Pericarditis Associated with *Hemophilus influenzae* Type B Pneumonia and Bacteremia in Two Adults*

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Two cases of pericarditis associated with *Hemophilus influenzae* bacteremia and pneumonia are reported and the literature reviewed. Both patients were treated with antibiotics alone and had resolution of their illnesses without apparent adverse sequelae. Neither patient required pericardial drainage. It is suggested that there is a stage in the development of *H influenzae* pericarditis at which antibiotic treatment alone can be curative.

*Hemophilus influenzae* has become an increasingly common cause of serious infections in adults. The incidence of Gram-negative organisms in the etiology of pyogenic pericarditis has also been increasing, yet only nine cases of *H influenzae* pericarditis have been reported in adults. All previously reported cases have been treated with surgical drainage in addition to antibiotics.

We report two cases of pericarditis occurring in the setting of *H influenzae* pneumonia and bacteremia. The signs and symptoms of pericarditis resolved in both patients with medical therapy alone.

**Case Reports**

**Case 1**

An 18-year-old white man was admitted to Metropolitan Nashville General Hospital with chest pain, fever, and tachypnea. Five days prior to admission, he developed a sore throat which was treated with erythromycin. Three days prior to admission, he had the sudden onset of stabbing, substernal chest pain which radiated to the shoulders and back. The pain was pleuritic and relieved by sitting upright.

On examination the patient was sitting and leaning forward because of chest pain. His temperature was 38.8°C; respirations, 40/min; pulse rate, 120/min, and blood pressure, 110/80 mm Hg without pulsus paradoxus. The chest was clear to auscultation and percussion. Cardiac examination was significant only for regular tachycardia.

Laboratory examination revealed a white blood cell count of 24,200 cu mm with a left shift. Chest x-ray examination initially showed clear lung fields and a heart size that was at the upper limits of normal. ECG demonstrated diffuse ST elevation consistent with pericarditis (Fig 1).

The patient was treated with intravenous gentamicin 60 mg every eight hours and intravenous cefazolin 1 g every six hours. He was afebrile and without chest pain within 24 hours. A three-component pericardial friction rub was heard by several observers on the second hospital day, and the chest x-ray film showed a right lower lobe infiltrate with a right pleural effusion. Thoracocentesis revealed this to be a sterile transudate. ECG continued to show diffuse ST elevation, but M-mode echocardiogram did not show a pericardial effusion. The blood cultures grew *H influenzae* (type B, biotype II) sensitive to ampicillin. The antimicrobial therapy was changed to intravenous ampicillin 1 g every six hours, and was continued for a total of seven days. Oral ampicillin 500 mg every six hours was then continued for ten more days on an outpatient basis. Follow-up
examination eight weeks later documented resolution of the pulmonary infiltrates and pleural effusion. The ST segments on ECG were no longer elevated.

CASE 2

A 33-year-old black man was admitted to Metropolitan Nashville General Hospital because of fever and productive cough. Three days before admission the patient began to feel febrile, and he developed a cough productive of purulent, blood-streaked sputum and bilateral pleuritic chest pain. He experienced rigors on the day of admission. His past history included alcoholism and intravenous drug abuse.

On examination, the blood pressure was 110/90 mm Hg; pulse rate 120/min; temperature 38.3°C; and respirations, 36/min. The pharynx was erythematous. Moist rales were heard at the left lung base posteriorly. There was regular tachycardia without murmur or rub.

The chest x-ray film revealed infiltrates in both lower lobes. Electrocardiogram was notable only for sinus tachycardia. White blood cell count was 16,800/cu mm with a left shift. He was treated with intravenous penicillin G, one million units every four hours, and intravenous oxacillin, 2 g every four hours. By the second hospital day, improvement was noted. His temperature was 37.2°C and the white blood cell count had decreased to 12,100 cu mm. He continued, however, to complain of bilateral pleuritic chest pain.

When results of five of six blood cultures and a sputum culture grew H influenzae (type B, biotype 1), the therapy was switched to intravenous ampicillin 1 g every six hours. The organism did not produce beta lactamase. On the seventh hospital day, the pleuritic chest pain was still present and a chest x-ray film revealed bilateral pleural effusion and an enlarged cardiac silhouette. Auscultation revealed a pericardial rub, and an M-mode echocardiogram was notable for a posterior pericardial effusion. With the addition of salicylates, the patient became afebrile, and he was without chest pain by the tenth hospital day. He was discharged on the twelfth hospital day. Follow-up examination six months after discharge found the patient asymptomatic with good exercise tolerance. Findings on repeat ECG, chest x-ray film and echocardiogram were normal.

DISCUSSION

There have been nine cases of H influenzae pericarditis previously reported in adults. The ages ranged from 23 to 59 years with all but three between 23 and 33 years. The length of illness prior to presentation varied from one to seven days. Five of nine had had sore throats and six of nine had had pulmonary infiltrates and pleural effusions. All the patients were treated with surgical drainage in addition to appropriate antimicrobials, and the hospital stay ranged from 14 to 55 days. Only one of the nine patients died. This was a young woman who apparently recovered from the purulent pericarditis only to die of complications related to sulfonamide-induced thrombocytopenia. This is a surprisingly low mortality rate in light of the nearly 80 percent mortality reported for purulent pericarditis in general.

Our cases had some characteristics in common with patients previously reported. They were young adults without serious underlying diseases. Presentation was abrupt, and the clinical focus was the respiratory system. Upper respiratory tract disease was accompanied by chills, fever and pleuro-pulmonary manifestations.

In neither patient was it unequivocally established whether the pericardial fluid was sterile or purulent. Both patients had a prompt clinical response to antimicrobial therapy specifically directed against a blood culture isolate, and did not develop hemodynamic derangement. It was therefore unnecessary to withdraw pericardial fluid for either diagnostic or therapeutic purposes.

The subsequent course of both patients has remained uncomplicated and they are now beyond the period usually associated with acute constrictive pericarditis. It is noteworthy that the hospital stays of seven and 12 days are shorter than for previously reported patients.

Pericarditis which complicates the course of bacterial pneumonia presumably begins with local inflammation of the parietal pericardium adjacent to the pneumonia. Inflammatory effusion follows and then bacteria invade the pericardial space, leading to purulent accumulation. The indications for removing and analyzing pericardial fluid in this setting may include: 1) the need for microbiologic diagnosis in a patient with suspected bacterial pericarditis; 2) continued evidence of uncontrolled infection in spite of appropriate antimicrobial therapy, or 3) evidence of hemodynamic compromise.

The recommended treatment of bacterial pericarditis has been appropriate antimicrobial therapy and pericardial drainage. Appropriate antimicrobial therapy requires a bacteriologic diagnosis. If the organism cannot be recovered from the blood or pleural fluid, an attempt should be made to obtain pericardial fluid for culture. If H influenzae is the etiologic agent, it is suggested that the patient receive chloramphenicol until it is known whether or not the organism produces beta-lactamase. Cefamandole has also been recommended in this setting. However, there has recently been a report of a treatment failure with cefamandole in a patient with ampicillin-resistant H influenzae pneumonia. If there is no beta-lactamase production by the organism, ampicillin can be used.

Regarding surgical management, some authors have advocated early pericardiectomy. They emphasize the inadequacy and hazards of drainage by pericardiocentesis, and underscore the incidence of recurrent effusion and acute constrictive pericarditis. Other writers advocate pericardiectomy in order to avoid the development of chronic constrictive pericarditis. However, all of these surgical recommendations have been forged from series of patients with either large effusions or tamponade. In addition, it has never been clearly demonstrated that chronic pericardial constriction can follow purulent pericarditis except when caused by tuberculosis.

These two cases illustrate the importance of early
diagnosis and institution of appropriate antimicrobial treatment in this illness. There is apparently a stage in the development of pericarditis associated with bacterial pneumonia at which antibiotic therapy can obviate the need for pericardial drainage. We suggest that when: 1) a bacteriologic diagnosis is known, 2) there is a prompt clinical response to antibiotics, and 3) there is no evidence of hemodynamic compromise, that *H influenzae* pneumonia with pericarditis can be managed without removing pericardial fluid.

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REFERENCES


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**Fourth Annual Symposium on Diagnostic Imaging**

The Section on Radiation Medicine, Brown University School of Medicine, and the Department of Diagnostic Radiology, Rhode Island Hospital, will cosponsor this symposium August 15-18 at the Sheraton-Islander Inn, Newport, Rhode Island. For further information, contact Educational Resources Associates, Inc., PO Box 369, Brookline, Massachusetts 02146 (617.738-8859).