An Asymptomatic Pleural Mass in a 72-Year-Old Man

Mark H. Gotfried, M.D., F.C.C.P.

(A Chest 1994; 106:572-74)

A 72-year-old man scheduled for cardiac surgery presented for evaluation of an intrathoracic mass noted on a preoperative chest roentgenogram. He denied chest pain, dyspnea, cough, weight loss, or fever. In the 1950s, while working above ground with coal, the patient had a routine chest roentgenogram that showed a left pleural mass. He remained asymptomatic, however, and the mass enlarged only minimally during long-term observation. Ten years ago, the patient underwent cardiac bypass surgery but now required revascularization because of unstable angina. The patient was an ex-smoker and denied exposure to asbestos or talc.

Physical Examination


Laboratory Findings

Hematocrit, 39 percent; WBC, 7,900/mm³; sodium, 138 mEq/L; potassium, 4.3 mEq/L; calcium, 96 mEq/L; bicarbonate, 26 mEq/L; BUN, 23 mg/dl; creatinine, 1.3 mg/dl; glucose, 87 mg/dl. ABG (room air): pH, 7.49; PCO₂, 37 mm Hg; PO₂, 70 mm Hg. Chest roentgenogram: shown above (Fig 1).

What is the most likely cause of the chronic pleural mass?

*From the University of Arizona and Vencor Hospital, Phoenix.*
Diagnosis: Chronic tuberculous empyema

Tuberculous involvement of the pleural space is a well-known clinical entity reported to occur in up to 5 percent of all patients with tuberculosis. Pleural effusions appear to develop in this condition when a subpleural focus of caseous necrosis ruptures into the pleural space and initiates a hypersensitivity reaction. Resulting pleural inflammation causes the patient to present with a subacute onset of chest pain and fever often associated with fatigue and varying degrees of respiratory symptoms. On occasion, an acute presentation may simulate a bacterial pneumonia with a parapneumonic effusion. Occurring most commonly in patients 40 to 50 years of age, pleural tuberculosis has been reported in all age groups.

Virtually always unilateral in location, tuberculous pleural effusions are exudates (pleural fluid protein >4 g/dl) with a predominance of lymphocytes (usually 90 to 95 percent lymphocytes). A distinctively characteristic feature is a paucity of mesothelial cells—mesothelial cell counts greater than 5 percent of nucleated cells serve to nearly exclude the clinical diagnosis. Pleural fluid eosinophilia greater than 10 percent of nucleated cells is also an unusual finding in pleural tuberculosis. Pleural fluid glucose concentrations may be below 60 mg/dl in 20 percent of patients. Additionally, a pleural fluid pH below 7.20 is found in 20 percent of patients, and the pleural fluid pH is rarely greater than 7.40.

Acid-fast smears of the pleural fluid are rarely positive because tubercle bacilli exist in low concentrations within the pleural space in this condition. Although pleural fluid cultures may be positive in approximately 30 percent of patients (reported range 20 to 70 percent), histologic examination and culture of pleural biopsy specimens greatly enhance the diagnostic yield. Up to 95 percent of patients with tuberculous pleurisy can be diagnosed by a combined approach that utilizes thoracentesis and pleural biopsy. A negative PPD skin reaction is seen in up to 30 percent of patients at clinical presentation; most patients will convert their skin tests to positive within 6 to 8 weeks. The initial absence of reactivity may be due to the presence of peripheral mononuclear suppressor cells.

Thoracoscopic-assisted pleural biopsy can further contribute to diagnosis when evaluation with thoracentesis and pleural biopsy is unrevealing. Patients who present with a positive PPD and an undiagnosed lymphocyte-predominate exudative pleural effusion, however, should receive antituberculosis therapy because of the high clinical likelihood of pleural tuberculosis. A response to therapy does not prove the diagnosis because most patients with tuberculous pleural effusions recover after several months with or without treatment.

Most patients with tuberculous pleural effusions follow a self-limited course and resolve their pleural effusions with or without antituberculosis therapy. A failure to diagnose and treat, however, creates a high risk for reactivation in the form of pulmonary tuberculosis (70 percent within 5 years). In the prechemotherapeutic era, clinical resolution of tuberculous pleurisy occurred with minimal morbidity, leaving patients with radiographically normal pleural contours or minor degrees of residual pleural thickening in 50 percent of instances. Rarely, however, untreated patients developed an active pleural infection that would progress to a chronic tuberculous empyema. The chest roentgenogram, in such instances, revealed pleural thickening with the eventual development of pleural calcification that represented the thick fibrocalkific rind of a chronic empyema.

In some patients with chronic tuberculous empyemas, the inflammatory process may exist for many years with a paucity of clinical symptoms. A prolonged asymptomatic course is attributable to the markedly thickened pleura that effectively confines the bacilli to the pleural space. In other patients, the empyema may progress to cause a bronchopleural fistula as a late clinical complication. The onset of bronchopleural fistulae not only initiates pulmonary symptoms in the patient but increases the risk of spreading the disease to contacts by contaminating respiratory secretions with acid-fast bacilli. The development of empyema necessitatis that presents as an anterior chest wall mass has also been reported.

The markedly thickened pleura surrounding the cavity of a tuberculous empyema may complicate drug therapy. The empyema rind presents a barrier to intrapleural penetration of drugs. The resulting potential for inadequate drug concentrations in regions of actively dividing tuberculous bacteria promotes the emergence of resistant pathogens. Although drug concentrations have not been measured within the contents of a tuberculous empyema, reports exist of treatment failures and conversion to resistant strains in patients treated with nominally adequate chemotherapeutic regimens. These observations suggest the importance of initiating therapy with aggressive, four-drug regimens with close monitoring of clinical response in patients with tuberculous empyemas.

The method for selecting patients with chronic tuberculous empyemas for drainage and decortication is not entirely clear. The risks of incomplete drug penetration and failure of initial therapy warrant considerations of empyemectomy in patients with adequate ventilatory reserve. Patients who fail initial therapy most certainly should be evaluated for surgery. Furthermore, medical therapy alone will not
allow resolution of the thick pleural rind in patients who present with respiratory impairment due to a trapped lung.

The present patient required urgent coronary artery revascularization, so the pleural mass was evaluated intraoperatively. An incision through the thick fibrous wall of the mass expressed odorless, viscour yellow fluid that grew *Mycobacterium tuberculosis* sensitive to all first line antituberculosis agents. After cardiac surgery, the patient was started on a regimen of isoniazid, rifampin, pyrazinamide, and ethambutol but discontinued therapy because of nausea and refused further surgical or medical treatment.

**Clinical Pearls**

1. *Tuberculous pleuritis* occurs in approximately 5 percent of patients with active tuberculosis and usually follows a self-limited course. After treatment with antituberculosis chemotherapy, residual pleural thickening results in 50 percent of patients.

2. *Chronic tuberculous empyema* occasionally develops as a late manifestation of untreated or poorly treated tuberculosis and may exist for many years without causing symptoms. Routine chest roentgenograms or the development of bronchopleural fistulae usually bring the patient to clinical attention.

3. Incomplete penetration of antituberculosis drugs into the empyema space has the theoretic potential for promoting resistant strains of tuberculous organisms and causing treatment failures. Aggressive drug regimens with four agents combined with considerations for surgical drainage are important therapeutic principles in patients with tuberculous empyemas.

**Suggested Reading**

Iseman MD, Madsen LA. Chronic tuberculous empyema with bronchopleural fistula resulting in treatment failure and progressive drug resistance. Chest 1991; 100:124-27
