenting with hypertension

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Abstract

Presentation
Pericardial tamponade is a potentially fatal condition requiring urgent intervention. A 64-year-old lady presented to the emergency department with abdominal pain and syncope, with noted subxiphoid tenderness and peripheral oedema.

Diagnosis
The patient was hypertensive at presentation and developed respiratory distress and anuria.

Treatment
Bedside echocardiography revealed a 6cm pericardial effusion with right ventricular diastolic collapse and urgent pericardiocentesis drained 1800mls within 24hrs. Clinical features can be variable and patients can present with hypertension prior to cardiovascular collapse.

Discussion
Tamponade diagnosis is dependent on urgent echocardiography, which is increasingly adopted among ICU doctors.

Introduction

In this report we discuss the physiology of tamponade presenting with hypertension and the basic echocardiographic features of cardiac tamponade.

Case Report

A 64-year-old lady presented to our emergency department with a several week history of progressive abdominal pain and episodes of syncope, she is noted to have subxiphoid tenderness and peripheral oedema. The patient was hypertensive and developed respiratory distress and anuria during admission.

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ECHO revealed a 6cm pericardial effusion with right ventricular (RV) diastolic collapse and small left ventricular cavity size. The inferior vena cava was distended with absent respiratory variation. Central venous pressure measured 62mmHg and pulsus paradoxus was evident. Blood gas analysis revealed a serum lactate of 11.3mmol.L^{-1}.

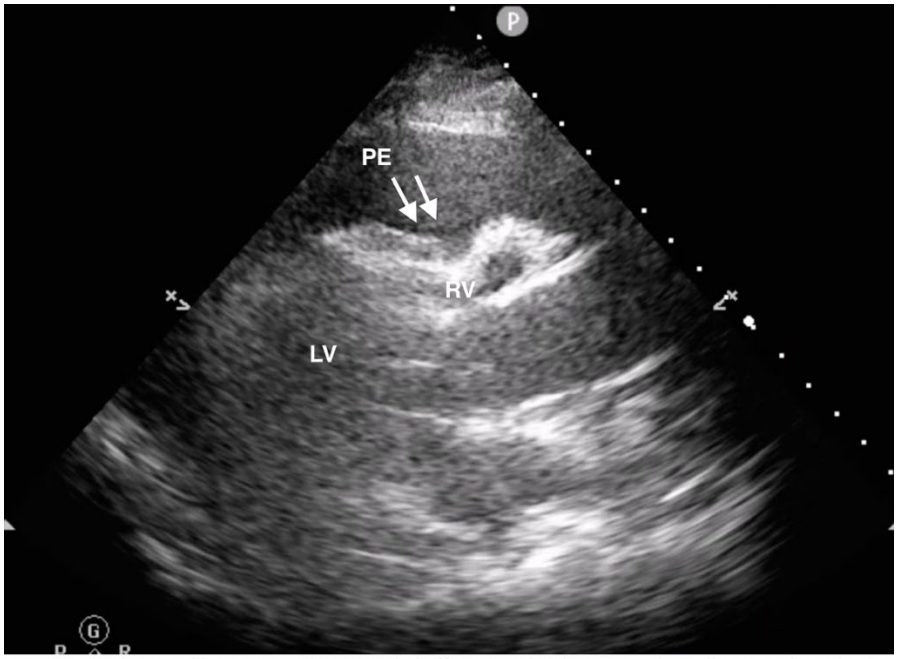

Figure 1 – parasternal long axis with pericardial effusion and right ventricular collapse in diastole. PE – pericardial effusion, RV – right ventricle, LV – Left ventricle, arrows depicting ventricular collapse during diastole.

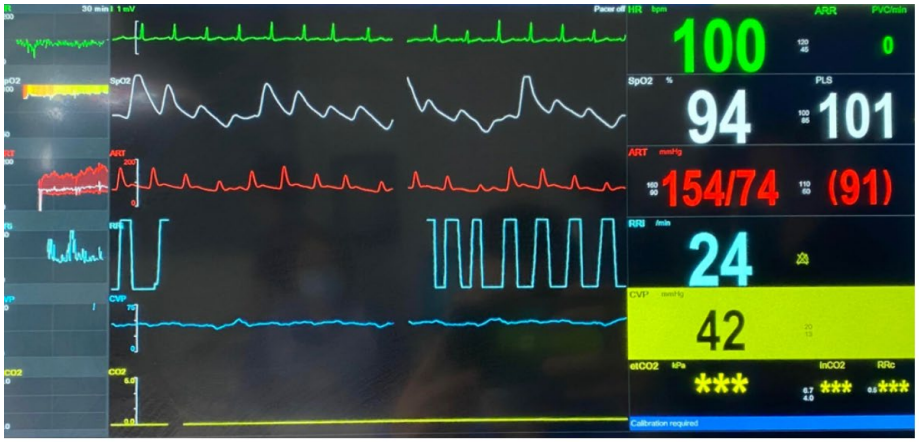

Figure 2 – ICU monitor displaying hypertension, pulses paradoxical and severely elevated CVP

Ultrasound guided pericardiocentesis drained 500mls. Subsequently, the patient was sedated and mechanical ventilation commenced. Renal replacement therapy was required for management of acidosis and hydrocortisone, colchicine and antibiotics were empirically

started. No cardiovascular support was required and the patient was liberated from mechanical ventilation after six days.

Pericardiocentesis drained 1800mls within twenty-four hours and continued to drain an average of 400mls per day during her ICU stay. Histology revealed malignant epithelial cells.

Discussion

The presentation of a pericardial effusion is dependent on it's acuity[1]. In acute cardiac tamponade there is a rapid rise in pericardial fluid volume pressure, classically found in haemorrhagic tamponade e.g. coronary or aortic dissection[2].

In subacute tamponade fluid accumulates slowly, allowing the parietal pericardium to expand and maintain a low pressure. However, as fluid accumulates the pericardium may no longer be able to compensate and further volume increases may result in dramatic pericardial pressure changes. This is associated with infections, uraemia and malignancy[2].

There is compensatory increased peripheral vascular resistance to maintain mean arterial pressure when cardiac output is reduced during tamponade. Brown et al.[3] identified 6 of 18 tamponade patients were hypertensive with a mean systolic blood pressure of 176(+/-26)mmHg. Following pericardiocentesis, peripheral vascular resistance fell in all patients resulting in reduced arterial pressure in the hypertensive group and elevated arterial pressure in the hypotensive group[3].

ECHO is essential in diagnosis. RV collapse occurs when the intra-cardiac pressure falls below the pericardial pressure, the RV wall is thin and vulnerable to pressure differences. The RV outflow tract collapses first followed by the basal segment. RV collapse is less sensitive but more specific than right atrial (RA) collapse[4]. If tamponade is as a result of regional collection e.g. blood clot post-cardiac surgery, then RV collapse may not be present.

RA pressure is lowest at early systole and collapse may be the first ECHO feature however hypovolaemia may also present similarly. Absence of either RA or RV collapse has a 90% negative predictive value for tamponade[4].

A plethoric inferior vena cava, >2.1cm, with less than 50% variation through the respiratory cycle, is suggestive of venous hypertension. This is a sensitive for cardiac tamponade however poorly specific[4].

Pulsus paradoxus represents exaggerated respiratory variation in systolic blood pressure. On inspiration, reduced pulmonary vascular resistance results in increased blood velocity through the tricuspid valve. In tamponade, end diastolic pressures of the right and left ventricles have equalised due to increased pericardial pressure, chambers are competing for space within the heart so during inspiration the interventricular septum deviates towards the left ventricle. Therefore, during inspiration there is increased blood flow through the right heart but less through the left heart and a drop in systolic blood pressure occurs. Pulse-wave doppler can identify a 25% decrease in velocity across the mitral valve and 40% increased velocity of blood flow across the tricuspid valve consistent with tamponade physiology[4].

Sub-acute tamponade presenting with hypertension is a rare feature. The absence of the classical Beck's triad placed this patient at risk of deterioration particularly around intubation and positive pressure ventilation. Timely use and interpretation of diagnostic imaging allowed early intervention and avoided significant instability for the patient.

Declarations of Conflicts of Interest:
None declared.

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