striction? It has been suspected that inspiratory stridor due to nonorganic airway obstruction is brought about by a psychogenic problem, and it is sometimes referred to as emotional or hysterical laryngospasm. The pharyngeal constriction as seen in our case also is most likely to have a psychogenic basis, because of the sudden onset of symptoms, their improvement with the use of sedatives and psychotherapy and the absence of organic abnormalities. Psychogenic pharyngeal constriction, leading to abnormal shape and movement of the epiglottis, may be an additional cause of functional upper airway obstruction with respiratory distress.

References

Rapid Evolution of Cardiac Tamponade due to Bacterial Pericarditis in Two Patients With HIV-1 Infection*

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We describe two HIV-seropositive patients with acute pneumococcal empyema and pericarditis. Cardiac tamponade evolved rapidly in each patient and was reversed with prompt surgical intervention. In each case, immunologic abnormalities were detected which could have facilitated local spread of infection and progression to tamponade. Pericarditis, an otherwise rare manifestation of pneumococcal infection in the antibiotic era, should be anticipated in HIV-seropositive patients with pneumococcal empyema. (Chest 1992; 101:1461-63)

Although echocardiograms of patients infected with HIV-1 commonly show pericardial fluid, we can find only one documented case of purulent pericarditis in such patients. In this report, we describe two HIV-seropositive subjects in whom contiguous spread of pneumococcal infection from the left pleural space caused pericarditis and rapid evolution of cardiac tamponade.

Case Reports
Case 1
A 32-year-old woman was admitted with fever, cough productive of yellow sputum, and left-sided pleuritic pain for three days.

Two years prior to this admission, she was treated with chemotherapy for malignant lymphoma of intermediate grade involving the central nervous system. Simultaneously, pulmonary tuberculosis was diagnosed. The lymphoma and TB were remitted with therapy. The patient was found to have antibodies to HIV-1 by Western blot. The only risk factor for HIV-1 infection was a history of sexual promiscuity.

One year prior to admission, the patient was treated with penicillin, oxacillin, and thoracentesis for a right middle lobe pneumonia with empyema; Gram stain of the pleural fluid documented Gram-positive cocci in pairs and clusters. Spinal fluid examination and a lymph node biopsy at that time showed no evidence of lymphoma.

On admission, the temperature was 39°C; the heart rate, 140 beats per minute; and the blood pressure, 110/80 mm Hg without pulsus paradoxus. The patient appeared toxic and had generalized lymphadenopathy. In the left posterior hemithorax, tactile fremitus was reduced, and percussion note was dull; breath sounds were absent, and egophony was evident. Chest roentgenogram showed left pleural effusion, bilateral reticulonodular infiltrates, and mediastinal lymphadenopathy; the infiltrates and lymphadenopathy had been stable for the previous two years. In the pleural fluid, pH 7.05; protein, 76 g/L; glucose, 1.11 mmol/L (20 mg/dl); LDH, 2961 IU/L; and WBC, 22,000/cmm with 42 percent polymorphonuclear leukocytes. Gram stains of the sputum and pleural fluid showed Gram-positive diplococci and Gram-negative coccobacilli. A Ziehl-Neelsen stain was negative. A chest tube was inserted in the left hemithorax, and intravenous penicillin G (4 million units every 4 h) and cefotaxime (2 g every 8 h) were prescribed. With this treatment, the patient defervesced within 48 h. Cultures of the pleural fluid

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21644/ on 06/26/2017)
A 27-year-old woman was admitted with the chief complaint of fever, chills, and left pleuritic chest pain for three days. These symptoms were preceded by generalized weakness, weight loss of 10 kg, and cough productive of yellow sputum for two months. The patient denied intravenous drug abuse and transfusion of blood products, but her husband was sexually promiscuous.

On admission, the temperature was 39°C; the heart rate, 120 beats per minute; and the blood pressure 110/70 mm Hg. Generalized lymphadenopathy was evident. The left posterior hemithorax was dull to percussion; breath sounds were absent, but egophony was detectable. A pericardial rub was not heard. A chest roentgenogram showed complete opacification of the left hemithorax and a shift of the mediastinum to the right (Fig 2). An ECG showed ST-segment elevations in the limb leads and in leads V₅, V₆. Analyses of pleural fluid revealed a pH of 6.5; glucose, 0.99 mmol/L (18 mg/dl); protein, 67 g/L; LDH, 7,300 IU/L; and WBC, 50,000/cu mm with 93 percent PMNs, and Gram-positive cocci in pairs and rarely in clusters. A Ziehl-Neelsen stain was negative. The patient defervesced in 48 h following chest tube drainage of empyema and intravenous penicillin G (4 million units every 4 h) and oxacillin (2 g every 6 h) in 48 h. Cultures of pleural fluid grew S pneumoniae.

On the third hospital day, the patient experienced shortness of breath. Examination revealed a heart rate of 120 beats per minute and blood pressure of 90/50 mm Hg with a pulsus paradoxus of 15 mm Hg and a pericardial rub. A chest roentgenogram showed a widened cardiac silhouette. An echocardiogram demonstrated a moderate pericardial effusion and right atrial diastolic collapse. A total of 250 ml of serosanguinous fluid was removed by subxiphoid pericardiotomy. The blood pressure rose immediately to 124/50 mm Hg. A Gram stain of the pericardial fluid showed Gram-positive diplococci. Cultures grew Staphylococcus aureus and Staph epidermidis, but the samples of pericardial fluid were taken from a drainage bag and were therefore thought to be contaminated. A Ziehl-Neelsen stain showed no mycobacteria. Microscopic examination of the pericardium showed fibrous pericarditis. Intravenous penicillin and oxacillin were continued, and the patient remained afebrile and hemodynamically stable.

The two-month history of cough with weight loss and a positive tuberculin test prompted antituberculosis treatment with isoniazid, rifampin, and pyrazinamide. Cultures of sputum and pleural fluid but not pericardial fluid eventually grew Mycobacterium tuberculosis. Antibody to HIV-1 was detected by Western blot.

Immunologic Studies

Studies of immune status are shown in Table 1. They demonstrate deficiencies of CD4+ cells, elevated levels of IgG and circulating immune complexes, and normal complement levels in both patients.

<table>
<thead>
<tr>
<th>Test</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cells, number/cu mm</td>
<td>6,840</td>
<td>5,900</td>
<td>4,500-11,000</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PMNs</td>
<td>5,814</td>
<td>4,400</td>
<td>1,800-7,700</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>608</td>
<td>919</td>
<td>1,000-4,800</td>
</tr>
<tr>
<td>Monocytes</td>
<td>116</td>
<td>314</td>
<td>0-800</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>27</td>
<td>71</td>
<td>0-450</td>
</tr>
<tr>
<td>Basophils</td>
<td>0</td>
<td>23</td>
<td>0-200</td>
</tr>
<tr>
<td>Lymphocyte surface markers, percent of total lymphocytes</td>
<td>84.8</td>
<td>82.4</td>
<td>60-80</td>
</tr>
<tr>
<td>CD3</td>
<td>23.1</td>
<td>22.6</td>
<td>35-55</td>
</tr>
<tr>
<td>CD4</td>
<td>59.4</td>
<td>55.5</td>
<td>18-35</td>
</tr>
<tr>
<td>CD4/CD8</td>
<td>0.39</td>
<td>0.41</td>
<td>&gt;1.0</td>
</tr>
<tr>
<td>CD21</td>
<td>5.5</td>
<td>8.4</td>
<td>5-15</td>
</tr>
<tr>
<td>Immunoglobulin levels, IU/ml</td>
<td>4,990</td>
<td>3,610</td>
<td>640-1,350</td>
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<tr>
<td>IgG</td>
<td></td>
<td></td>
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<tr>
<td>IgM</td>
<td>150</td>
<td>388</td>
<td>56-352</td>
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<tr>
<td>IgA</td>
<td>124</td>
<td>181</td>
<td>70-312</td>
</tr>
<tr>
<td>IgE</td>
<td>26</td>
<td>&lt;5</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Complement levels, CH₅₀, U/ml</td>
<td>359</td>
<td>298</td>
<td>150-310</td>
</tr>
<tr>
<td>C₅₇, mg/dl</td>
<td>155</td>
<td>149</td>
<td>83-177</td>
</tr>
<tr>
<td>C₅₇, mg/dl</td>
<td>20</td>
<td>32</td>
<td>15-45</td>
</tr>
<tr>
<td>Circulating immune complex levels, μgEq/ml</td>
<td>10.8</td>
<td>10.6</td>
<td>&lt;4</td>
</tr>
</tbody>
</table>
**DISCUSSION**

The patients described above had several features in common. Both were women who had acquired HIV-1 infection through sexual exposure. The first patient had a history of tuberculosis, and the second was diagnosed to have it during her hospitalization. Both presented with left-sided pneumococcal empyema, which was treated promptly with antibiotic therapy and chest tube drainage. In each case, pericarditis and tamponade were detected on the third hospital day. Both patients responded to pericardiectomy with defervescence, normalization of hemodynamics, and eventual resolution.

Pericardial effusions are commonly found in patients infected with HIV-1. The effusions typically produce no symptoms or signs and rarely cause tamponade. An infectious agent is usually not found in the fluid, but pericarditis due to opportunistic pathogens has been associated with AIDS.

Although overwhelming bacterial infections occur with increased frequency in HIV-1-seropositive patients, only one case of pericarditis caused by pyogenic bacteria has been described. The subject of that case report presented with pneumonia and bilateral pleural effusions. Thoracentesis was unsuccessful, but *Staphylococcus aureus* was cultured from pericardial fluid. The source of the staphylococcal infection was not determined.

Pneumococcal infection and bacteremia are several times more common in patients infected with HIV-1 than in the general population. Attenuation of CD4+ cells in HIV-1 infection results in deficiency of opsonic antibodies and ineffective elimination of encapsulated bacteria. Attenuated γ interferon production by CD4+ cells causes defective antigen presentation, decreased production of toxic O2 species by phagocytes and diminished clearance of IgG coated bacteria and immune complexes by reticuloendothelial system. These derangements explain the elevated levels of circulating immune complexes in our patients.

Although the pericarditis of our patients could have resulted from generalized bacteremia, it was more likely due to local spread of infection from the left lung and pleura. Communications between the bronchial and pericardial arterial systems could have facilitated this progression.

Pneumococcal pericarditis became quite uncommon once penicillin was widely available, and in fact was not seen at all in the 529 episodes of pneumococcal bacteremia described by Austrian and Gold in 1964. Our cases, therefore, suggest that in HIV-seropositive patients, inadequate immune responses facilitate local spread of pneumococcal infection as well as bacteremia. In view of the rapidity with which tamponade evolved in our cases, we believe that frequent cardiovascular examination must be integrated into the care of HIV-seropositive patients with pneumococcal pneumonia or empyema.

**ACKNOWLEDGMENTS:** We thank Ms. Margaret Leonard for performing and retrieving the echocardiograms, Dr. Arnold Einhorn for interpreting the echocardiograms and Dr. Stephan Kamholz for numerous helpful suggestions.

**REFERENCES**


**Disseminated Mililiary Blastoymcosis Leading to Acute Respiratory Failure in an Urban Setting**

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