CASE REPORT

Thoracic empyema and bacteremia due to Mycobacterium abscessus in a patient with liver cirrhosis

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Received 17 April 2013; accepted 21 May 2013

KEYWORDS
Bacteremia;
Empyema thoracis;
Liver cirrhosis;
Mycobacterium abscessus

Introduction

Nontuberculous mycobacteria (NTM) are ubiquitous in the environment and, although most NTM species cause clinically insignificant colonization, a few species have been implicated in an increasing number of human diseases around the world.1–7 The clinical manifestations of NTM disease are heterogeneous and include pneumonia, lymphadenitis, skin/soft tissue infection, disseminated infection, central nervous systemic infection, and genitourinary infections.7–12 Empyema thoracis caused by NTM is rare.13,14 Herein, we report a case of Mycobacterium abscessus empyema and mycobacteremia in a patient with liver cirrhosis.

Case report

A 44-year-old man with diabetes mellitus, alcohol-related liver cirrhosis, and hepatocellular carcinoma presented to
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the emergency department with a 1-week history of productive cough, fever, and progressive dyspnea. He denied any accompanying symptoms such as chest pain, nausea, vomiting, diarrhea, or dysuria. After initial workup, he was admitted under the diagnosis of pneumonia with parapneumonic pleural effusion. His sputum culture yielded *Klebsiella pneumoniae*. Ertapenem (1 g every 24 hours) was administered, and the patient’s fever and dyspnea gradually subsided. During hospitalization, however, reaccumulation of fluid in the right pleural cavity and relapse of dyspnea were noted. A pigtail catheter was used to drain the pleural effusion, and laboratory examination of the pleural fluid revealed transudative characteristics indicative of hepatic hydrothorax. On hospital Day 30, the patient was dyspneic with fever (38.2°C). His pulse rate was 122 beats per minute, respiratory rate was 22 breaths per minute, and blood pressure was 134/83 mmHg. The only notable finding on physical examination was decreased breathing sounds in the right lung field. Laboratory examinations revealed the following values: white blood cell count, 16,600/mm³ (85% neutrophils); total bilirubin, 2.61 mg/dL; albumin, 2.8 g/dL; creatinine, 2.0 mg/dL; and C-reactive protein, 52.4 mg/L (reference range < 6 mg/L). Chest radiograph showed massive pleural effusion in the right lung (Fig. 1). Abdominal sonography revealed liver cirrhosis, a hepatic nodule, a moderate amount of ascites fluid, and right-sided pleural effusion. Analysis of pleural fluid revealed the following values: pH, 8.0; lactate dehydrogenase, 106 IU/L (serum level, 162 IU/L); total protein, 0.6 g/dL (serum level, 5.7 g/dL); and white blood cell count, 1069/mm³ (91% neutrophils). The result of Gram staining was negative and that of acid-fast staining was positive. Paracentesis was performed, and analysis of ascites fluid revealed a white blood cell count of 288/mm³ (73% neutrophils) and negative results for Gram or acid-fast staining. Ciprofloxacin (400 mg every 12 hours) was administered after cultures of blood, pleural fluid, and ascites fluid had been collected. On hospital Day 45, cultures of two sets of blood samples and pleural effusion yielded acid-fast bacilli; however, the culture of ascites fluid did not yield any pathogen. Therefore, antimycobacterial agents comprising isoniazid, rifampin, ethambutal, and clarithromycin were administered. However, his clinical condition did not improve and repeated culture of pleural effusion still grew acid-fast bacilli. Sixty days after admission, the isolate was identified as *M. abscessus* by conventional methods. Antibiotics were switched to imipenem (500 mg every 6 hours), amikacin (250 mg daily), and clarithromycin (500 mg every 12 hours). Thereafter, the volume of fluid drained from the pigtail catheter gradually decreased and the patient’s clinical condition improved. Repeated culture of pleural fluid and blood did not yield any mycobacteria. However, the patient died due to another episode of healthcare-associated pneumonia 2 months later. During the course of the disease, *M. abscessus* was not detected in sputum or other specimens.

Discussion

The clinical significance of NTM isolates from pleural effusion remains unclear. In a study by Shu et al., of 49 patients with pleurisy had pleural effusion cultures positive for NTM, and the most common NTM causing pleurisy was *Mycobacterium avium* complex, followed by *Mycobacterium fortuitum*, *Mycobacterium kansasii*, and *Mycobacterium chelonae*. Empyema thoracis caused by *M. abscessus* has rarely been reported in the English literature. In our patient, *M. abscessus* was confirmed to be the causative agent of thoracic empyema based on the isolation of *M. abscessus* from cultures of sterile pleural effusion and blood. Taiwan is endemic for tuberculosis, and most cases of mycobacterial pleurisy are caused by *Mycobacterium tuberculosis*. However, our case demonstrates that pleurisy can be caused by NTM species, particularly *M. abscessus*.

In our patient, *M. abscessus* was not isolated from sputum and the lung parenchyma appeared normal, indicating that pneumonia-related empyema due to this organism was unlikely. Infection of the pleural cavity in our patient might have occurred as a result of the direct translocation of mycobacterium from the peritoneal cavity or as a result of transient mycobacteremia that infected the pleural space. We suspect that our patient’s immunocompromised condition due to liver cirrhosis contributed to the development of NTM empyema thoracis and mycobacterial bacteremia.

NTM pleurisy is associated with a high 1-year mortality rate, although anti-NTM therapy may provide >1-year survival. The outcome in our patient was poor even though he initially responded well to anti-NTM therapy and drainage. The underlying comorbidities in our patient are a possible explanation for the poor outcome.

In conclusion, *M. abscessus* should be considered as a possible cause of empyema thoracis and bacteremia in patients with liver cirrhosis.

Conflicts of interest

All authors declare no conflicts of interest. No financial support was received for this study.

References


