Case Report

A case of purulent pneumococcal pericarditis☆

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A R T I C L E   I N F O

Article history:
Received 1 February 2019
Accepted 10 February 2019

Keywords:
Ultrasound, diagnostic
Emergency department
Pericardiocentesis
Pericarditis
Streptococcus pneumoniae

A B S T R A C T

Background: Purulent bacterial pericarditis is a rare and potentially fatal disease. The course may be fulminant, and the presentation may pose a diagnostic challenge.

Case report: An otherwise healthy 75-year-old male was brought to the emergency department in a state of general deterioration, confusion, and shock. Bedside ultrasound showed a significant pericardial effusion. His condition quickly deteriorated and the resuscitation included emergent bedside pericardiocentesis. The drainage was purulent and later cultures grew out Streptococcus pneumoniae.

Why should an emergency physician be aware of this?: Purulent pericarditis is extremely rare but should be considered in the patient with a fulminant infectious process (particularly pneumonia) and signs of pericardial effusion. Treatment should include appropriate antibiotics and early drainage.

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1. Introduction

Purulent pericarditis is a rare disease, particularly in the western world and especially in the era of pneumococcal vaccination. Among all cases of pericarditis in the United States, ~1% are bacterial. Pneumococcal pericarditis is very rare and can be fulminant and fatal. It is usually a complication of another pneumococcal disease such as pneumonia. There are only a few cases of purulent pneumococcal pericarditis published in the literature and it is known to have a high mortality rate. We describe a case of an otherwise healthy adult who presented to the emergency department (ED) with general deterioration, confusion, and shock, who was diagnosed with pneumonia and purulent pericarditis causing overwhelming sepsis.

2. Case report

A 75-year-old previously healthy male was brought to the ED by emergency medical services after he was found lying confused on the floor of his apartment. On arrival to the ED, the patient was moaning in response to questions and his blood glucose level was 54 mg/dL. His blood pressure was 94/60 mm Hg, heart rate 120 beats/min, and pulse oximetry of 95% on room air. He was afebrile. His physical examination revealed diminished heart sounds and rales in the right lower lobe of the lungs. The abdomen was soft and not tender. His pupils were equal and reactive to light and he had rightward gaze deviation and weakness of the left hand. He immediately received 20 mL of dextrose 50%. Point-of-care ultrasound (POCUS) according to the rapid ultrasound for shock and hypotension (RUSH) protocol was performed which revealed a medium to large pericardial effusion without sonographic signs of cardiac tamponade, i.e. collapse of the right side of the heart during diastole or plethoric inferior vena cava (IVC). The IVC actually appeared collapsed which suggested hypovolemic shock. Aggressive fluid resuscitation was started with the blood pressure increasing to 119/85 mm Hg and heart rate decreasing to 107 beats/min. His first venous blood gas demonstrated a compensated metabolic acidosis with pH of 7.43, PCO2 26 mm Hg and lactate of 7.8 mmol/L. There was concern about the possibility of intracranial hemorrhage, so he was then taken to computed tomography (CT) scan of the brain which was negative for bleeding. Due to a high lactate level, a CT scan of the chest and abdomen was performed which revealed bilateral pulmonary infiltrates and the known pericardial effusion (Fig. 1). Intravenous ceftriaxone was started, and an intensive care unit consultation was obtained. His temperature increased to 38 °C. His laboratory results came back with a white blood count of 3400 cells/μL, absolute neutrophil count 2780 cells/μL. He had acute kidney injury with a creatinine level of 3.76 mg/dL (normal ~1.1 mg/dL), elevated C reactive protein, and elevated cardiac troponin I. The patient’s level of consciousness started deteriorating and pulse oximetry dropped to 92% on 4 L per minute of oxygen via nasal cannula, but he remained hemodynamically stable. Using rapid sequence intubation with etomidate and rocuronium, an endotracheal tube was placed without complications. Chest X-ray performed after the intubation showed a large cardiac shadow and large right lung infiltrate (Fig. 2). Shortly afterwards the physician was called to the room because the end tidal CO2 (ETCO2) decreased rapidly from 24 mm Hg to 12 mm Hg. Repeat

☆ The authors have no commercial associations or sources of support that might pose a conflict of interest. We acknowledge that there are no sources of funding for this paper.
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POCUS was performed which demonstrated severely impaired global contraction without classic sonographic signs of cardiac tamponade. As an epinephrine drip was prepared, the ETCO2 dropped below 10 mm Hg and heart rate dropped below 30 beats/min.

Cardiopulmonary resuscitation (CPR) was initiated and emergent bedside pericardiocentesis was performed. An initial attempt was made at dynamic ultrasound guided drainage using the needle from a central line kit positioned sub-xiphoid and directed at 45 degrees towards the left shoulder. This was unsuccessful but a second attempt at the same approach without using ultrasound resulted in the removal of 170 mL of purulent drainage. POCUS was repeated during CPR showing minimal residual pericardial effusion. Despite continuous efforts at resuscitation the patient expired. Fluid from the pericardial effusion and blood cultures were sent to the microbiology laboratory with both positive for *Streptococcus pneumoniae*, without antibiotic resistance.

### 3. Discussion

Bacterial infection is an infrequent cause of acute pericarditis in the United States, accounting for <1% of all cases [1-3]. Risk factors include alcoholism, chest trauma or cardio-thoracic surgery, and immunodeficiency. It is even more rare in an otherwise healthy immunocompetent patient, especially in the era of routine vaccinations [4]. It is usually a fulminant and fatal disease with a very high mortality rate. Pneumococcus, other streptococci, and staphylococcus are the most common microbes infecting the pericardium and pericardial space [5,6]. *Streptococcus pneumoniae* is a major human pathogen causing pneumonia, meningitis, and other invasive diseases. Pneumococcal pericarditis is a rare, potentially fatal, complication of pneumococcal pneumonia, especially when untreated [7-10]. It may occur by direct spread of an intrathoracic, myocardial, or subdiaphragmatic focus, a perforating injury to the chest wall, or hematogenous dissemination [11-13].

There are very few case reports describing pneumococcal pericarditis. Some of them were in third world countries or immunodeficient patients [4,9,10,14] with the minority in otherwise healthy immunocompetent patients [1,2,15]. Many cases ended in patient death [11-13]. In those where the patient survived there was a rapid...
diagnosis and early aggressive antibiotic and surgical treatment [1-4,9,16]. The type of surgical treatment includes pericardiocentesis, pericardial drainage, or a wide pericardial window. The patient presented here also had neurologic signs. A lumbar puncture was not performed due to the severe and fulminant course of the disease and since the presence of meningitis would not have changed the treatment.

Austrian syndrome also known as Osler’s triad is the very rare combination of pneumonia, meningitis, and endocarditis caused by hematogenous dissemination of Streptococcus pneumoniae [17]. There have also been cases where other organ systems have been involved [18]. Could this have been such a case with additional involvement of the pericardium?

4. Why should an emergency physician be aware of this?

Purulent pericarditis is potentially a fulminant and fatal disease. In the era of POCUS, patients complaining of chest pain or shortness of breath associated with clinical, radiological, or ECG signs suggestive of pericardial effusion should undergo a quick bedside ultrasound exam [19]. One should still consider performing the RUSH exam in patients with hypotension from known sepsis as not only can it help guide fluid management but may also lead to the specific diagnosis. Infectious causes of pericardial effusion should be in the differential diagnosis of patients with significant effusions and fever. If a significant effusion is found one should treat with appropriate antibiotics and consider early drainage even in the absence of classic signs of tamponade.

References