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CASE REPORT
Heart failure

A case report of cardiac tamponade after a road accident: think beyond trauma

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Background Cardiac tamponade is a life-threatening compression of the heart caused by the accumulation of fluid in the pericardial sac. Although central venous catheters (CVCs) are essential in modern medicine, they carry a certain risk of complications including cardiac tamponade.

Case summary A 12-year-old female was involved in a road accident reporting multiple severe traumatic injuries, including a left humerus fracture and subdural haemorrhage. After 2 days in the intensive care unit, she suddenly developed hypotension and cardiac tamponade was diagnosed. Analysis of the pericardial fluid showed high glucose levels comparable to the parenteral nutrition that she was receiving. Retraction of the CVC allowed resolution of the effusion.

Discussion Cardiac tamponade is a rare but serious adverse event after CVC insertion, mostly among younger patients. Awareness of this risk allows physicians to promptly recognize and treat this dangerous complication.

Keywords Central venous catheter • Cardiac tamponade • Parenteral nutrition • Cardiac intensive care • Case report

ESC curriculum 6.4 Acute heart failure • 6.6 Pericardial disease • 7.3 Critically ill cardiac patient • 7.1 Haemodynamic instability • 9.2 Trauma to the aorta or the heart

Learning points

- Cardiac tamponade is a rare but serious complication after central venous catheter insertion. Infants and children are the most likely to experience this complication.
- Analysis of pericardial fluid is important to obtain the correct diagnosis and thus provide the correct treatment.

Introduction

Cardiac tamponade is a life-threatening compression of the heart caused by fluid accumulation in the pericardial sac[1] and can be complicated by right ventricular failure and obstructive shock.[2] Up to 20% of cardiac tamponade cases are caused by iatrogenic pericardial effusion.[3,4] Central venous catheters (CVCs) are common among critically ill patients and represent an essential tool to administer fluids, drugs, and nutrition and to obtain blood samples. Although safe in the vast majority of cases, CVCs carry a certain risk of complications. Obstructive shock must be included among the differential diagnoses when a patient with a CVC suddenly develops hypotension and tachycardia as, even if relatively uncommon, it could be the result of cardiac wall perforation and subsequent cardiac tamponade.

We report a case of a young woman who developed obstructive shock secondary to CVC-induced atrial wall perforation. Echocardiographic evaluation and analysis of the pericardial fluid allowed to reach the correct diagnosis and to provide the best treatment for the patient.

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Summary figure

Time 0	Road accident with skull and humerus fracture
	Intensive care unit (ICU) admission and CVC placement
+2 days	Tachycardia, hypotension, and anuria.
	Diagnosis of cardiac tamponade and subsequent pericardiocentesis with drainage of milky fluid
	Pericardial fluid analysis revealed high glucose levels
	Retraction of CVC
	Progressive reduction of the pericardial effusion
+3 days	Multiorgan failure (predominantly kidney and liver)

Case presentation

A 12-year-old female with no relevant medical history was involved in a major road accident. The patient was transported to the emergency department of a local hospital where she was stabilized and intubated. The patient was later transferred to the major trauma centre of the region. Upon arrival, her vital signs were as follows: heart rate 109 b.p.m., blood pressure (BP) 158/85 mmHg, respiratory rate 16 breaths per minute, and body temperature 36°. Physical examination revealed left frontoparietal swelling, left upper arm haematoma, no heart murmurs or pericardial rubs, bilateral vesicular breath sounds, and no abdominal tenderness or guarding. Total body computed tomography (CT) revealed right frontotemporal and left frontoparietal subdural haemorrhage, right subarachnoid Sylvian fissure haemorrhage, right temporal bone fracture, and left humerus fracture. The patient was therefore admitted to the ICU for further care. Here, an arterial line for invasive BP control and a right subclavian (7.8 in, 8.5 Fr) CVC for drug infusion were placed under ultrasound guidance. The patient remained haemodynamically stable until the second day of hospitalization, when she suddenly developed sinus tachycardia, refractory hypotension (down to 80/40 mmHg, unresponsive to maximal doses of vasopressors), and anuria. Haemogas analysis showed a marked increase in lactic acid [up to 4.7 mmol/L (normal value <2 mmol/L)].

Given the recent major trauma, the main differential diagnoses of haemodynamic deterioration included (i) haemorrhagic shock, (ii) neurogenic shock, (iii) cardiac tamponade, and (iv) tension pneumothorax.

Under suspicion of haemorrhagic shock due to active occult bleeding, the patient underwent thoracoabdominal CT angiography, which excluded active bleeding but suggested the presence of pericardial effusion. A cardiology consult was therefore requested: physical examination revealed pulsus paradoxus and bedside transthoracic echocardiography showed normal biventricular function with large circumferential pericardial effusion and signs of haemodynamic compromise. On transthoracic echocardiography, the tip of the CVC was visible at the level of the right atrium.

Given the rapid deterioration in her vitals, transoesophageal echocardiography was not performed. Subcostal pericardiocentesis was performed, and 200 mL of milky fluid was collected and sent for chemical, microbiological, and cytological analyses. A pericardial catheter was positioned in the pericardial sac and continued to drain ~50 mL/h of milky fluid. A review of the CT images excluded thoracic duct rupture. Cultural analysis of the pericardial fluid showed the presence of *Enterococcus faecalis* while chemical analysis revealed the following: proteins 0.3 g/dL, albumin <1 g/dL, lactate dehydrogenase 86 U/L, and glucose 986 mg/dL (*Table 1*). Surprisingly, these characteristics were similar to those of the parenteral nutrition that the patient was receiving. Moreover, a review of the chest radiograph (*Figure 1A* and *C*) and CT angiography (*Figure 2*) showed how the tip of the CVC pointed towards the right atrium free wall. A diagnosis of atrio-pericardial fistula was made. The CVC was retracted under transthoracic echocardiographic guidance without complications, and the pericardial catheter stopped draining the milky fluid. Chest radiography confirmed appropriate repositioning of the device (*Figure 1B* and *D*). The day after CVC retraction, 45 mL of haematic pericardial effusion was drained. To avoid bleeding at the level of the atrial wall breach, we aimed to obtain low central venous pressure. In the coming days, the pericardial effusion resolved but the patient progressively developed multiorgan failure with kidney and liver involvement [creatinine up to 3.3 mg/dL (normal value for age < 0.85 mg/dL) and spontaneous international normalized ratio (INR) up to 2.8 (normal value 1)]. Given the resolution of pericardial effusion and multiorgan failure, anti-inflammatory therapy for the prevention of pericarditis was not initiated. The pericardial catheter was removed 2 days after pericardiocentesis without complications. The patient survived multiorgan failure and continued neurological care. After a long hospital stay, the patient was transferred to a neurological rehabilitation centre.

Table 1 Biochemical characteristics of the pericardial effusion

Biochemical analysis of the pericardial fluid	
Proteins	0.3 g/dL
Albumin	<1.0 g/dL
Lactate dehydrogenase	86 U/L
Glucose	986 mg/dL

Discussion

Central venous catheters are commonly used in modern medicine, mostly in critically ill patients. Approximately 8% of hospitalized patients require CVC, and >5 million CVCs are inserted annually in the USA.[5,6] Although essential for haemodynamic monitoring, delivery of medications, and nutritional support, CVC insertion carries a risk of complications that can be classified as early (<24 h) or late (>24 h after insertion)[7] and can be further divided[6] into mechanical (5–19% of the patients),[8–10] infectious (5–26%),[8,11,12] or thrombotic (2–26%).[8]

While the most common CVC-related cardiac injury is dysrhythmia induced by cardiac irritation, cardiac tamponade can also occur in both early and late time periods.[7] Guidewire or dilator injuries are more likely to present in an acute manner, while later onset is usually associated with catheter erosion and subsequent necrosis of the cardiac wall.[7] Although rare in adults, a review of the literature shows that the incidence of pericardial effusion and cardiac tamponade due to CVC is higher in infants and children,[13,14] presumably because of the use of devices that are not adequately sized in the paediatric population. Moreover, in infants, it has been shown that the fluid found in CVC-associated pericardial effusion is often consistent with the infusate. Several mechanisms could be implicated in this phenomenon, ranging from frank perforation that spontaneously seals to tip adhesion to the myocardial wall and subsequent diffusion into the pericardial space.[13]

Knowledge of this rare but serious complication of CVCs is of paramount importance for cardiologists, as pericardial effusion represents a frequent reason for cardiology consultation. Analysis of the pericardial fluid and comparison with the chemical characteristics of the ongoing infusions allows to diagnose CVC perforation and to provide the potentially life-saving treatments.

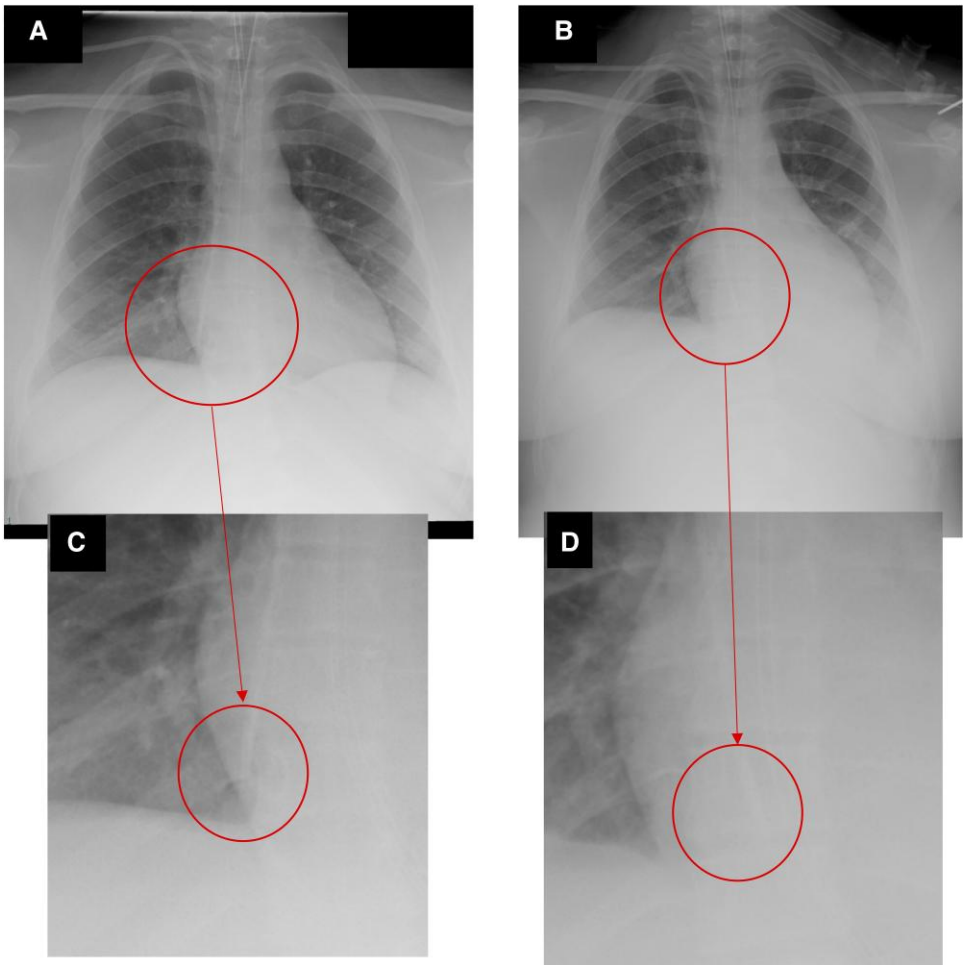

Figure 1 (*A* and *C*) Chest X-ray of the patient performed the morning before the diagnosis of cardiac tamponade. Notably, the tip of the central venous catheter points to the free wall of the right atrium. (*B* and *D*) Chest X-ray of the patient performed after retraction of the central venous catheter.

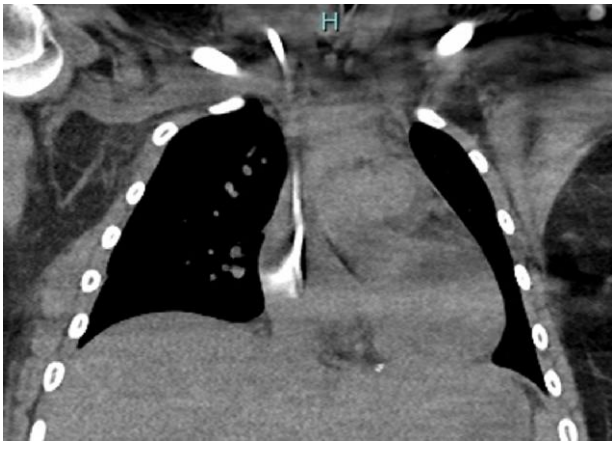

Figure 2 Chest computed tomography image of the patient after cardiac tamponade diagnosis. Even in the presence of artefacts, it is possible to appreciate how the tip of the catheter points towards the free wall of the right atrium.

Lead author biography

Giovanni is a cardiologist and currently a PhD student at the University of Padua. Clinically, he is interested in heart failure, cardiac involvement of systemic diseases, and coronary microcirculation. His research interest also includes microvascular dysfunction after acute coronary syndromes and cardiac allograft vasculopathy. During his cardiology fellowship, Giovanni did clinical rotations at Treviso Hospital, where he worked in the cardiac catheterization lab and in the cardiac intensive care unit.

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Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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