Patient with Small Cell Lung Carcinoma and Suspected Right Upper Lobe Abscess Presenting with a Purulent Pericardial Effusion

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Conflict of interest: None declared

Patient: Male, 61
Final Diagnosis: Streptococcus pneumoniae pericarditis
Symptoms: —
Medication: —
Clinical Procedure: Pericardiocentesis
Specialty: Critical Care Medicine

Objective: Rare disease
Background: Cardiac tamponade caused by pericardial effusion has a high mortality rate; thus, it is important to diagnose and treat this condition immediately. Specifically, bacterial pericarditis, although now very rare, is often fatal because of its fulminant process.

Case Report: We present a case of a 61-year-old man with metastatic small cell lung cancer undergoing chemotherapy who presented with fatigue, poor appetite, and altered mental status. He was found to have a large-volume pericardial effusion with tamponade physiology. He underwent emergent pericardiocentesis. The pericardial effusion was nonmalignant, with cultures growing Streptococcus pneumoniae. It was only after his emergent pericardiocentesis that previous imaging from one month prior was able to be reviewed, which showed possible right upper lobe abscess.

Conclusions: Most pericardial effusions in cancer patients are related to their malignancy, either due to direct metastasis or secondary physiologic effects. This case is a unique example of a lung cancer patient presenting with a pneumococcal pericardial effusion, which in itself is a rare phenomenon. This case report demonstrates the importance of considering early antibiotic therapy in patients presenting with pericardial effusion, especially given the high mortality rates of infectious pericardial effusions.

MeSH Keywords: Cardiac Tamponade • Pericardial Effusion • Pneumococcal Infections • Small Cell Lung Carcinoma

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Background

There are many causes of pericardial effusion: infectious, autoimmune, malignant, metabolic, drugs/toxins, traumatic, and idiopathic [1]. The most common etiology of large-volume pericardial effusions varies depending on the population studied [1]. Determining the etiology can be challenging, but is critical for subsequent management, treatment, and prognosis.

Specifically, bacterial pericarditis has a high mortality rate of 40% despite treatment, usually due to cardiac tamponade as a complication [2–4]. Mechanisms of infection include direct spread from a pneumonia, empyema, or other mediastinal infection and hematogenous spread [5]. However, bacterial pericarditis is often difficult to recognize, especially since it is now rare in developed countries: the incidence is reported to be less than 1% [6,7]. Streptococcus and Staphylococcus species are the most common bacteria to cause pericarditis [3,4]. Streptococcus pneumoniae was the most prevalent microbe prior to the advent of the pneumococcal conjugate vaccine and antibiotics [8]. Klaksman et al. reviewed autopsy reports of 145 patients with purulent pericarditis from 1889 to 1943, of which 51% were secondary to S. pneumoniae [9]. In contrast, there were only 15 reported cases of pneumococcal pericarditis from 1980 to 1994 [10].

We present a unique case of a patient with small cell lung carcinoma who presented with an infectious, rather than malignant, pericardial effusion secondary to S. pneumoniae.

Case Report

A 61-year-old African American man with stage IV small cell carcinoma of the lung, status post whole brain radiation and currently undergoing chemotherapy, was sent to the emergency department (ED) directly from clinic after he was found to be hypotensive with a blood pressure of 66/29 mm Hg. The patient had altered mental status, decreased appetite, vomiting, fatigue, and generalized pain for the past two days. On the day of admission, he was on cycle 2, day 3 of chemotherapy (IV cisplatin and etoposide on day 1 followed by two days of oral etoposide).

In the ED he had a temperature of 36.1°C, heart rate of 140–160 beats/minute, respiratory rate of 18 breaths/minute, and a blood pressure of 67/47 mm Hg. He received a 2-L normal saline bolus, which briefly raised his systolic blood pressure to 85, but it then returned to the 60s. On exam, he was in acute distress, tachypneic, and alert but disoriented. Jugular venous distension was present up to the jaw, without pulsus paradoxus or Kussmaul sign. He had muffled heart sounds and was tachycardic with an irregularly irregular rhythm. He did not have peripheral edema.

His electrocardiogram showed atrial fibrillation with a rapid ventricular response (AFib-RVR), and decreased QRS voltages with no ST segment or T wave changes (Figure 1). Electrical alternans was absent. Portable chest radiograph showed a cardiac silhouette within normal limits for a portable exam and no widened mediastinum (Figure 2). He had an elevated white blood cell count of 12,000/µL with 93% neutrophil predominance and a venous lactate of 11.2 mmol/L. Serial troponins were negative.

Bedside echocardiogram demonstrated a large pericardial effusion, the heart swinging in the pericardial fluid, and diastolic collapse of the right ventricle (Figure 3). The inferior vena cava was dilated and noncollapsing with the respiratory cycle despite the severe volume depletion (Figure 3).

The patient underwent emergent percutaneous pericardio-centesis under fluoroscopic guidance within 15 minutes in the interventional cardiology laboratory; 450 mL of milky, purulent fluid was drained. Within less than 2 minutes and after draining less than 150 mL of the fluid, the blood pressure increased to 143/73 mm Hg despite persistence of the Afib-RVR at a rate of 150s/min. This improvement confirmed that the hemodynamic compromise was tamponade related rather than RVR related.

Because the patient was in septic shock and immunocompromised, broad spectrum IV antibiotics were begun: vancomycin, piperacillin-tazobactam, and micafungin. Later that evening, the pericardial fluid Gram stain showed 4+ gram-positive cocci (Figure 4). The fluid had a white cell count of 228,850/µL, 99% neutrophil predominance, glucose <20, total protein 4.6, and lactate dehydrogenase >20,000. The next morning, pericardial fluid as well as peripheral blood cultures grew pansensitive S. pneumoniae. His antibiotic coverage was narrowed to ceftriaxone 2 g IV every 12 hours. Pericardial fluid cytology did not reveal malignant cells, only markers of acute infection.

Discussion

This case presents a rare combination of a patient with primary lung cancer who presents with a nonmalignant, infectious pericardial effusion caused by S. pneumoniae. In fact, malignant effusion was initially highest on our differential diagnoses. Malignancy is often the most common cause of pericardial effusion and subsequent tamponade in patients presenting to a tertiary-care academic center [11]. Klatt and Heitz found that lung cancer was the most common primary site of cardiac metastases and that pericardial effusions were present with 33% of epicardial metastases [12]. The prognosis of malignant pericardial effusions is poor and is worsened if the fluid cytology is abnormal [11,13,14]. Thus, it is a critical etiology.
to rule in or out. In fact, sometimes cytology is the only clue for diagnosis; for example, there was a recent case report of a patient without any solid masses or lymphadenopathy who was diagnosed with small cell carcinoma solely from pleural effusion cytology [15].

However, many studies have shown that pathologic evaluation of pericardial fluid, specifically, is not always diagnostic. Also, having a primary malignancy can in fact confound the picture. El Haddad et al. studied 1645 cancer patients at the MD Anderson Cancer Center with pericardial effusions, 212 of whom underwent percutaneous pericardiocentesis [16]. Of those, 41% were shown to have direct cancer involvement of the pericardium, while 57% had effusions of undetermined etiology. Similarly, Porte et al. studied 112 patients with a history of cancer who underwent pericardiocentesis, 38% of whom had malignant effusions, while 61% had nonmalignant effusions of undetermined etiology [17]. These nonmalignant effusions are hypothesized to be caused by indirect effects of malignancy and subsequent chemotherapy and radiation, such as lymphatic obstruction, pericardial fibrosis, fluid accumulation, and inflammation [16]. Thus, the majority of pericardial effusions in patients with a malignancy still appear to be secondary to the malignancy. In our patient’s case, however, the presence of a primary lung malignancy proved to be a complete red herring, because the pericardial effusion did reveal any malignant cells and instead grew *Pneumococcus*.

Infectious effusion was second on our differential diagnosis, especially because the patient was immunocompromised.

Figure 1. Electrocardiogram (ECG) on admission (A) with cardiac tamponade showing atrial fibrillation with a rapid ventricular response with lower voltages, and ECG after pericardiocentesis (B) showing sinus rhythm with low voltages.
Despite being afebrile, his presentation was concerning for septic shock, given his elevated white blood cell count with left shift and lactate of 11.2. Reviewing a computed tomographic angiogram of the chest from one month prior to admission demonstrated progression of the right perihilar and mediastinal mass, and adenopathy causing superior vena cava occlusion and obstructive collapse of the right upper lobe. Within this collapsed lobe was an area of low density concerning for abscess, which may have seeded the pericardium (Figure 5).

As discussed, bacterial pericardial effusions, specifically due to *S. pneumoniae*, are very rare in developed countries, with...
only isolated case reports published. However, this case is especially unique because there are very few reports of primary lung cancer patients developing a purely bacterial pericardial effusion. For example, of the 212 cancer patients with pleural effusions from the El Haddad et al. study, only 4 patients (2%) had infectious effusions: 2 bacterial, 1 viral, and 1 fungal [16]. One other publication found after a thorough literature search was a case report of a patient with squamous cell lung cancer and pericardial effusion with both malignant squamous cells and S. pneumoniae in the pericardial fluid [18].

In a time when antibiotic stewardship is so important (with increasing microbial resistance due to liberal and often knee-jerk administration of antibiotics), this case report is an example of a specific clinical situation where early empiric antibiotic therapy changed the patient’s outcome for the better, even when the objective data show that infectious pericarditis is extremely rare in patients with malignancies.

Conclusions

The etiology of pericardial effusions in patients with malignancy can be confounded by many factors. However, the majority of these effusions are still related to direct and indirect effects of the malignancy and treatment therapies. However, although infectious pericarditis is especially rare in patients with malignancies, physicians should always consider early empiric therapies including antibiotics in similar patient populations while awaiting pericardial fluid analysis.

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