Sudden Onset of Large Pericardial Effusion in a 27-Year-Old Man With AIDS*

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A 27-year-old bisexual man with AIDS was admitted to the hospital because he had fever, dry cough, and mild shortness of breath. One month prior to admission, he had documented Pneumocystis carinii pneumonia and had received treatment with trimethoprim-sulfamethoxazole for 3 weeks.

Physical Examination

Vital signs: temperature, 39.4°C; pulse, 124/beats per minute; respirations, 20/min; and BP, 110/60 mm Hg. Positive finding on physical examination: seborrheic dermatitis on the face and thrush.

Laboratory Findings

Hematocrit, 35%; WBC count, 7,000/mm³, with 78% neutrophils, 2% band cells, 17% lymphocytes, and 3% monocytes; platelet count, 174,000/mm³; CD4 cell count, 22/mm³. Arterial blood gas levels (room air): pH, 7.48; PaCO₂, 30 mm Hg; PaO₂, 77 mm Hg. Lactate dehydrogenase, 293 IU/L; total protein, 6.9 g/dL. Chest radiograph: Figure 1.

Hospital Course

The patient was treated with IV trimethoprim-sulfamethoxazole and methylprednisolone for presumptive P carinii pneumonia with hypoxemia. He also received ketoconazole for thrush and zidovudine. Blood cultures were negative. Sputum smear: negative for acid-fast bacilli (AFB) and fungi. Sputum cytological studies: negative. On the 4th hospital day, the patient was tachypneic but hemodynamically stable, and a subsequent chest radiograph (Fig 2) demonstrated in addition to right paratracheal adenopathy, a markedly enlarged cardiac silhouette and blunting of the right costophrenic angle. On the 6th day of hospitalization, CT of the chest (Fig 3) revealed a large pericardial effusion, right paratracheal adenopathy, bilateral small pleural effusions, and areas of consolidation in the lower lobe of the left lung with air bronchograms. Methylprednisolone therapy was discontinued and diagnostic evaluation of the pericardial effusion was initiated. The echocardiogram showed a moderate to large anterior and posterior pericardial effusion with partial collapse of the right atrium during diastole. Left ventricular systolic function was preserved. Catheterization of the right side of the heart revealed normal hemodynamic values. On the 10th hospital day, a percutaneous pericardiocentesis was performed, and 1,000 mL of orange cloudy fluid was drained. Pericardial fluid: pH, 6.62; WBC count, 1,750/mm³ with 93% neutrophils and 7% lymphocytes; RBC count, 90,000/mm³; glucose level, 8 mg/dL; and protein value, 4.5 gm/dL. A 5F pigtail catheter was left in place for continuous drainage.

What is the cause for the sudden onset of a neutrophilic exudative pericardial effusion in this patient with AIDS?

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Diagnosis: Rapid dissemination of tuberculosis due to corticosteroid therapy and HIV infection; tuberculous pericardial effusion secondary to direct extension from tracheobronchial lymph nodes.

The route of spread of infection to the pericardium is most often from a contiguous focus of infection, usually mediastinal or hilar nodes, but could be from the lung, and less commonly as part of miliary tuberculosis (TB). The rate of pericardial fluid formation in TB varies depending upon the route of spread. Tuberculous pericardial effusions secondary to tracheobronchial lymph node erosion into the pericardial sac accumulate rapidly and tend to be large. Minimal pericardial fluid collection is common when the mechanism of spread is from hematogenous dissemination. Rapid accumulation of large pericardial effusions from TB has been reported to occur in HIV-seropositive patients.

Pleural effusion is common in patients with pericardial TB; it was reported in 39% of a large series of patients with pericardial effusion. Pleural effusion has been observed in 35% of HIV-positive patients and 25% of HIV-negative patients with tuberculous pericarditis. Tuberculous pericardial fluid is usually a lymphocytic exudate which may be bloody, blood-tinged, or purulent. Polymorphonuclear leukocyte predominance, as seen in the present patient, may occur in the early effusive phase of tuberculous pericarditis.

Corticosteroid treatment has been shown to cause progression of TB in humans and animals not being treated with appropriate chemotherapy. Accelerated progression of primary TB is common in persons coinfected with HIV. TB pericarditis, with rapid accumulation of large effusions, has been described as the initial infectious manifestation of AIDS.

Pericardial involvement is a life-threatening complication of TB and accounts for up to 4% of all cases of acute pericarditis and 7% of all cases of cardiac tamponade. TB has a mortality rate of less than 3% for all cases, while pericardial TB has a mortality rate of 14 to 40%. This is primarily due to difficulty in establishing the diagnosis and the adverse effects on the mechanical efficiency of the heart.

AFB smears of pericardial fluid in HIV-negative patients are almost invariably negative, but cultures are positive in 50 to 86% of cases. In contrast, the AFB smear and culture are positive in virtually 100% of patients with AIDS and tuberculous pericarditis. Pericardial biopsy showing histologic evidence of TB has been reported in 83 to 100% of HIV-negative patients. Pericardial fluid adenosine deaminase levels of 60 U/L or greater have a diagnostic sensitivity and specificity of close to 80%. In patients with unexplained culture-negative pericarditis, the use of DNA probes with amplification and hybridization of tuberculous DNA present in pericardial fluid or biopsy tissue may allow early diagnosis of mycobacterial infection.

For good therapeutic results, specific anti-TB therapy and adjunctive timely drainage is required. The use of corticosteroids as adjunctive therapy with anti-TB chemotherapy has been reported to improve survival, reduce the death rate, and lessen the need for another pericardiocentesis or open surgical drainage. Percutaneous pericardiocentesis with continuous catheter drainage remains the treatment of choice for patients with cardiac tamponade or large pericardial effusion demonstrated by echocardiography. Open surgical drainage at the time of admission eliminates the need for subsequent pericardiocentesis. The clinical outcome of surgical treatment is not significantly different from percutaneous pericardiocentesis since it does not reduce the need for pericardietomy for pericardial constriction. Pericardiectomy-associated surgical mortality is higher during the late calcific or chronic phase of constrictive pericarditis. Surgical resection of the pericardium is indicated in life-threatening tamponade, persistent elevation of central venous pressure unrelied by pericardiocentesis or catheter drainage.
and a nonresolving effusion.

In the present patient, the pericardial fluid AFB smear was 3+ positive; culture of the fluid grew *Mycobacterium tuberculosis*. Anti-TB chemotherapy with isoniazid, rifampin (rifampicin), pyrazinamide, and ethambutol hydrochloride was started. Prednisone, 40 mg/d, was added as adjunctive therapy and was continued for 4 weeks in tapered doses. The pericardial catheter was removed on the 2nd day. The patient’s temperature spikes decreased to low-grade fever within 48 h of initiation of chemotherapy and catheter drainage of the pericardial fluid. A follow-up chest radiograph after 3 weeks showed a normal cardiac silhouette and resolution of the paratracheal adenopathy and pleural effusions. Clinically, there was no evidence of constrictive pericarditis.

**Clinical Pearls**

1. Tuberculous pericardial effusion resulting from direct extension of tuberculous lymph nodes can be the initial infectious manifestation of AIDS.

2. Tuberculous pericardial effusion is usually a lymphocytic exudate; however, a polymorphonuclear predominant, hemorrhagic, smear positive fluid may be seen in the acute phase.

3. In AIDS patients, prior to adding corticosteroids as adjunctive therapy for established indications (eg, *P carinii pneumonia* with hypoxemia), concomitant TB needs to be excluded because corticosteroids can lead to rapid dissemination of TB with life-threatening complications.

4. Even with effective chemotherapy, pericardial TB continues to have a high mortality rate. The successful treatment of pericardial TB consists of early diagnosis, specific anti-TB therapy combined with adjunctive corticosteroid therapy, prompt symptomatic relief of a large effusion by catheter or open drainage procedure, and early pericardiectomy in selected cases.

**Suggested Readings**


Fowler NO. Tuberculous pericarditis. *JAMA* 1991; 266:99


Scott H, Beattie J. The distribution of tuberculous lesions in man and other primates with an account of the lymphatic glands and ves¬sels of the thorax and upper abdomen. *J Path Bact* 1928; 31:49-87
