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Purulent *Streptococcus pneumoniae* pericarditis presenting as cardiac tamponade in an immunocompromised patient: case report

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ABSTRACT

Background: Purulent bacterial pericarditis is a rare but rapidly progressive cause of acute pericarditis in the antibiotic and pneumococcal vaccination era. Although uncommon, delayed recognition can rapidly lead to cardiac tamponade, septic shock, and death.

Case Presentation: We report a 64-year-old man with pulmonary and hepatic sarcoidosis (not receiving corticosteroids, biologics, or other immunosuppressive therapy) and well-controlled human immunodeficiency virus (HIV) infection, on Biktarvy with an undetectable viral load and a CD4 count of 217 cells/µl, who presented with pleuritic chest pain and progressive dyspnea. Initial electrocardiogram revealed diffuse ST-segment elevations, while transthoracic echocardiography demonstrated a moderate pericardial effusion without tamponade physiology. Computed tomography demonstrated multifocal pneumonia, prompting empiric antimicrobial therapy. Despite treatment, the patient rapidly developed hypotension and acute hypoxic respiratory failure. Repeat echocardiography demonstrated evolving cardiac tamponade requiring emergent pericardiocentesis. Blood cultures drawn at presentation prior to antibiotic administration and pericardial fluid both grew *Streptococcus pneumoniae*, confirming invasive pneumococcal purulent pericarditis. Although recurrent pericardial effusion developed following drain removal, the effusion remained hemodynamically stable, and the concurrent clinical deterioration was attributed to enlarging pleural effusions. The patient recovered with pathogen-directed antimicrobial therapy without requiring repeat pericardial intervention. At one-year follow-up, echocardiography demonstrated complete resolution of the pericardial effusion without evidence of constrictive physiology.

Conclusion: This case underscores the diagnostic challenge of purulent pneumococcal pericarditis in immunocompromised patients presenting with concurrent pneumonia and initially absent tamponade physiology. Serial echocardiography, prompt pericardial drainage, microbiologic confirmation, and pathogen-directed antimicrobial therapy are critical to improving outcomes in this rare but life-threatening disease.

Keywords: Purulent pericarditis, *Streptococcus pneumoniae*, Cardiac tamponade, Human immunodeficiency virus, Sarcoidosis, case report.

Type of Article: CASE REPORT **Specialty:** Cardiology, Infectious Diseases

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Introduction

Bacterial pericarditis is a rapidly progressive and life-threatening infection associated with mortality rates of 20%-30% despite prompt drainage and appropriate antibiotic therapy [1,2]. Historically, gram-positive cocci, particularly *Staphylococcus aureus* and *Streptococcus pneumoniae*, were the predominant pathogens, typically spreading hematogenously or by contiguous extension from pulmonary or mediastinal infections [1,3]. However, widespread antibiotic use and routine pneumococcal vaccination have rendered purulent bacterial pericarditis an exceedingly rare clinical entity in the modern era [3,4]. In North America and Western Europe, most cases of acute pericarditis are idiopathic or presumed viral, with bacterial etiologies accounting for only a small minority [5].

When bacterial pericarditis does occur, it is most commonly associated with immunosuppression, chronic illness, prior cardiothoracic surgery, or chest trauma [1]. Patients with human immunodeficiency virus (HIV) remain at increased risk for pericardial disease despite combination antiretroviral therapy [6,7], while sarcoidosis may also involve the pericardium, with pericardial effusion and cardiac tamponade representing well-described manifestations [8]. Consequently, distinguishing infectious pericarditis from more common inflammatory or idiopathic etiologies can be particularly challenging in immunocompromised patients, especially when cardiac

tamponade is absent on initial presentation. Despite its rarity, purulent pericarditis requires a high index of suspicion because delayed diagnosis may result in fulminant hemodynamic compromise and death; historically, up to half of cases were diagnosed only at autopsy [2,3]. Furthermore, contemporary literature describing culture-proven pneumococcal purulent pericarditis with recurrent pericardial effusion following successful pericardiocentesis remains limited.

We present a rare case of culture-proven *S. pneumoniae* purulent pericarditis complicated by evolving cardiac tamponade in a patient with concurrent HIV infection and pulmonary/hepatic sarcoidosis. This report highlights the diagnostic challenges posed by overlapping infectious and inflammatory causes of pericardial disease, underscores the importance of serial echocardiographic surveillance and microbiologic confirmation, and demonstrates the individualized management of recurrent pericardial effusion.

Case Report

A 64-year-old man with pulmonary and hepatic sarcoidosis and HIV infection presented with progressive exertional dyspnea and pleuritic chest pain. His HIV was treated with Biktarvy (bictegravir/emtricitabine/tenofovir alafenamide), with an undetectable viral load and a CD4 count of 217 cells/µl prior to admission. He was not receiving corticosteroids, biologic agents, or any other immunosuppressive therapy for sarcoidosis. He had previously received the PCV20 vaccine (20-valent pneumococcal conjugate vaccine).

Initial vital signs were within normal limits. Physical examination demonstrated mild bibasilar crackles. There was no jugular venous distension, heart sounds were normal, and there was no lower extremity edema or focal neurologic deficits. Initial laboratory investigations, including high-sensitivity troponin, were unremarkable. Complete blood count was notable for marked leukocytosis (white blood cell count >50 × 10^9/l) and reactive thrombocytosis. Electrocardiogram demonstrated diffuse ST-segment elevations. As the patient's chest pain with ST-segment elevation was concerning for an acute coronary syndrome, urgent coronary angiography was performed to exclude an obstructive coronary culprit lesion. Coronary angiography revealed severe ostial right coronary artery disease without significant obstructive disease elsewhere and no acute culprit lesion. The combination of diffuse (rather than territorial) ST-segment elevation, an unremarkable troponin, and the absence of an angiographic culprit favored pericarditis rather than acute myocardial infarction.

Bedside transthoracic echocardiography (TTE) demonstrated a mildly reduced left ventricular ejection fraction with normal right ventricular systolic function and a moderate circumferential pericardial effusion without echocardiographic evidence of tamponade (Figure 1). Specifically, there was no right ventricular diastolic collapse, no significant respiratory variation in mitral inflow, and a non-dilated, non-collapsible inferior vena cava. The patient was treated for acute pericarditis with colchicine and high-dose aspirin. Computed tomography (CT) of the chest demonstrated a moderate pericardial effusion and multifocal pneumonia, prompting initiation of broad-spectrum intravenous antibiotics. Empiric therapy at admission consisted of piperacillin-tazobactam, azithromycin, and trimethoprim-sulfamethoxazole.

Despite antibiotic treatment, the patient rapidly developed worsening chest pain, hypotension, and acute hypoxic respiratory failure requiring non-invasive ventilation. Repeat TTE demonstrated evolving cardiac tamponade requiring emergent pericardiocentesis and drain placement. Drainage yielded frankly purulent fluid, and an indwelling pericardial catheter was left in place from hospital day 1 through day 4. Pericardial fluid analysis revealed turbid, yellow fluid with a white blood cell count of 72,750/µl (100% segmented neutrophils, 0% lymphocytes), red blood cell count <3,000/µl, pH <6.8, glucose <2 mg/dl, total protein 6.5 g/dl, lactate dehydrogenase 8,177 U/L, and an elevated adenosine deaminase of 158 U/L, findings consistent with purulent bacterial pericarditis. Although ADA is typically associated with tuberculosis, TB pericardial effusions are characteristically lymphocyte-predominant, whereas this fluid was 100% neutrophils with heavy growth of pneumococcus on culture, making TB highly unlikely and identifying pyogenic infection as the ADA source.

Blood cultures obtained at presentation, prior to the first antibiotic dose, grew *Streptococcus pneumoniae*, and pericardial fluid culture also showed growth of *S. pneumoniae*, establishing a definitive microbiologic diagnosis of invasive pneumococcal purulent pericarditis. The isolate

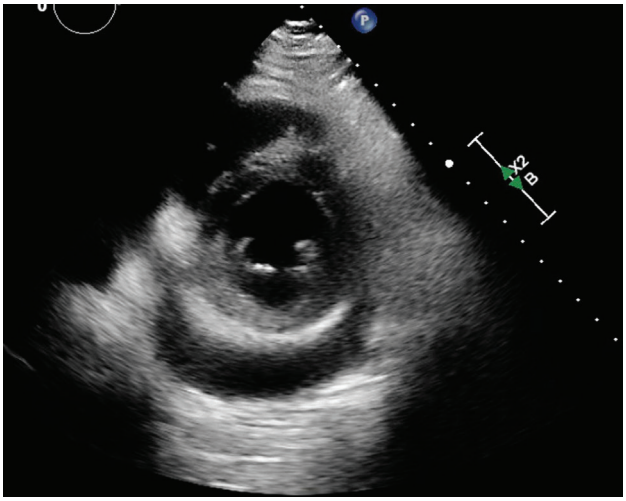


Figure 1. *Initial transthoracic echocardiogram demonstrating a moderate circumferential pericardial effusion without evidence of cardiac tamponade.*

was susceptible to penicillin, cefotaxime, ceftriaxone, levofloxacin, meropenem, and vancomycin, and resistant to erythromycin and tetracycline; pneumococcal serotyping was not performed. Recovery of the same organism from both the bloodstream and the pericardial space supported invasive pneumococcal disease with either hematogenous or contiguous spread from the concurrent multifocal pneumonia.

On hospital day 2, antimicrobial therapy was narrowed to intravenous ceftriaxone 2 g daily with the addition of vancomycin, and piperacillin-tazobactam, azithromycin, and trimethoprim-sulfamethoxazole were discontinued. Vancomycin was discontinued once susceptibilities returned, leaving ceftriaxone 2 g daily as definitive therapy. The pericardial drain was removed on day 4 following a decline in daily output, clinical improvement, and echocardiographic confirmation of a small residual effusion. The effusion was non-loculated at that time, and neither intrapericardial fibrinolysis nor surgical drainage was required for the pericardial space. The leukocytosis and thrombocytosis normalized by day 5.

Following drain removal, the patient developed recurrent hypotension and altered mental status. Repeat TTE demonstrated re-accumulation of a small-to-moderate pericardial effusion (Figure 2) that remained hemodynamically stable, without right atrial or right ventricular collapse, without respiratory inflow variation, and without inferior vena cava plethora. CT of the chest on Day 6 demonstrated an improved, small-to-moderate pericardial effusion but interval enlargement of moderate-to-large bilateral pleural effusions with near-complete collapse of the lower lobes. Since the pericardial effusion was improving and without tamponade features, while the pleural effusions were enlarging, the hemodynamic and respiratory deterioration was attributed to the pleural process rather than to the recurrent pericardial effusion; thus, repeat pericardiocentesis and surgical pericardial window were deferred in favor of continued echocardiographic surveillance.

Given concern for a complicated parapneumonic process, the patient underwent right-sided thoracentesis with chest tube placement on day 10. Pleural fluid was orange and turbid with a white blood cell count of 267/µl (83% segmented neutrophils, 12% lymphocytes), pH 7.63, glucose 98 mg/dl, total protein 4.4 g/dl, lactate dehydrogenase 714 U/L, albumin 1.8 g/dl, and amylase 50 U/L. By Light's criteria, this represented an exudate, but the near-normal pH and glucose were not consistent with frank empyema. Pleural fluid cultures showed no growth. Intrapleural tissue plasminogen activator and dornase alfa were initiated on Day 11 for the loculated collection. The chest tube was removed on day 13 following decreasing output and continued clinical improvement.

The patient improved with continued intravenous ceftriaxone 2 g daily, which was maintained through day 19. He was discharged on oral high-dose amoxicillin 1 g three times daily to complete a total 4-week course, a duration

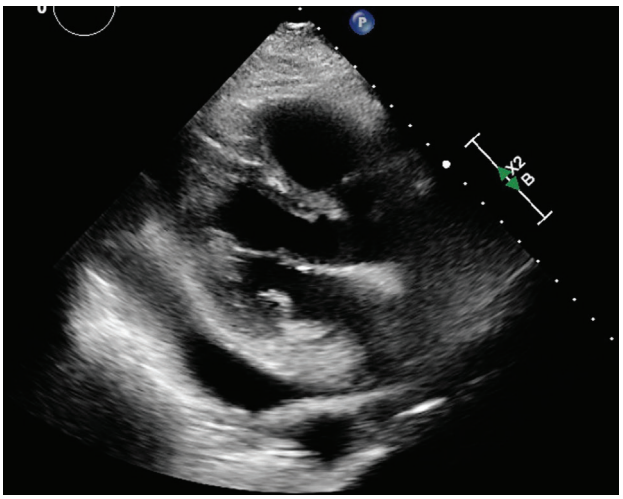


Figure 2. *Repeat transthoracic echocardiogram demonstrating recurrent pericardial effusion following pericardiocentesis.*

Table 1. *Timeline of the patient's hospital course.*

Hospital Day	Event
Day 1-2	Presented with pleuritic chest pain, ECG with diffuse ST-segment elevations, coronary angiography without culprit lesion, TTE with moderate pericardial effusion without tamponade, CT Chest with multifocal pneumonia and pericardial effusion. Further chest pain and hypotension, with repeat TTE now showing tamponade physiology; pericardiocentesis with drain placed.
Day 2	Pericardial fluid analysis: blood and pericardial fluid grew *S. pneumoniae*.
Day 4	Pericardial drain removed, Ceftriaxone monotherapy continued.
Day 4-5	Recurrent hypotension and altered mental status. TTE with stable, recurrent effusion without tamponade.
Day 6	CT chest with improved pericardial effusion, enlarging bilateral pleural effusions.
Day 10	Right thoracentesis, pleural fluid cultures negative.
Day 13	Chest tube removed.
Day 19	Discharged on oral amoxicillin 1 g PO TID.
Day 28	Antibiotic course completed (total 4 weeks).

selected in accordance with the patient's invasive pneumococcal purulent pericarditis with concurrent pneumonia and pleural involvement. Colchicine and high-dose aspirin were continued during admission and at discharge. At early outpatient follow-up, he reported improvement in his symptoms, including resolution of chest pain and improvement in dyspnea. Given symptom resolution, colchicine and aspirin were discontinued at the first outpatient visit.

At approximately 1-year follow-up, repeat TTE demonstrated a normal left ventricular ejection fraction of 55% with complete resolution of the pericardial effusion and no echocardiographic features of constrictive physiology. There was no evidence of recurrent infection over the follow-up period. Table 1 summarizes the clinical course.

Discussion

This case highlights several clinically important findings, including the rarity of pneumococcal purulent pericarditis in the modern era, the coexistence of HIV infection and sarcoidosis as competing contributors to pericardial disease, the diagnostic challenge posed by the absence of tamponade physiology at initial presentation, and the individualized management of recurrent pericardial effusion following pericardiocentesis. A particular strength of this report is the isolation of *S. pneumoniae* from both blood and pericardial fluid cultures, providing definitive microbiologic confirmation of invasive pneumococcal purulent pericarditis. The recovery of an identical organism from the bloodstream and the pericardial space, in the setting of concurrent multifocal pneumonia, supports pneumonia as the probable primary source with subsequent invasive spread to the pericardium. The detailed clinical course, including progression from uncomplicated pericardial effusion to cardiac tamponade, recurrence after drain removal, and successful pathogen-directed therapy without repeat pericardial intervention, provides additional educational value. Serial echocardiography proved essential in identifying progression to tamponade and subsequently confirming hemodynamic stability after effusion re-accumulation.

Purulent bacterial pericarditis is now exceedingly uncommon in developed countries, where most cases of acute pericarditis are idiopathic or presumed viral [5]. Historically, *S. pneumoniae* was among the leading causes of bacterial pericarditis but has become an exceptional pathogen following widespread pneumococcal vaccination and antibiotic use [3,4]. The 2025 American College of Cardiology (ACC) Expert Consensus Statement continues to recognize pneumococcus as a bacterial cause of infective pericarditis while emphasizing the predominance of idiopathic and viral etiologies in Western populations [9].

Previously reported cases have similarly described pneumococcal pericarditis in association with pneumonia, pleural disease, immunosuppression, and cardiac tamponade [3,4,10]. However, the present case is distinctive because of the coexistence of HIV infection and pulmonary/hepatic sarcoidosis, both of which are independently associated with pericardial disease, thereby broadening the differential diagnosis of pericardial effusion. Although combination antiretroviral therapy has reduced the incidence of HIV-associated pericardial disease, HIV remains an important risk factor for invasive infection with encapsulated organisms such as *S. pneumoniae* [6,7]. Notably, invasive pneumococcal disease developed despite prior vaccination with PCV20, underscoring that vaccination does not eliminate the risk of invasive pneumococcal infection in immunocompromised hosts, whether through infection with a non-vaccine serotype, an incomplete serologic response to vaccination in the setting of HIV, or breakthrough disease. Sarcoidosis may also cause pericardial effusion and cardiac tamponade, further complicating the initial diagnostic assessment [8].

The initial presentation of pleuritic chest pain, diffuse ST-segment elevations, and pericardial effusion was consistent with acute pericarditis, and treatment with colchicine and aspirin aligned with current guidance [9]. However, the concurrent finding of multifocal pneumonia raised suspicion for bacterial pericarditis. Hematogenous dissemination or contiguous extension from the pulmonary or pleural space are recognized mechanisms of pneumococcal pericardial infection [3,4]. The subsequent growth of *S. pneumoniae* from both blood and pericardial fluid cultures confirmed invasive pneumococcal purulent pericarditis.

Bedside echocardiography was critical because the initial study showed an effusion without tamponade physiology, whereas repeat imaging during clinical deterioration demonstrated evolving cardiac tamponade. This progression underscores the dynamic nature of pericardial disease and supports serial echocardiographic reassessment when symptoms or hemodynamics worsen [9].

Pericardiocentesis remains first-line therapy for cardiac tamponade because it provides immediate hemodynamic relief while permitting microbiologic diagnosis and guiding antimicrobial therapy [9]. In this case, recurrent effusion developed after drain removal, but serial echocardiography demonstrated no recurrent tamponade. The subsequent episode of hypotension and altered mental status coincided with interval enlargement of loculated bilateral pleural effusions and worsening atelectasis, while imaging showed the pericardial effusion to be improving; thus, the deterioration was attributed to the pleural process rather than the pericardial space. Although current ACC guidance supports surgical pericardial window creation for recurrent large effusions or persistent tamponade after prior pericardiocentesis [9], the recurrent effusion remained hemodynamically stable, allowing conservative management without repeat pericardial intervention

under close echocardiographic monitoring. We emphasize that this approach reflects the course of a single, carefully monitored patient and should not be generalized. Purulent pericarditis carries a substantial risk of recurrent, loculated infection and constrictive pericarditis, and the decision between repeat drainage, intrapericardial fibrinolysis, and surgical pericardial window should be individualized based on hemodynamics, drainage adequacy, infection control, and early surgical assessment. The concurrent, culture-negative but exudative pleural effusions were consistent with a complicated parapneumonic process contributing to respiratory compromise and reflect contiguous pleuropulmonary spread of pneumococcal infection [4].

Purulent pericarditis should remain in the differential diagnosis of immunocompromised patients presenting with pericardial effusion and concurrent pulmonary infection, even when initial echocardiography does not demonstrate tamponade. Worsening dyspnea, hypotension, or respiratory failure should prompt urgent repeat echocardiography.

Management requires both source control and pathogen-directed antimicrobial therapy. Empiric broad-spectrum antibiotics should be initiated while blood and pericardial fluid cultures are pending, followed by targeted therapy once susceptibilities are available. In this case, intravenous ceftriaxone followed by oral amoxicillin was used to complete a four-week course. Multidisciplinary collaboration among cardiology, infectious disease, critical care, and procedural teams can help optimize drainage strategy, antimicrobial selection, and monitoring.

This report is limited by its single-patient design. As with all case reports, the findings are not generalizable but provide clinically relevant insights into the diagnosis and management of this rare condition.

Long-term follow-up is essential because constrictive pericarditis remains an important late complication of purulent pericardial inflammation [1]. Serial echocardiography should document effusion resolution and assess for features of pericardial constriction [9]. This case reinforces that early recognition, repeated imaging, microbiologic confirmation, prompt drainage, and tailored antimicrobial therapy remain fundamental to improving outcomes in this rare but potentially fatal disease.

Conclusion

This case is distinguished by four important features: the rarity of pneumococcal purulent pericarditis in the antibiotic and vaccine era, the coexistence of HIV and sarcoidosis predisposing to invasive pneumococcal disease, the diagnostic challenge of differentiating infectious from noninfectious causes of pericardial effusion, and the management of recurrent pericardial effusion following pericardiocentesis. This report reinforces the importance of maintaining a high index of suspicion for bacterial pericarditis in immunocompromised patients presenting with acute pericarditis and concurrent pulmonary infection. Early echocardiographic assessment, prompt pericardial drainage, microbiologic confirmation, and targeted antimicrobial therapy remain the cornerstones of management. The favorable outcome with conservative management of the recurrent effusion should not be interpreted as generally applicable. Decisions regarding repeat drainage versus conservative management should be individualized based on hemodynamics, drainage adequacy, infection control, and surgical assessment. Long-term follow-up is essential to monitor for constrictive pericarditis and other late sequelae. This case adds to the limited contemporary literature on pneumococcal purulent pericarditis and reinforces principles of early recognition and management.

What is new?

Culture-proven pneumococcal purulent pericarditis is an uncommon but rapidly progressive cause of cardiac tamponade. This case demonstrates how repeated echocardiography, timely pericardial drainage, and pathogen-directed antimicrobial therapy can be lifesaving in immunocompromised patients.

List of Abbreviations

ACC	American College of Cardiology
ADA	Adenosine deaminase
CD4	Cluster of differentiation 4 (T-lymphocyte count)
CT	Computed tomography
ECG	Electrocardiogram
HIV	Human immunodeficiency virus
PCV20	20-valent pneumococcal conjugate vaccine
PO	By mouth
ST	ST segment
TB	Tuberculosis
TID	Three times daily
TTE	Transthoracic echocardiography

Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

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Consent for publication

Written informed consent for publication of this case report and the accompanying images was obtained from the patient.

Ethical approval

Ethical approval was not required at our institution for the publication of this anonymous case report.

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Summary of the case

1	Patient (gender, age)	64-year-old male
2	Final diagnosis	Culture-proven *Streptococcus pneumoniae* purulent pericarditis with cardiac tamponade
3	Symptoms	Pleuritic chest pain, progressive dyspnea
4	Medications	Colchicine, high-dose aspirin, broad-spectrum intravenous antibiotics, intravenous ceftriaxone, oral amoxicillin
5	Clinical procedure	Coronary angiography, serial transthoracic echocardiography, emergent pericardiocentesis with drain placement, right-sided thoracentesis and chest tube placement
6	Specialty	Cardiology, Infectious Diseases