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CORRESPONDENCE

Empyema necessitatis due to *Mycobacterium tuberculosis*



To the Editor,

Empyema necessitatis (EN) is characterized by the emission of pus from the pleural cavity into adjacent anatomical structures such as the chest wall, the esophagus, the pericardium, and the mediastinum.¹ In 1940, Sindel reported that the majority of cases of EN were caused by *Mycobacterium tuberculosis* (73%), followed by *Streptococcus pneumoniae* and *Actinomyces* spp. (especially *Actinomyces israelii*).¹ The overall mortality rate was 66% (87% for *M. tuberculosis*, 28% for pyogenic organisms) in that era.¹ Although new cases of EN caused by *M. tuberculosis* have been reported in a few countries in recent years,^{1–3} no case of EN due to *M. tuberculosis* has been reported in Taiwan, a country with an intermediate burden of tuberculosis.²

Herein, we report an 89-year-old bedridden man with a history of cerebrovascular accident and tracheostomy. In April 2013, the patient presented to the emergency department with a 12-day history of dyspnea. Physical examination revealed a malnourished individual in a vegetative state, with a nasogastric tube. Left-side chest auscultation sounds were diminished. In addition, a non-erythematous, fluctuant, nontender, subcutaneous mass measuring 12 cm × 9 cm in diameter was found extending from the left fifth to ninth anterolateral intercostal space. Chest radiography showed prominent infiltrates in both lung fields and a blurred left-side costophrenic angle. Chest sonography showed loculated, thick pleural fluid in the left lung with low-density echogenic material in the mass of the left anterolateral chest wall. Fine-needle aspiration of fluidlike material obtained 12 mL of creamy, malodorous, thick pus. Acid-fast staining of the pus revealed numerous acid-fast bacilli, although bacterial culture did not grow significant pathogens. The acid-fast stain of sputum was also positive for acid-fast bacilli, although bacterial culture of sputum did not grow any pyogenic pathogen. Chest computed tomography revealed a loculated thick-walled pleural collection of low attenuation, and direct extension

to the left chest wall (Fig. 1). A chest tube was inserted into the left side of the chest and 1100 mL of purulent fluid was drained. Many acid-fast bacilli were also found in the drained fluid. Cultures of the pus, sputum, and drained fluid were all positive for *M. tuberculosis* but negative for other aerobic/anaerobic bacteria or fungi. The *M. tuberculosis* isolates were susceptible to isoniazid (0.2 µg/mL and 1.0 µg/mL), rifampicin (1.0 µg/mL), ethambutol (5.0 µg/mL), streptomycin (2.0 µg/mL), capreomycin (10.0 µg/mL), ofloxacin (2.0 µg/mL), levofloxacin (1.0 µg/mL), ethionamide (10.0 µg/mL), and para-aminosalicylic acid (8.0 µg/mL) by using modified proportional disk elution methods. Antituberculosis agents were initiated on hospital Day 3. The patient recovered well and was discharged at Day 28 of hospitalization. The patient was treated successfully with isoniazid, rifampicin, ethambutol, and pyrazinamide for the initial 2 months, followed by isoniazid and rifampicin for an additional 4 months.

M. tuberculosis accounts for only 10% of all cases of pleural empyema.³ By contrast, 73% of cases of EN were attributable to *M. tuberculosis* in the preantibiotic era.¹ The chronic process of EN can be explained by the fact that its common microbe etiologies include *M. tuberculosis* and bacterial species of low virulence (*Actinomyces* spp., streptococci, obligative anaerobes, etc).^{1,4} Despite the availability of effective antibiotics, *M. tuberculosis* is responsible for 35–50% of cases of EN.³ Recent reviews, however, have shown that the mortality rate among patients with EN is less than 5%.^{1,3}

Significant costal bony destruction in EN is most often due to *Actinomyces* spp.^{1,5} or *Aspergillus* spp.¹ However, our patient did not have risk factors for acquiring invasive pulmonary aspergillosis (cavitary lung lesions, use of steroid with equivalent dosage of prednisolone ≥15 mg per day for >3 weeks, or prolonged neutropenia after chemotherapy). Finally, according to the observation reported by Sindel,¹ EN tends to rupture at the upper anterior portion of the chest wall because the lung parenchyma is adherent



Figure 1. Computed tomography scan of the chest with contrast enhancement demonstrates a large, loculated, thick-walled left pleural empyema with communication of empyema to the left anterolateral chest subcutaneous tissue. A large, centrally located, fluid-filled chest wall lesion is also visible.

posteriorly at the apex and base. This observation is consistent with the findings in our patient.

In conclusion, we remind readers that EN should be considered in patients with a fluctuant mass over the anterolateral chest wall and ipsilaterally significant pleural effusion.

Conflicts of interest

All authors declare no conflicts of interest.

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