A chest wall mass in a 73-year-old man

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A 73-year-old man presented to the emergency department with a productive cough, shortness of breath, and an enlarging chest wall mass 2 months after the completion of antituberculous therapy. He had a cough productive of brown sputum. He denied fever, chills, weight loss, hemoptysis, and night sweats. His medical history was notable for hypertension and also for Mycobacterium tuberculosis (MTB) infection that had been diagnosed by sputum culture with a compatible chest radiograph 14 months prior to admission. At the time of diagnosis, the patient had a pleural effusion, but he refused to undergo thoracocentesis. The MTB isolate was sensitive to all drugs, and the patient was treated using direct observed therapy with isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months and with isoniazid and rifampin for 11 more months. Two months prior to stopping therapy, he underwent a diagnostic thoracocentesis, which did not reveal tuberculosis.

The patient's physical examination revealed a thin man in no distress with a BP of 145/102 mm Hg, a pulse rate of 92 beats/min, and a respiratory rate of 20 breaths/min. He had no fever. A chest examination showed a right-sided anterior 12 × 10-cm fluctuant chest mass. His right lung had decreased breath sounds with crackles throughout and dullness to percussion at the base. A left lung examination revealed crackles. The patient had clubbing of his fingers and toes. An evaluation of laboratory tests revealed a normochromic normocytic anemia with a WBC count of 9,600 cells/mL and sterile pyuria. The results of an HIV enzyme-linked immunosorbent assay were negative.

The patient’s chest radiograph (Fig 1) and a CT scan (Fig 2) are shown.

What is the diagnosis?

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Diagnosis: Empyema necessitatis from MTB empyema

Discussion

Pleural effusions occur in approximately 5% of patients with MTB infections. The vast majority are due to tuberculous pleuritis, which is a hypersensitivity phenomenon. The remainder of pleural effusions due to MTB are tuberculous empyemas, which represent active infection of the pleural space.1–3

Tuberculous pleuritis results from an exaggerated inflammatory response to a localized bacillary pleural infection. It results from a ruptured subpleural caseous focus into the pleural space.1,2 The mycobacterial antigen in the pleural space leads to interaction with the T cells that were previously sensitized to MTB. This delayed hypersensitivity reaction leads to increased capillary permeability, which leads to fluid formation. It is the delayed hypersensitivity reaction rather than the actual infection of the pleural cavity with tuberculous bacteria that leads to the pleural fluid formation, resulting in a high negative culture rate. Fluid is typically serous and is never bloody. The tuberculous pleuritis usually resolves within 1 to 4 months with or without chemotherapy and rarely leads to tuberculous empyema.

Tuberculous empyema is a much less common complication of tuberculosis. It represents chronic active infection. The infection can be an extension of pulmonary tuberculosis, thoracic adenopathy, or hematogenous spread. It is usually a large effusion that may lead to an entrapped lung. Clinical symptoms may be absent until the patient develops a broncho-pleural fistula or empyema necessitatis.1–3

The hallmark of empyema necessitatis is a mass, usually on the anterolateral chest, but also occasionally on the posterior trunk or the abdominal wall. It is the consequence of burrowing pus. Drainage sites include the pericardium, vertebral column, esophagus, or breast, or through the diaphragm into the abdominal cavity or retroperitoneum. Masses also have been described in the flank, back, groin, thigh, or psoas muscle, or perianally.4–6 A medical history might reveal a previous case of active tuberculosis that was either several decades old, when many of our currently available antituberculous medications were not available, or had been insufficiently treated, or tuberculosis with the development of drug resistance secondary to low drug penetrance into the pleural cavity.7,8 The mass might be painful and fluctuant. Empyema necessitatis due to MTB typically lacks heat or redness on the protruding mass (cold abscess). Pleuritic chest pain, nonproductive cough, low-grade fevers, night sweats, and weight loss may or may not be present.

The chest radiograph reveals a pleural effusion, with thickening of the pleura at the involved site. There may be an enlargement of the overlying ribs with a thick, calcified pleural rind. A CT scan may show a thick-walled, possibly well-encapsulated, pleural mass that protrudes through the chest wall or into the abdominal wall, which is virtually diagnostic of empyema necessitatis. The actual fistulous tract from the pleural cavity may be visualized,6 but more often it is not because of the small size of the tract.2

In tuberculous empyema necessitatis, the fluid will be grossly purulent. The pleural fluid WBC count is often > 100,000 cells/mL with a neutrophil predominance. The fluid pH is typically < 7.2, the fluid glucose is often < 20 mg/dL, and the fluid protein can be > 5 g/dL. The pleural fluid lactate dehydrogenase level can be > 1,000 U/L. The performance of a culture is obligatory to rule out other forms of infection or coinfections. MTB will grow in only 10 to 47% of cases.2

The treatment, drainage and proper antituberculous medications, must be individualized.

References
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