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A Case Presentation of Pericarditis Associated with Haemophilus Influenzae Bacteremia

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INTRODUCTION

Acute pericarditis, or inflammation of the pericardial sac, is a clinical condition which can often be attributed to a variety of underlying etiologies, including infection, autoimmune disease, trauma, and malignancy. While viral infections are commonly implicated in the etiology of pericarditis, bacteria known to be associated with pericarditis include staphylococcus species, streptococcal species, tuberculosis, and in children, Haemophilus influenzae.¹ Here we present a rare case of pericarditis in an adult male patient which occurred in association with Haemophilus influenzae bacteremia.

CASE PRESENTATION

A 53-year-old male with hypertension, hyperlipidemia, poorly controlled type 2 DM (insulin dependent though noncompliant with home medications), crack cocaine use presented to the ED with several days of shortness of breath with associated pleuritic chest pain that was not affected by positional changes. The shortness of breath developed suddenly four days prior to admission in conjunction with the pleuritic chest pain and a dry cough.

His vital signs on admission were notable for temperature of 97.3 degrees Fahrenheit, heart rate of 138 beats per minute, blood pressure 113/77, respiratory rate 18 breaths/minute, and oxygen saturation of 96% on room air. Labs on admission were significant for troponins (Troponin T Hs) of 16-->18, erythrocyte sedimentation rate >130 mm/hr, and c-reactive protein of 44.70 mg/dL. White blood cell count was 18.4 B/L with a hemoglobin of 11.4 g/dL, and platelets of 328 B/L and hemoglobin A1c of 9.7. The urine drug screen was positive for cocaine. EKG was notable for diffuse anterolateral and inferior ST elevations with frequent atrial ectopy (**Figure 1**). This progressed during hospitalization to both paroxysmal atrial fibrillation and atrial flutter.

A CT angiogram of his chest to rule out pulmonary embolism (PE) was negative but did show a small pericardial effusion with pericardial thickening and mediastinal fat stranding with small mediastinal lymph nodes concerning for acute pericarditis. Echocardiogram showed an ejection fraction of 60% with a small to moderate pericardial effusion. No tamponade physiology was present (**Figure 2**).

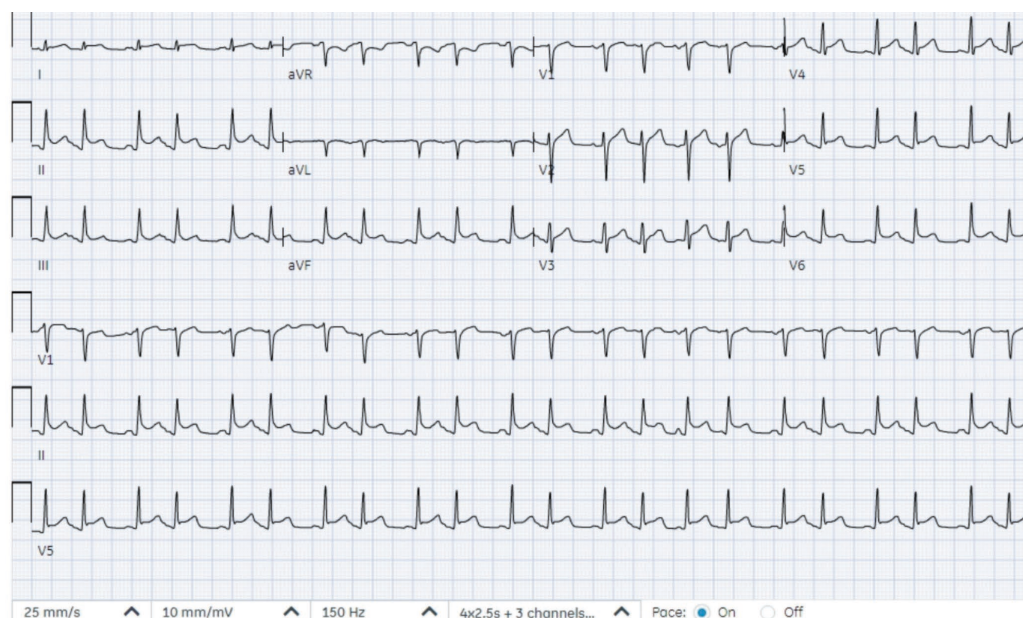


Figure 1: EKG



Figure 2: Echo

Our respiratory pathogen viral panel nasal swab (which includes adenovirus, rhinovirus, human metapneumovirus, parainfluenza, multiple coronaviruses, and others), SARS-CoV-2, and influenzae testing were all negative. Interestingly, blood cultures collected on the day of admission were positive for *Haemophilus Influenzae* growing in two sets of blood cultures; one in 14 hours and 57 minutes, and the other in 16 hours.

The primary hospital medicine team requested consultations from infectious disease and cardiology for co-management. Pericardiocentesis was recommended for diagnostic and therapeutic purposes, however due to the small size of the effusion, the risks were considered too great. The patient's antibiotics were narrowed from piperacillin-tazobactam to ceftriaxone based on blood culture sensitivities and he was eventually transitioned to levofloxacin on discharge for a total of 4 weeks of antibiotic therapy. High dose aspirin and colchicine were both started as well with plans for a 6 week and 3-month course respectively.

DISCUSSION

Acute pericarditis is an infrequent cause of chest pain in patients presenting to the emergency department, accounting for only 0.1% of patients that are hospitalized with chest pain.² However, clinicians must be quick to recognize this condition, as untreated pericarditis can result in devastating complications including cardiac tamponade and constrictive pericarditis.^{1,3} In addition, patients with pericarditis have an increased risk for development of recurrent pericarditis.²

Diagnosis of pericarditis is made with a combination of detailed history taking, physical exam findings, lab work, and imaging. Patients with pericarditis will almost always

present with chest pain, which may be alleviated by sitting up and leaning forward, and may present with fever, dyspnea, and cough. While a pericardial friction rub is almost 100% specific for pericarditis, its sensitivity is much more variable.¹ EKG changes vary with different stages of pericarditis, but include diffuse ST segment elevation, ST depression in AVR or V1, PR depression, and T wave inversions.⁴ Lab work is less specific but includes leukocytosis, elevated troponins, and elevated inflammatory markers (ESR, CRP). Chest Xray is often normal, but ultrasound and CT imaging may show evidence of pericardial effusion and/or inflammation. Guidelines for diagnosing pericarditis involve meeting at least two of the four criteria as follows: 1- chest pain; 2- pericardial friction rub; 3- EKG changes including ST elevations/PR depressions; 4- new or worsening pericardial effusion.¹ If a pericardial effusion is large enough, a pericardiocentesis should be performed for diagnostic and often therapeutic purposes.

Our patient was unique in that his pericarditis was thought to be associated with *Haemophilus influenzae* (H. flu) bacteremia. H. flu is a gram-negative coccobacilli that causes many different pathologies, especially in the pediatric population. These include otitis media, meningitis, cellulitis, and upper respiratory tract infections. Vaccination against H. flu type b is offered to children in a 3-4 dose series, but the vaccine does not protect against other strains of H. flu, and those that are unvaccinated and immunocompromised are also at increased risk for this disease.⁵ While very rare, a few case reports have shown H. flu pericarditis in immunocompetent individuals (confirmed through pericardiocentesis).^{5,6} In fact, an older case report in CHEST did describe two cases of pericarditis in association with H. flu bacteremia.⁶

In our patient, the risk of pericardiocentesis outweighed the benefits secondary to the small size of the effusion. Therefore, it cannot be definitively stated that his pericarditis was purely secondary to *Haemophilus influenzae* without pericardial fluid bacterial isolation. However, the patient was treated under clinical suspicion and objective evidence that H. flu was the likely underlying etiology of pericarditis. He improved rapidly with targeted antibiotic treatment towards H. flu, his chest pain resolved, his effusion remained stable on ultrasound, and he was discharged to a substance use disorder treatment facility. One month after discharge, he presented to the infectious disease clinic and was feeling almost completely back to his baseline after finishing his antibiotic course two weeks prior to that visit.

CONCLUSION

Our case report is one of only a few others in the literature that demonstrates a possible association between H. flu and pericarditis in an otherwise immunocompetent adult. Clinicians must be astute in recognizing pericarditis as a potential cause of chest pain, as prompt treatment can reduce the risk of life-threatening complications.

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