Case report on pulmonary disease due to coinfection of *Mycobacterium tuberculosis* and *Mycobacterium abscessus*: Difficulty in diagnosis

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**Abstract**

*Mycobacterium abscessus*, which is ubiquitous environmental organism, is more likely to cause pulmonary infection in the presence underlying lung disease and immunosuppression. We report a case of pulmonary disease due to coinfection of *Mycobacterium tuberculosis* (MTB) and *Mycobacterium abscessus* (*M. abscessus*) in an immunocompetent patient without underlying lung disease.

Healthcare professionals should be aware of co-infection with MTB and *M. abscessus*, and treatment should be based on clinical suspicion and/or epidemiological circumstances.

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1. Case presentation

A 77-year-old Vietnamese male presented with a one-week history of hemoptysis. He had had a progressive cough for 6 months for which he was seen by his primary care physician 5 months prior to admission. Chest X-ray (CXR) done at the time revealed right middle lobe infiltrate. He was treated with antibiotics without improvement. Two months prior to admission, he started experiencing night sweats, low grade intermittent fever, fatigue, loss of appetite and 4 pound weight loss.

The patient was a chronic hepatitis B carrier but was not on treatment. There was no history of chronic pulmonary disease, pulmonary tuberculosis (PTB) or contact with PTB, HIV test was negative. He was a Vietnamese prisoner of war who migrated to United States of America 21 years previously. He lived with his son and two grandchildren. There was no history of cigarette smoking.

On examination, he was cachectic with a temperature 98.7 F, heart rate 81/minute and regular, respiratory rate 21/minute, blood pressure 120/82 mmHg, oxygen saturation 95% in room air. Chest examination was remarkable for right upper lung bronchial breath sounds.

The leukocyte count was 8400/µL (normal range 4800–10800/µL) with normal differentials, and hemoglobin concentration was 11.8g/dl (normal range 14–18g/dl). A comprehensive metabolic profile was within normal limits.

A chest radiograph revealed extensive right upper lobe destruction and fibrotic atelectasis (Fig. 1) while a CT scan of the chest with intravenous contrast showed pulmonary parenchymal fibrosis and chronic cystic bronchiectasis of the right upper lobe with consequent superior migration of the right horizontal fissure and rightward mediastinal shift (Fig. 2). Acid fast bacilli (AFB) were detected in the expectorated sputum. The patient was treated with RIPE (Rifampicin, isoniazid, pyrazinamide and ethambutol).

Although the initial amplified mycobacterium tuberculosis direct (MTD) test was negative, RIPE therapy was continued because of high clinical suspicion for PTB. Despite RIPE therapy, sputum AFB smear continued to be positive with all broth medium mycobacteria growth indicator tube (MGIT) cultures growing in 7–10 days. Subsequently, DNA PCR detected *M. abscessus* but did not detect *Mycobacterium avium-intracellulare* (*MAI*).

RIPE therapy and airborne isolation were discontinued on day 14; and treatment with amikacin, cefoxitin and meropenem was started. MTD test was repeated and positive on day 19. RIPE therapy was restarted and airborne isolation was reinstated while continuing the treatment for *M. abscessus*. Five weeks later, sputum culture from day 19 grew MTB in addition to *M. abscessus*.

Expectorated sputum samples became negative for AFB in the 5 week of admission and patient was subsequently discharged home to continue treatment for both MTB and *M. abscessus*. The patient’s son and grandchildren were diagnosed with latent TB infection and were currently being treated.

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**2. Discussion**

*M. abscessus*, the most clinical significant rapidly growing mycobacterium (RGM) found abundantly in the environment worldwide, is more likely to cause pulmonary infection immunocompromised patients and people with underlying lung disease [1]. *M. abscessus* can also cause extrapulmonary infections; lymphadenitis, skin and soft tissue infection, musculoskeletal infection, prosthetic device infection, surgical site infection and catheter-related infections [2]. Pulmonary disease accounts for about 50% of cases [3].

A case was reported of *M. abscessus* lung disease in a patient whose lung disease progressed after lobectomy and medical treatment of PTB [4]. It becomes more clinically challenging to differentiate PTB from NTM pulmonary infection especially for patients with epidemiological predisposition [4]. In our case, we faced the challenges associated with the diagnosis of the coexistence of both infection in a patient with epidemiological predisposition to pulmonary tuberculosis.

The clinical features of PTB and pulmonary infection due to NTM like *M. abscessus* are similar. However, it is pertinent to differentiate both disease entities because the treatments are different. Moreover, PTB is of public health importance. The radiographic findings can also be similar. In about 40% of cases of pulmonary infection due to NTM, the chest radiograph shows an interstitial pattern, mixed interstitial and alveolar infiltrates, and a reticulonodular pattern as well as multilobar involvement in about 50% of cases [2]. The most commonest radiologic feature of pulmonary infection due to *M. abscessus* is nodular bronchiectasis [5].

Based on the patient’s country of origin, history of having been a prisoner of war, clinical presentation and the findings of investigations, he was started on therapy for presumed PTB which was continued despite a negative MTD. RIPE therapy was discontinued only when culture showed of *M. abscessus*. Although *M. abscessus* usually cause pulmonary disease in immunocompromised patients and patients with underlying lung disease, Varghese, and his colleagues reported pulmonary infection due to *M. abscessus* in immunocompetent patients without underlying lung disease [6].

The positive predictive value of nucleic acid amplification test (NAAT) like MDT in AFB positive specimen is 95% [7]. It is recommended that test for inhibitors (seen in 3–7% sputum specimen) should be performed and an additional specimen should be tested with NAAT if the first specimen from an AFB positive specimen is negative on MDT test [8]. Even if the second specimen is negative, the decision to treat for PTB should be based on clinical suspicion. Retrospectively, the RIPE therapy in this case should not have been discontinued considering the clinical and epidemiological circumstances. With treatment of both MTB and *M. abscessus*, sputum AFB became negative after one week of combined therapy and patient clinically improved.

*M. abscessus*, like other RGM, are susceptible to antibacterial agents but resistant to the antituberculosis agents. The *M. abscessus* in this case was susceptible to amikacin, cefoxitin and azithromycin and did not demonstrate the inducible macrolide resistance gene or *erm* gene. The patient was discharged home when his sputum AFB became negative and we are expecting a prolonged period of treatment.

### 3. Conclusion

We describe the case of a patient with pulmonary disease due to co-infection with MTB and *M. abscessus*. The difficulties we encountered in diagnosis and treatment made it pertinent for clinicians to be aware of dual infection. Treatment of PTB should not be delayed or discontinued solely because of negative test results, and treatment should be based on clinical suspicion and/or epidemiological circumstances. This also points to the need for consideration of NAA for NTM in the presence risk factors and progression of pulmonary disease despite antituberculosis therapy.

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