CASE REPORT

Low-pressure cardiac tamponade masquerading as severe sepsis diagnosed with a bedside ultrasound and as the initial presentation of malignancy

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Objective: We report a patient with low-pressure cardiac tamponade masquerading as sepsis and as the initial presentation of malignancy. A quick diagnosis was done by the intensivist performing a bedside ultrasound.

Background: The diagnosis of low-pressure cardiac tamponade is a challenge because the classic physical signs of cardiac tamponade can be absent. It is made even more challenging when the vital sign changes and physical examination findings mimic severe sepsis. One of the benefits of a bedside ultrasound in the assessment of a patient with an initial diagnosis of severe sepsis or septic shock is the rapid diagnosis of cardiac tamponade if it is present.

Data Source and Synthesis: A 55-year-old male presented to the emergency department with weakness, cough, and syncope. His examination was notable only for dusky mottling of his cheeks, chest, and neck. Specifically, there was no jugular venous distension or pulsus paradoxus. A chest radiograph showed a right upper lobe infiltrate, whereas his electrocardiogram showed only sinus tachycardia. His white blood cell count and lactic acid were elevated. The sepsis protocol was started and a bedside ultrasound revealed signs of cardiac tamponade. The patient immediately improved after a pericardiocentesis. Analysis of the pericardial biopsy revealed adenocarcinoma, later determined to be from a pulmonary primary source.

Conclusions: Because low-pressure cardiac tamponade is life-threatening and difficult to diagnose, evaluation of the pericardium with a bedside ultrasound should be considered in patients with syncope, severe sepsis, or shock.

Keywords: cardiac tamponade; bedside ultrasound; sepsis; pericardium effusion

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Low-pressure cardiac tamponade has been described as a form of cardiac tamponade in which a comparatively low pericardial pressure results in cardiac compression in patients with intravascular fluid depletion (1, 2). The central venous pressure is normal or decreased and hence jugular venous distention is absent (1–3). Pulsus paradoxus is another clinical sign of tamponade that is often absent (2). Low-pressure cardiac tamponade is an uncommon form of cardiac tamponade, as is the occurrence of cardiac tamponade as the first manifestation of malignancy (1, 4). Also, cardiac tamponade can be initially misdiagnosed as sepsis or septic shock (5, 6), and a high index of suspicion is necessary for appropriate management. Currently, the availability of bedside ultrasound in the emergency department (ED) has allowed for more rapid diagnosis of cardiac tamponade (7), and it is suggested that a bedside ultrasound should be done in patients diagnosed with sepsis or shock (7, 8). Experts argue passionately for incorporating echo training into intensive care fellowship requirements (8). We report a peculiar case of a low-pressure cardiac tamponade that presented as the first manifestation of malignancy, initially misdiagnosed as severe sepsis and quickly diagnosed by a bedside ultrasound.

Case report

This is a 55-year-old male who presented to the ED with weakness, light-headedness, muscle aches, non-productive cough, and wheeze for 5 days. He did not measure his temperature at home but he noted frequent shivering.
There was no complaint of shortness of breath or chest pain. He had a previous evaluation at the ED 2 days before this visit with the same symptoms. The patient was discharged after intravenous hydration and with a Penicillin V prescription because no other significant abnormalities were found besides a positive rapid test for Group A Streptococcus. His medical history was positive for asthma, hypertension, and diet-controlled type 2 diabetes. He was taking lisinopril and hydrochlorothiazide, and had a 25 pack-year smoking history but quit 16 years ago. The family history was non-contributory. The patient had syncope while already in the ED. During his examination, he was lying supine with a blood pressure of 109/87 mmHg without pulsus paradoxus, a heart rate 109 beats per minute, respiratory rate of 24 breaths per minute, temperature of 36.5°C, and his oxygen saturation was 100% on room air. His skin was notable for mottling of his cheeks, chest, and neck. There was no jugular venous distension or lymphadenopathy. He had clear lungs, no distant cardiac sounds, a regular heart rate and rhythm, normal S1/S2, no heart murmur, and an unremarkable abdomen and extremities. A portable chest radiograph showed a right upper lobe infiltrate and a borderline enlarged cardiac silhouette, but it was noted by the radiologist that it could be artifactual given the antero-posterior projection of the chest radiograph (Fig. 1). The electrocardiogram showed only sinus tachycardia and no tall QRS complex. Specifically, electrical alternans was absent (Fig. 2). The white blood cell count was 16.6 \times 10^3/\mu\text{L} and hemoglobin 13.4 g/dL. The basic metabolic panel showed sodium 129 mmol/L, potassium 4.2 mmol/L, carbon dioxide 21 mEq/L, creatinine 1.0 mg/dL, and glucose 152 mg/dL. Cardiac enzymes were normal, B-natriuretic peptide 915 pg/mL, and his lactic acid level was 3.8 mmol/L.

Antibiotics were started and fluid resuscitation was initiated out of concern that the patient’s constellation of symptoms represented severe sepsis. However, three liters of normal saline did little to improve the patient’s symptoms and also did not change his blood pressure, heart rate, or dusky mottling. There was no identifiable jugular venous distention after the initial fluid resuscitation. The intensivist was consulted and performed a bedside ultrasound that revealed a pericardial effusion with diastolic collapse of the right atrium (Fig. 3) and right ventricle (Fig. 4). The ejection fraction was normal and no other significant abnormalities were found; however, the subcostal view was suboptimal and a precise evaluation of the inferior vena cava was not possible. The patient then experienced another syncopal episode while supine on the ED stretcher, and the decision was made to proceed with

**Fig. 1.** Chest X-ray at supine position showing possible enlarged cardiac silhouette.

**Fig. 2.** Electrocardiogram showing no tall QRS complex, but without meeting criteria for low QRS voltage.
bedside pericardiocentesis. Two hundred milliliters of bloody pericardial fluid was drained and the patient’s symptoms and cyanosis immediately improved. Several hours later the patient continued symptom free, hemodynamically stable, and an open pericardiotomy and a pericardial window placement were performed. Repeat chest radiograph showed a smaller cardiac silhouette (Fig. 5) and a repeat electrocardiogram showed slightly increase in voltage of QRS complexes (Fig. 6). A CT scan showed a pericardial drainage tube well positioned, trace pericardial effusion, a right upper pneumonic infiltrate, and small bilateral pleural effusion. The analysis of the pericardial biopsy and pericardial effusion fluid revealed adenocarcinoma with an immune stain suggesting a pulmonary primary source. A bronchial brushing and washing was also suggested of adenocarcinoma. The final results of blood, urine, and bronchial-alveolar washing cultures were negative. The patient was discharged to follow up as an outpatient at the oncology clinic.

Discussion
Cardiac tamponade has classically been considered a syndrome in which cardiac chambers are compressed by a tense pericardial effusion, resulting in limited cardiac inflow and the typical findings of jugular venous distention, hypotension, and pulsus paradoxus (1). However, a form of cardiac tamponade in which a comparatively low pericardial pressure results in cardiac compression in patients with intravascular fluid depletion was clinically described in 1979 (1, 3). There is no established clinical definition of low-pressure cardiac tamponade and frequently the typical clinical findings of tamponade are absent (1, 3). This makes prompt diagnosis a challenge and accounts for the low reported prevalence.

Our patient could be fluid depleted because of poor fluid intake, the spreading malignancy, and the use of diuretics. Hemodynamic parameters improvement have been shown to respond to fluid challenge in cases of low-pressure cardiac tamponade, but our patient might have needed more than three liters of normal saline to have some improvement. Furthermore, the definitive treatment of a low-pressure cardiac tamponade is pericardiocentesis or pericardietomy.

Cardiac tamponade can be misdiagnosed as severe sepsis or septic shock (5, 6). This misdiagnosis might
delay urgent pericardiocentesis that could promptly relieve life-threatening cardiac tamponade. Our reported patient was initially diagnosed with severe sepsis because he was tachycardic and tachypneic; along with leukocytosis, an elevated lactic acid; and a chest radiograph that was suggestive of a right upper lobe pneumonia. After the pericardiocentesis, the patient’s symptoms improved and, together with negative cultures and additional tests, it became clear that the patient did not have an infection but an inflammatory response caused by the cardiac tamponade and a lung malignancy.

The initial presentation of malignancy as cardiac tamponade is uncommon (4). Metastatic spread of tumors to the pericardium is found in 11.6% of patients with known malignancy at postmortem examination (9), however the majority of patients have no or few symptoms and cardiac tamponade is even less frequent (4, 9). Malignant cardiac tamponade usually develops as an acute event superimposed on a large and slowly developing pericardial effusion (4). Consequently, a large cardiac silhouette usually will be present on the chest radiograph, which was not initially apparent in the reported patient.

Syncope is a common condition; however cough-induced syncope is a rare manifestation of pericardial effusion (10). Syncope associated with structural heart disease has a poor prognosis and the presence of syncope in the case reported was one of the reasons for a quick cardiac evaluation by a bedside ultrasound. Echocardiography was originally developed more than 50 years ago, but only after the late 1980s did some intensivists started using it as the preferred first-line technique for evaluation of patients with hemodynamic instability (8). Currently, the widespread availability of a bedside ultrasound in the ED has allowed an almost immediate cardiac ultrasound in patients with shock and a rapid diagnosis of cardiac tamponade (7, 8). Clinical findings suggestive of hemodynamically instability associated with ultrasonographic findings suggestive of diastolic cardiac collapse confirm the diagnosis of cardiac tamponade (7, 8). Specifically, the bedside echocardiographic findings suggestive of tamponade are a circumferential pericardial effusion with right atrial and ventricular diastolic collapse and a plethoric inferior vena cava with loss of respiratory variation.

**Conclusion**

This is a report describing a patient with low-pressure cardiac tamponade that had clinical findings suggestive of severe sepsis. He did not have any known risk factors for pericarditis and his pericardial tamponade was the initial presentation of a lung adenocarcinoma. A quick diagnosis was done by the intensivist using bedside ultrasonography when the patient was still in the ED.

Because low-pressure cardiac tamponade is life-threatening and difficult to diagnose, evaluation of the pericardium with a bedside ultrasound should be considered in patients with syncope, severe sepsis, or shock.

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