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A LATE DIAGNOSIS OF BACTERIAL PERICARDITIS

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ABSTRACT

Background: Cardiac tamponade in patients with bacterial pericarditis has a high mortality rate. A timely diagnosis is crucial and can only be achieved through repeated systematic clinical evaluation, the use of POCUS and awareness of cognitive biases in clinical reasoning.

Case description: A 56-year-old male was admitted to the emergency room with shortness of breath and chest pain. The patient became haemodynamically unstable and was treated for a septic shock. A POCUS showed signs of a cardiac tamponade, but these were not acknowledged as such. The diagnosis was delayed until the patient had a circulatory arrest. He was successfully resuscitated, and a pericardiocentesis showed a haemophilus influenzae pericarditis. The patient recovered well after pericardiocentesis and treatment with antibiotics.

Discussion: For unstable patients, it is essential to conduct systematic assessments at regular intervals and whenever there is a change in clinical status. Cardiac and lung POCUS have proved to be useful in undifferentiated shock patients. Importantly, pericardial effusion should always be ruled out. For every clinician, it is important to be aware of possible cognitive biases in clinical reasoning and to use strategies to mitigate these risks.

KEYWORDS

Bacterial pericarditis, cognitive bias

LEARNING POINTS

- The central venous pressure should be assessed in all shock patients before and after fluid resuscitation.
- POCUS should be performed on all patients in shock to evaluate central venous filling and rule out pericardial effusion.
- If any significant pericardial fluid is found, tamponade should be considered as a contributing cause of hypoperfusion.
- Pericardial fluid removal rapidly improves haemodynamic status; the patient will not recover unless this is done.
- Awareness of cognitive biases in clinical reasoning, and strategies to mitigate them, are essential because such biases can contribute to medical errors.

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INTRODUCTION

Cardiac tamponade occurs in 3.1% of all cases of pericarditis but in 26% of patients with bacterial pericarditis[1,2]. Cardiac tamponade in patients with bacterial pericarditis has a mortality rate between 10 and 41%[1].

A timely diagnosis is crucial and can only be achieved by repeated systematic clinical evaluation, the use of point of care ultrasound (POCUS), and awareness of cognitive biases in clinical reasoning. We demonstrate this in a patient who presented with fever and haemodynamic instability.

CASE DESCRIPTION

A 56-year-old male with no relevant medical history was admitted to the emergency room with two days of progressive shortness of breath and chest pain radiating to both arms and back. Physical examination was notable for a respiratory rate (RR) of 36/min on room air without hypoxemia, a blood pressure (BP) of 105/80 mmHg, a heart rate (HR) of 131/min and a body temperature of 37.6°C. The electrocardiogram and chest X-ray showed sinus tachycardia and a possible consolidation of the left lower lung.

One hour later the BP was 81/47 mmHg. Fluid resuscitation was started, blood and urinary cultures were taken, and cefuroxime and gentamicin were administered under the presumed diagnosis of septic shock. Laboratory results were notable for leucocytosis, leukocyturia and an elevated C-reactive protein level (339 mg/l). Five hours after presentation and 1.6 litres of fluid later, the intensive care (IC) department was consulted for refractory shock. Examination showed a diaphoretic patient with RR 32/min, HR 104/min, BP 60/40 mmHg, muffled heart sounds and cold extremities. A POCUS showed pericardial effusion (PE) and a distended, non-collapsing inferior vena cava. A re-evaluation of the first electrocardiogram showed PQ segment depression and ST segment elevation in the leads III and V1–3. The patient was admitted to the IC unit with the working diagnosis of septic shock due to a urinary tract infection with reactive pericarditis and possible cardiac tamponade. Noradrenaline infusion was started, and fluid therapy continued. The cardiologist was consulted for evaluation of the PE and if necessary pericardial drainage. Eight hours later, a transthoracic echocardiogram (TTE) was performed and evaluated by the attending cardiologist with the following conclusion: 1 cm PE, impaired relaxation and a distended inferior vena cava; no signs of tamponade were evident. Further analysis would follow later without other specifications. Ten hours after presentation the patient became hypoxemic. The abdomen was distended with high-pitched bowel sounds; an abdominal sepsis was considered. Twelve hours later, a computer tomography of the thorax and abdomen was performed in a search for a focus of the sepsis. This showed a PE with a heterogeneous aspect, compression of the right ventricle and pericardial thickening *(Fig. 1)*, thickening of the gallbladder wall and signs of duodenal ulcer without free abdominal air.

Metronidazole and vancomycin were added to the antibiotic regimen to cover anaerobic bacteria and methicillin-resistant *Staphylococcus aureus*. A surgeon was consulted and ruled out a perforation of a hollow viscus since free air was absent.



Figure 1. CT with contrast shows pericardial thickening, pericardial effusion and collapsing of the right ventricle.



Figure 2. Subcostal 4 chamber view cardiac ultrasound with 2.32 cm of pericardial effusion.

Sixteen hours after presentation, the patient went into circulatory arrest with pulseless electrical activity and received cardiopulmonary resuscitation for seven minutes, after which return of spontaneous circulation occurred. Another cardiologist was consulted under the presumed diagnosis of cardiac tamponade. He repeated an echocardiogram, which showed increasing PE and a distended inferior vena cava and confirmed a diagnosis of cardiac tamponade. A pericardiocentesis was performed with drainage of 600 ml brown-red purulent fluid with a foul-smelling odour, and a drain was left in situ. The patient improved haemodynamically, and the noradrenaline infusion could be tapered.

The next day both blood and pericardial fluid cultures became positive for haemophilus influenzae; the urinary culture was negative. The antibiotic regimen was changed to ceftriaxone for four weeks. An immunocompromised status was excluded by additional testing.

DISCUSSION

This patient presented with chest pain and shortness of breath that, within hours, evolved into septic shock with a cardiac tamponade. A final diagnosis of haemophilus influenzae pericarditis with septic shock and cardiac tamponade was made. We found only seven case reports of haemophilus influenzae pericarditis with cardiac tamponade in adults. Bacterial pericarditis is usually associated with thoracic surgery, pneumonia, dialysis, chemotherapy, acquired immunodeficiency syndrome and rarely, complement deficiency, all conditions that did not apply to this patient[1,3,4].

In the early stages of clinical presentation, there was a disagreement between the intensivist and the cardiologist. After the first TTE was done there were, in hindsight, enough clinical signs to make a diagnosis of cardiac tamponade. TTE showed a PE of 23.2 mm *(Fig. 2)*, a distended inferior vena cava and an increased respiratory variation of mitral inflow velocity *(Fig. 3)*. Following the guidelines for TTE assessment of pericardial effusion, an effusion more than 20 mm is considered large and a >25% fall in mitral inflow velocity in the first beat after inspiration is suggestive for a cardiac tamponade[5]. Pericardiocentesis should have been performed directly following the TTE. Even a small pericardial effusion should raise suspicion for cardiac tamponade, particularly in the presence of additional signs such as a dilated non-collapsing inferior vena cava, increased respiratory variation in mitral inflow, pulsus paradoxus (sensitivity 75–98%) or electrical alternans (rare), or a decrease in pulse pressure typically falling below 30 mmHg. If the diagnosis is not made immediately, it is essential to assess the effects of fluid therapy and vasopressors on systemic BP, cardiac output and central venous pressure. Passive leg raising is a manoeuvre to assess fluid

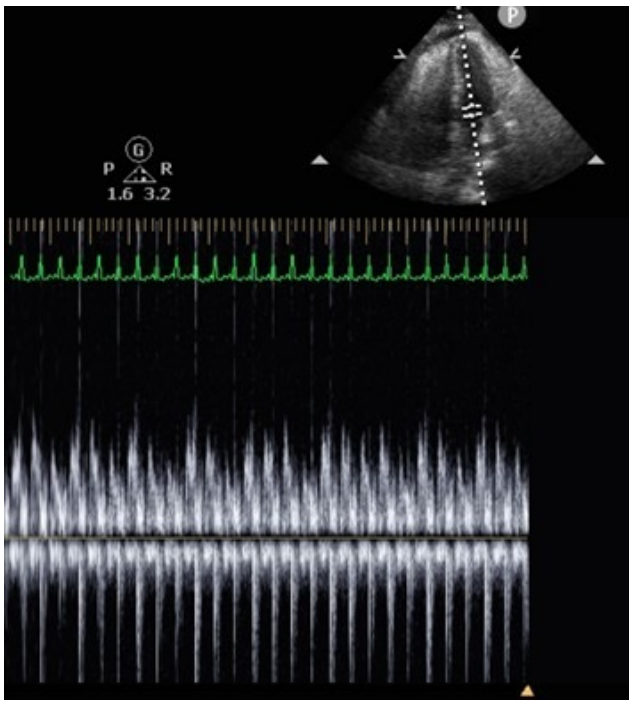


Figure 3. Pulse wave Doppler measuring respiratory cycle changes in the mitral valve inflow velocity on an apical four-chamber view. The gate is placed at the tips of the valve leaflets.

Review the process	
Did I take a thorough and complete medical history?	If not, complete your history taking.
Did I perform a thorough and complete physical examination?	If not, complete your physical examination.
Did I order additional tests to test my first hypothesis?	If not, order the needed tests.
Is there any contradicting information?	If yes, review your hypothesis and think of alternative hypothesis and how to confirm or rule out these alternate hypotheses.
Time out	
Could I be biased?	For example, did I think about diagnoses outside my own medical discipline or an atypical presentation of a disease? If yes, note the symptoms of the patients and physical signs objectively and take them into account. If needed, repeat the previous steps.
Do I feel emotions towards this patient?	If yes, note the symptoms of the patients and physical signs objectively and take them into account. If needed, repeat the previous steps.
Did I take my own tiredness into account?	If you are tired, check the previous steps and think about consulting a colleague about the case.
Are there diagnoses that can cause the complaints which I don't want to miss?	If yes, rule these diagnoses out before proceeding.
Is it possible that there are more diagnoses at the same time?	If yes, try to confirm or rule out these diagnoses.
Do I have to make the diagnosis now?	If not, treat the symptoms and continue with the diagnostic process later. If the patient does not respond to the chosen therapy or deteriorates, continue the diagnostic process.

Table 1. Tips for preventing diagnostic mistakes.

responsiveness and POCUS should be employed to evaluate central venous pressure and cardiac output, and progression of the pericardial effusion should be monitored, especially when haemodynamic status fails to improve.

In clinical settings where decisions need to be made in a limited amount of time and with limited resources, intuitive thinking and biases are unavoidable[6]. Biases are 'predictable deviations from rationality' and are more likely when we use intuitive instead of analytical thinking. An important lesson of this case is to be aware of our biases and know how to mitigate them. There are a few high-risk situations during which biases occur more frequently such as fatigue, cognitive overload or a patient handed over from a previous shift[7]. Multiple biases played a role in this case. The first cardiologist anchored on the signs of sepsis (anchoring and confirmation bias) and ignored the signs on TTE that pointed to another cause of haemodynamic instability (premature closure)[8]. Omission bias[7], the tendency towards inaction and rooted in the principle of non-maleficence, may have caused a delay in performing an intervention due to possible complications of a pericardiocentesis. Overconfidence bias[8], a tendency to place too much faith in our own opinions, often without having gathered sufficient information, could also have contributed to the misinterpretation of the TTE and the disagreement in the working diagnosis. Authority bias, the uncritical acceptance of the views of authority, was a contributing factor. The IC physician accepted the dismissal of the working diagnosis of a cardiac tamponade by the cardiologist without rediscussing the diagnosis.

Luckily, there are strategies to mitigate the risks for biases[9]. We formulated questions to reflect on the diagnosis and prevent biases *(Table 1)*. Importantly, all clinicians need to learn about cognitive hazards in clinical reasoning. To that purpose, we need to use structural data acquisition through systematic evaluation of the patient. We need to deliberately disengage from intuitive judgements but engage in analytical processes to verify initial impressions.

With unstable patients, it is important to perform systematic assessment at regular times and when there is a change in clinical status. Cardiac and lung POCUS have been proved to be useful in undifferentiated shock patients[10]. It is imperative that POCUS training is integrated into clinical practice and that ultrasound machines are readily available across hospital wards, emergency departments and IC units. If uncertainty persists about the diagnosis, worst-case scenarios must be ruled out, and it is prudent to discuss the case in a multidisciplinary consultation with all healthcare professionals involved. If there is a disagreement with a colleague from another medical specialty, it is advised that all specialists re-evaluate the patient together to clarify any misunderstanding.

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