

Received: 2013.10.01
Accepted: 2014.03.14
Published: 2014.11.19

ISSN 1941-5923
© Am J Case Rep, 2014; 15: 504-507
DOI: 10.12659/AJCR.889851

Massive Purulent Pericardial Effusion Presenting as Atrial Fibrillation with Rapid Rate: Case Report and Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF **Amit Kathrotia**
ABCDF **Mohan R. Hindupur**

Department of Cardiology, Heartland Regional Medical Center, St. Joseph, MO, U.S.A.

Corresponding Author: Amit Kathrotia, e-mail: amit.kathrotia@gmail.com
Conflict of interest: None declared

Patient: Male, 59
Final Diagnosis: Pleural and pericardial effusion from a *Streptococcus pneumoniae*
Symptoms: Chest pain • chills • cough • fever • shortness of breath
Medication: —
Clinical Procedure: Pericardiocentesis • pericardial window
Specialty: Cardiology

Objective: Rare disease
Background: Although pericardial effusion with afib is not rare, the combination of purulent pericardial effusion presenting as afib is not a common occurrence particularly in the developing world. The more common symptoms associated with purulent pericardial effusion are fever, dyspnea, and tachycardia. Without prompt recognition followed by antibiotics and surgical drainage, tamponade, and shock can potentially lead to death.

Case Report: A 59-year-old male was transferred to our hospital for evaluation of afib with rapid rate associated with cough and dyspnea. He reported fevers, chills, cough and sputum for 1 week. Complaints included chest pain with relief upon lying down. Patient was afebrile with a pulse of 101 and blood pressure of 119/89. WBC 39,200 cells/ml. Chest X-RAY showed right lower lobe pneumonia and EKG revealed afib, rapid ventricular response, and secondary ST changes inferolaterally. Pericardial effusion and thickened pericardium were evident on echo. Patient was treated for community acquired pneumonia, along with heparin and IV amiodarone. Both sputum cultures and pericardiocentesis revealed *S. Pneumoniae*. Cardioversion reestablished sinus mechanism. Initially pericardial effusion resolved, but later reaccumulated at which point it was decided to perform a subxiphoid pericardial window. Follow up showed no effusion and patient was asymptomatic.

Conclusions: Purulent pericardial effusion with atrial fibrillation and rapid ventricular rate needs to be recognized promptly. Because friction rub and chest pain are not present in every case, prompt management in the setting of pneumonia and minor hemodynamic derangements can aid in the treatment of this potentially life threatening disease.

MeSH Keywords: Atrial Fibrillation • Community Acquired Pneumonia • Purulent Pericardial Effusion

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/889851>



1032



3



8



Background

Purulent pericardial effusion, even in the antibiotic era, has a high mortality. Purulent pericardial effusion commonly presents with fever, shortness of breath, and tachycardia. Atrial fibrillation as a presenting manifestation is unusual. Friction rub and chest pain, which are commonly associated with viral pericarditis, are often very minor or not appreciated at all with purulent effusion [1]. The amount of fluid recovered from pericardiocentesis plays a role in recurrence of the effusion and the development of constrictive pericarditis. Pericardial window and pericardiectomy along with targeted antibiotic therapy play an important role in resolution of the disease process. The association of purulent pericardial effusion presenting as atrial fibrillation has not been well documented. Commonly implicated etiologies include *Staphylococcus aureus*, *Streptococcus*

species, and a variety of gram-negative agents. We present this case of a massive purulent pericardial effusion due to community-acquired pneumonia in a non-immunocompromised patient who presented with atrial fibrillation requiring electrical cardioversion, pericardiocentesis, and pericardial window procedure due to recurrence of effusion.

Case Report

A 59-year-old white male was transferred from an outlying community hospital for evaluation of atrial fibrillation with a rapid rate, with chief complaints of cough and shortness of breath for 1 week, and was found to have right lower-lobe pneumonia. He reported having 1 episode of fever, chills, and rigors, with rusty colored sputum production of 1-week duration. He complained

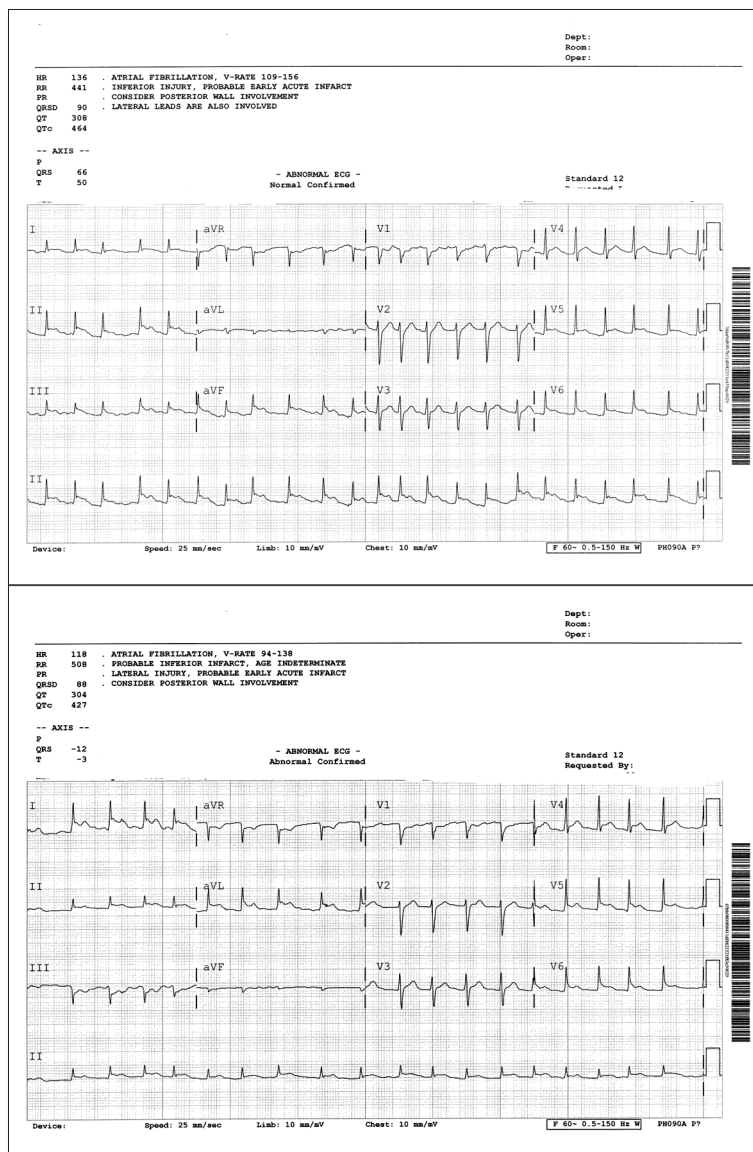


Figure 1. There is evidence of Atrial Fibrillation due to variation of the QRS intervals and P wave morphology as well as alternation of the amplitude to indicate pericardial injury.



Figure 2. Chest X-ray purulent pericardial effusion.

of pleuritic chest pain relieved upon lying down. The patient was afebrile, with a blood pressure of 119/89, pulse of 101, and respiratory rate of 18. Lab test results showed WBC of 39 200 cells/ml. EKG showed atrial fibrillation, rapid ventricular response, and secondary ST changes inferolaterally (Figure 1). A chest X-ray showed right lower lobe pneumonia (Figure 2). Echocardiogram demonstrated mild concentric left ventricular hypertrophy, moderate pericardial effusion with echogenic material in the pericardial space, along with a thickened pericardium (Figure 3). He was started on Azithromycin PO and IV Ceftriaxone for community-acquired pneumonia and heparin protocol was begun. IV Amiodarone was initiated to abate the atrial fibrillation. Blood culture results were negative, but sputum cultures grew *Streptococcus pneumoniae*. After 48 h, pericardiocentesis was done and 1200 ml of yellow colored pus-like fluid with a slightly foul-smelling odor was aspirated. The fluid grew *Streptococcus pneumoniae* and showed sensitivity to Azithromycin, Cefotaxime, Ceftriaxone, Levofloxacin, and Meropenem. CT showed thickened pericardium with cardiac enlargement. The next day the WBC gradually decreased to 33 800 cells/ml and a subsequent echocardiogram done on day 5 showed resolving effusion. Ceftriaxone was switched over to a combination of IV Meropenem and IV Vancomycin. The patient's antibiotic regiment was then changed to IV Ceftriaxone and Levofloxacin PO. On day 10, the WBC count increased to 35 700 cells/ml and a CT scan showed reaccumulation of pericardial fluid, thickened pericardium, and air. At this time electrical cardioversion was done and the recent onset of atrial fibrillation/flutter was converted to sinus mechanism. In view of this rapid recurrence of effusion, it was felt that open drainage was preferable over repeat pericardiocentesis. Subxiphoid pericardial window was then performed on day 12, draining 500 ml of purulent fluid. Pericardial biopsy showed fibropurulent material consistent with acute pericarditis. The patient was discharged home on day 16 with Levofloxacin PO and Linezolid PO; WBC decreased to 17 200 cells/ml. Amiodarone was discontinued at week 5. A follow-up echocardiogram at

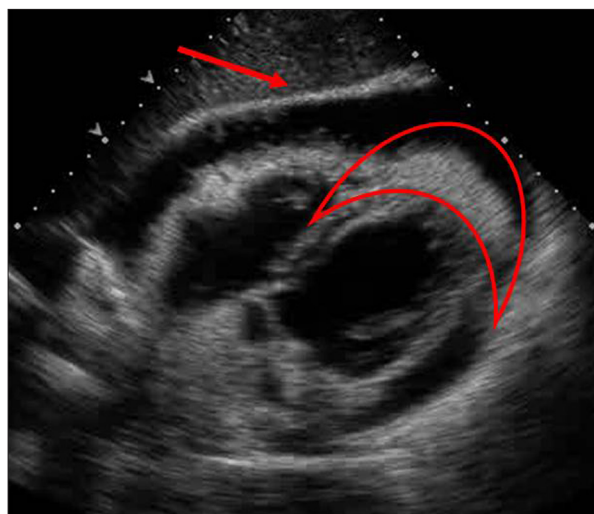


Figure 3. Echocardiographic density suggesting fibrinous material over the epicardium (crescent) and a thickened pericardium (arrow).

week 9 showed no evidence of effusion, and the patient remained asymptomatic.

Discussion

Atrial fibrillation is commonly seen with pericarditis and effusion of any etiology. Generally, arrhythmias are evident after a few days of active inflammatory course. Atrial fibrillation with rapid rate as a presenting symptom of large purulent pericardial effusion is rare and very few documented cases are reported in literature. The cases we wish to review in our discussion were within the United States and involved adult patients. We chose this population because of the focus of our clinical experience. Purulent pericarditis is rarely seen in the developing world today. Cases of pericarditis are often related to viral infection, neoplasia, radiation, connective tissue disorders, and metabolic syndromes [2]. Previously, children and young adults were more often diagnosed with purulent pericarditis than older adults were, but that has reversed, partially due to the advent of antibiotics, standard vaccination protocols, and modified surgical techniques [2]. A large study examining patients with purulent pericarditis from 1889 to 1975 demonstrated that before 1944, 43% of patients were below the age of 10 years, but after 1944 that percentage was only 13% [2]. The most common etiologies include *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Viridans streptococci* [3]. Immunocompromised patients have a higher incidence of Staph effusion. Commonly, infection spreads locally from another primary focus (e.g., the lung is a source of *Streptococcus* species) [4]. Our patient presented with CAP with secondary involvement of the pericardium. This commonly resolves within 3–4 days of treatment, as was the

case with our patient, indicated by the decreasing WBC count, abating symptoms, and echocardiographic findings. We therefore postulated that the reaccumulated purulent pericardial effusion was responsible for the atrial fibrillation. This was indicated by the rise in WBC and the presence of atrial fibrillation/flutter during this time. The outcomes of pericarditis are often associated with the etiology. Purulent forms often lead to a high mortality rate due to arrhythmias and tamponade if not promptly recognized and evacuated [5]. Signs of tamponade may not be clinically apparent until shock ensues, making a high index of suspicion in the setting of pneumonia and minor hemodynamic derangements important in treatment. Of particular note in our patient was his extremely elevated WBC count of 39 000 cells/ml, which seems to follow the trend in patients with gram-positive cocci and who are above the age of 50 years [6,7]. Long-term consequences include constriction. Current treatment modalities, including

targeted antibiotics and surgical drainage, have led to reductions in mortalities [8].

Conclusions

Purulent pericardial effusion presenting with atrial fibrillation as an initial manifestation is rare. Early and effective drainage of purulent material seems to have the best outcome. Surgical drainage with window should be considered early in the treatment course.

Acknowledgements

Mohan Hindupur, Robert Grant, and Mazda Biria provided scientific guidance, participated in discussion, and provided resources for data collection. Paul Shandley provided technical assistance and edited images.

References:

1. Patel S, Maves R, Barrozo CP et al: Mycotic pseudoaneurysm and purulent pericarditis attributable to methicillin-resistant *Staphylococcus aureus*. *Mil Med*, 2006; 171(8): 784–87
2. Klacsmann PG, Bulkley BH, Hutchins GM: The changed spectrum of purulent pericarditis: An 86 year autopsy experience in 200 patients. *Am J Med*, 1977; 63(5): 666–73
3. Bhaduri-McIntosh S, Prasad M, Moltedo J, Vázquez M: Purulent Pericarditis Caused by Group A Streptococcus. *Texas Heart Inst J*, 2006; 33(4): 519–22
4. Saenz RE, Sanders CV, Aldridge KE, Patel MM: Purulent Pericarditis with Associated Cardiac Tamponade Caused by a *Streptococcus pneumoniae* Strain Highly Resistant to Penicillin, Cefotaxime, and Ceftriaxone. *Clin Infect Dis*, 1998; 26(3): 762–63
5. Rosenthal A: Massive purulent pericarditis and cardiac tamponade caused by *Staphylococcus aureus* urosepsis: case report. *J Cardiovasc Surg*, 2002; 43(6): 837–39
6. Parikh SV, Memon N, Echols M et al: Purulent Pericarditis: Report of 2 Cases and Review of the Literature. *Medicine*, 2009; 88(1): 52–65
7. El-Ahdab F, East M, Sexton D, Bashore T: *Staphylococcus aureus* Pericardial Abscess: Case Report. *South Med J*, 2003; 96(9): 926–27
8. Wilkins RB, Jarvis FJ, King RL: Purulent pericarditis due to hemophilus influenzae, type B. *Am Heart J*, 1951; 42(5): 749–57