CASE IN POINT

A Forgotten Cause of Cardiac Tamponade

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Although purulent pericarditis is rare, it is essential to recognize its clinical features due to the high mortality rate in patients with a missed diagnosis.

Purulent pericarditis is an infection within the pericardial space rarely seen in the modern antibiotic era. Most cases are secondary to another infectious process of bacterial, viral, fungal, or parasitic origin.[1,2] Predisposing factors include malignancy, chronic kidney disease, immunosuppression, diabetes mellitus, and alcohol misuse disorder.[1] Although purulent pericarditis has been described extensively in the literature, it is a challenging diagnosis if it is not initially considered within the differential diagnosis repertoire.[1-4] Most authors agree that this may be because it has become an infrequent diagnosis.[1,2] In addition, purulent pericarditis may have an atypical presentation when compared with a classic case of pericarditis.[2,3] The authors believe that this forgotten entity will be revisited through this case.

CASE PRESENTATION

A 66-year-old-man was transferred to Veterans Affairs Caribbean Healthcare System (VACHS) from a community hospital with a diagnosis of community-acquired pneumonia (CAP) and bilateral pleural effusions. Four days prior to arrival at the community hospital, the patient had developed diffuse, watery diarrhea, which resolved in 3 days. After resolution of diarrhea, he began experiencing shortness of breath on exertion that progressed to onset at rest. The patient reported no fever, chills, nausea, vomiting, cough, or contact with others who were not healthy. He had a history of alcohol misuse without liver cirrhosis and reported no chronic diseases or use of medications. The patient had no history of tuberculosis exposure or pneumococcal vaccination, and had a negative interferon gamma release assay.

On admission to the community hospital, the patient was treated for CAP with ceftriaxone and azithromycin. On hospital day 3, the patient developed hypoxemia and an altered mental status. He was started on supplemental oxygen and transferred to the intensive care unit (ICU). Antibiotic therapy consequently was changed to levofloxacin and meropenem. However, no clinical improvement was noted on the following days.

On hospital day 7, the patient developed acute respiratory failure that required mechanical ventilation while being transferred to VACHS via air ambulance. His vital signs on arrival were the following: temperature, 97° F; heart rate, 86 beats/min; blood pressure, 103/61 mm Hg; respiratory rate, 14 breaths/min and SaO_2 of 97%, measured while he breathed supplemental oxygen at an FiO_2 of 0.4. Laboratory results revealed a white blood cell count (WBC) of 11.2 × 103/µL, with 92% neutrophils, 4% bands, 1% lymphocytes; lactic acid, 1.3 mmol/L; and high sensitivity C-reactive protein, 43.1 mg/L. A chest radiograph demonstrated bibasilar pulmonary opacities, bilateral pleural effusions, and cardiomegaly (Figure 1).

Hours after arrival, the patient developed sinus tachycardia and hypotension. A bedside 2D echocardiogram demonstrated a large pericardial effusion with diastolic collapse of the right atrium (Figure 2). Aggressive fluid resuscitation with normal saline and inotropic support with dopamine were started as an immediate percutaneous pericardiocentesis was performed with drainage

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FIGURE 1 Chest Radiograph

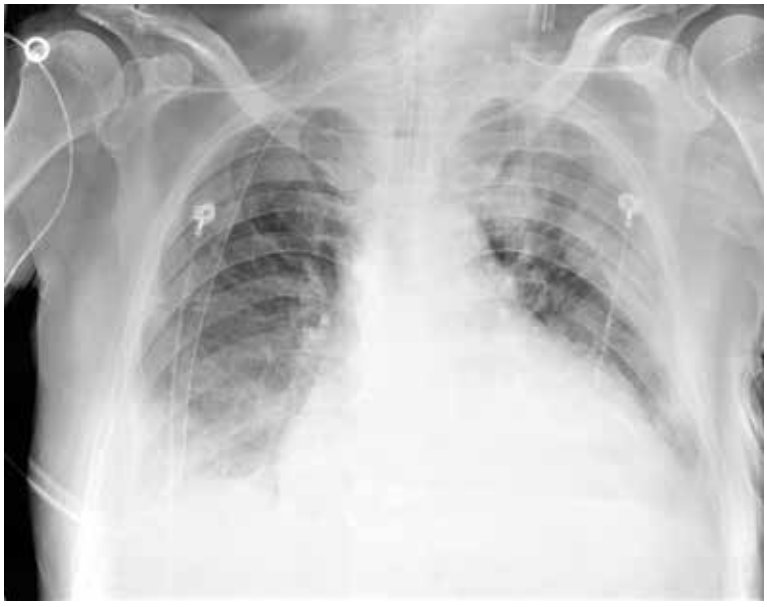
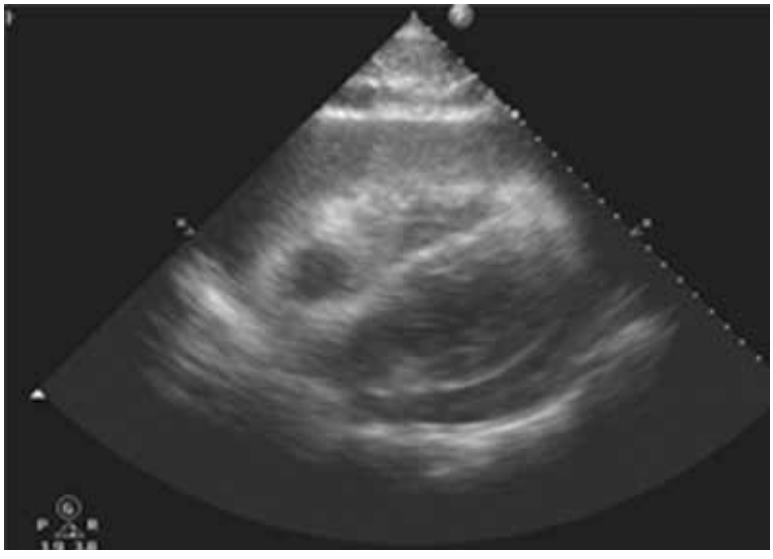


Image shows a globular heart hanging down the mediastinum, silhouette known as the water bottle heart.

FIGURE 2 Echocardiogram Subcostal View Showing Large Circumferential Pericardial Effusion



of 400 mL of frank purulent fluid (Figure 3). After percutaneous pericardiocentesis was completed, a temporary catheter was placed into the pericardial space using the Seldinger technique in order to aspirate fluid as needed

The patient's clinical condition improved following drainage of pericardial fluid, with no further need for inotropic support. Antibiotic therapy was changed to vancomycin and meropenem. Initial microbiologic samples from pericardial fluid demonstrated Gram-positive diplococci, suggestive of *Streptococcus pneumoniae* (*S pneumoniae*) (Figure 4). Other diagnostic pericardial fluid test results included: WBC count 25,330 cmm, with 99% neutrophils and 1% lymphocytes; total protein, 3.8 mg/dL; glucose, < 2.0 mg/dL, LDH, > 2,500 U/L, potassium hydroxide preparation. The tests found no fungus, and the acid fast *bacilli* smear revealed no *Bacillus*. However, the pericardial fluid culture failed to demonstrate growth of any organism. Blood cultures also were negative.

The patient underwent anterior thoracotomy with partial pericardiectomy, and a pericardial tube was left in place connected to drainage. During the procedure, an abundant amount of fibrinous tissue was evacuated from the pericardial space (Figure 5). The patient's status improved the following day, and inotropic support once again was stopped. Bilateral pleural tubes were placed for evacuation of loculated effusions. Laboratory results from left-sided fluid were suggestive of complicated parapneumonic effusion with a WBC count of 6,683 cmm and glucose, 12 mg/dL. Right-sided pleural fluid was consistent with exudate with a WBC count of 2,006 cmm; pH 7.49; LDH, 1450 U/L; and glucose, 66 mg/dL. Gram stain and cultures of pleural fluid were negative.

The patient was extubated, pericardial and pleural tubes were removed, and he was transferred to the internal medicine ward 24 days after admission to the ICU. He received in-patient physical rehabilitation while completing a 6-week course of IV antibiotics (vancomycin and meropenem). After completion of therapy, the patient received the pneumococcal polysaccharide vaccination, and an echocardiography was repeated. No significant re-accumulation of pericardial effusion or constrictive pattern was evidenced. The patient was discharged to his out-of-state home, and follow-up was consequently lost.

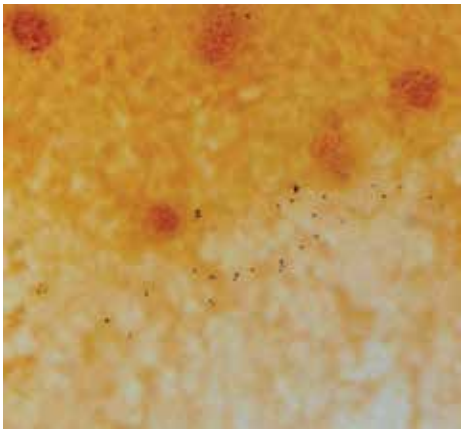
DISCUSSION

Purulent pericarditis is an infection localized within the pericardial space. Most cases are secondary to an infectious process

elsewhere, which could be of bacterial, viral, fungal, or parasitic etiology.[1] Five mechanisms could lead the infecting organism to infect the pericardial space; contiguous spread from intrathoracic site, hematogenous spread, extension from myocardial site, perforating injury or surgery, and extension from a subdiaphragmatic site.[1] Predisposing factors for the development of this condition include malignancy, chronic kidney disease, immunosuppression, diabetes mellitus, and alcohol misuse. Pericarditis is an infection localized within the pericardial space.

FIGURE 3 Purulent Fluid Drained From Pericardial Cavity

FIGURE 4 Gram-Positive Diplococci on Gram Stain



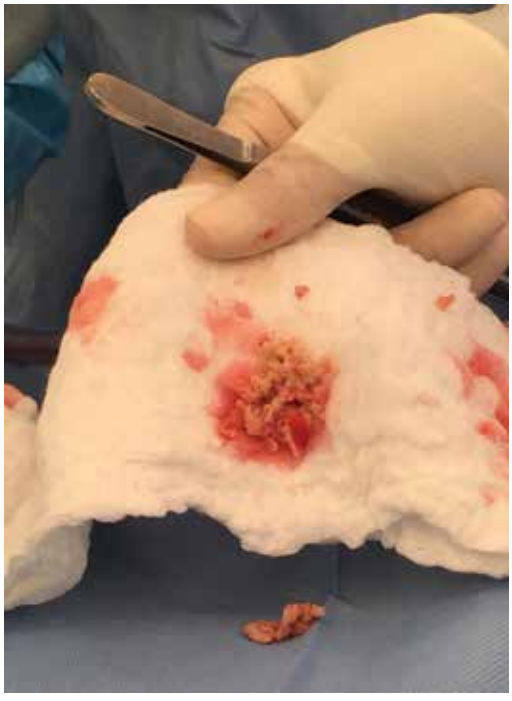
Purulent pericarditis has become a rare entity in the antibiotic era.[2] Prior to the development of antibiotics, most cases were secondary to *S pneumoniae*.[1,2,5,6] As per Cilloniz and colleagues, about 40% to 50% of all cases of purulent pericarditis are caused by Gram-positive bacteria, mostly *S pneumoniae*.[5] In this case study, bacterial culture did not reveal growth of an organism—most likely because the patient had received antibiotics elsewhere. However, Gram-positive cocci were seen within the initial pericardial aspirate. This organism was suspected to have spread contiguously from a pulmonary focus, which also led to pleural effusions.

Since the patient in this case study had no history of thoracic surgery, malignancy, or other immunosuppression, the patient's history of alcohol misuse was the only predisposing factor for development of purulent pericarditis. Contrary to the common presentation of pericarditis, purulent pericarditis may not have the common clinical findings, such as chest pain, pericardial friction rub, and distended neck veins.[2,3] Furthermore, according to Parikh and colleagues, about 35% of affected patients may have a normal electrocardiogram.[2] Hence, the diagnosis of purulent pericarditis often is missed because the classic signs of pericarditis are often absent, and other nonspecific symptoms are attributed to initial underlying infection.[7]

A high index of suspicion is needed to diagnose purulent pericarditis. Once a diagnosis is made, initial treatment should consist of prompt drainage of pericardial fluid combined with systemic antibiotic therapy. Vancomycin and a third-generation cephalosporin may be started empirically until results of pericardial fluid cultures become available.[3] Drainage can be achieved by pericardiocentesis, pericardiotomy, or pericardiectomy (partial or total).[1] In cases of hemodynamic instability due to cardiac tamponade, sonographically guided pericardiocentesis should be undertaken and an indwelling pericardial catheter left in place.[1] Although this is the simplest and fastest method of evacuation, it may not be effective when dealing with thick, fibrinous fluid. In such cases, intrapericardial fibrinolysis may be considered. This approach may be undertaken early in the process, after drainage insertion, or as salvage therapy, when there has been incomplete evacuation of purulent material or open surgical drainage is not available.

Streptokinase, urokinase, and tissue plasminogen activator have been used for intrapericadial fibrinolysis.[1] However, there is no definite data on dosage or frequency at which these medications should be administered. No matter the therapeutic approach, effective drainage of the pericardial fluid is crucial to avoid the development of pericardial constriction. Constrictive pericarditis occurs

FIGURE 5 Fibrinous Tissue Evacuated During Partial Pericardiectomy



when fibrosis and adhesions create a dense pericardium that encases the heart. This causes impaired ventricular filling that can lead eventually to heart failure.[4] Pericardiectomy is the definitive treatment for constrictive pericarditis.

CONCLUSION

Although purulent pericarditis has become a rare diagnosis since the development of antibiotics, knowledge of how to identify it is essential since mortality reaches 100% if the diagnosis is missed.[4] Even when the condition is promptly diagnosed and treated, mortality is 40%, mainly due to cardiac tamponade, septic shock, or constriction.[1] The case presented here illustrates the clinical features associated with this condition. Knowing these features can translate in a successful patient outcome.

Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

Disclaimer

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