



A case of an immunocompetent young man obtaining community-acquired disseminated *Nocardia brasiliensis*

Jinna Li^a, Jie Cao^{a,*}, Yueqing Wu^a, Nansheng Wan^a, Li Pan^b, Yuanbao Chen^a

^a Respiratory Department, Tianjin Medical University General Hospital, Tianjin 300052, China

^b Respiratory Department, Fourth Center Hospital, Tianjin 300142, China

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ABSTRACT

Nocardiosis is a rare but severe pyogenic or granulomatous disease and caused by *Nocardia* that mainly infects immunocompromised patients. We report here a case of an immunocompetent 24-year-old male student with community-acquired pneumonia with asymptomatic disseminated cerebral abscess by *Brasiliensis nocardiosis*. The patient was fully recovered after receiving optimized antimicrobial therapy without relapse. This case suggests the health professionals such as the physicians of pulmonary, infection, neurology department and et al should always think about unusual cause of community acquired pneumonia, even in immunocompetent patients and when having pulmonary nocardiosis we should do a radiological neurological work up, even with the absence of neurological finding or symptom.

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Introduction

Nocardiosis is an uncommon infection affecting mainly immunocompromised patients. We herein present a case of an immunocompetent young man infected with community-acquired pneumonia with asymptomatic disseminated cerebral abscess by *Brasiliensis nocardiosis*.

Case report

A 24-year-old male student was admitted into our hospital with coughing for more than 2 months and intermittent fever for more than 1 month. More than 2 months before the admission, he began to have whooping cough with a small amount of sticky and white sputum, and had needle-like pain on the left back, without apparent reason. He did not have fever, headache, nasal congestion, throat soreness, chest pain, night sweat, nausea, vomiting nor rash. Some medications were taken by himself (details unknown), but no significant relief of the symptoms was achieved. About one month ago, his body temperature slowly increased from 37.5 °C to 40 °C with chills but no shivers. Right after mild exercises, he had intense

cough, vomiting small amount of yellow sputum that occasionally had traces of blood in it. The patient had intermittent fever even after anti-inflammatory therapy. His spirit, sleep and appetite were very poor.

The patient had no history of hypertension, diabetes mellitus, hepatitis, tuberculosis, trauma, surgery, blood transfusion and was not allergy to medication or foods. Besides, he had no history of alcoholism, cigarette smoking or drug abuse. He was unmarried and had no history of sexual intercourse. His family members were healthy without hypertension, diabetes mellitus, cerebral-vascular diseases or other hereditary diseases. At the time of admission, his body temperature was 39.6 °C with 106 bpm pulse, 22 bpm respiratory rate and normal blood pressure.

He was normal personal with medium alimentation and acute illness facial features with normal consciousness. No jaundice, petechia or enlarged superficial lymph nodes was noted. His neck was soft with no resistance with symmetry chest without no pressing pain on his chest wall. His respiratory sounds were low and some moist rales could be heard in the lower left lung. Heart rate was 106 bpm with regular rhythm. The physical examination indicated that his abdomen and nervous system were normal.

Chest computed tomography (CT) showed that he had a consolidation in the left inferior lobe (Fig. 1). The blood test showed that his white blood cell (WBC) count was 17.1×10^9 , Na was 74.6%, L was 15.4%, M was 9.5%, C-reactive protein (CRP) was 16.00 mg/dl (<0.8 mg/dl), Porphyria cutanea tarda (PCT) was <0.5 ng/ml. He was negative for G-test, Legionella, immunoglobulins, ANA, dsDNA,

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* Corresponding author. Tel.: +86 131 320 88076; fax: +86 22 60361632.

E-mail address: tjcaojie@sina.com (J. Cao).



Fig. 1. Enhanced CT scans of the chest prior to antibiotic treatment showing consolidation of left inferior lobe.

anti-SmAb, anti-SSA, anti-SSB, anti-Scl-70, blood culture and HIV. T-cell subset distribution was found normal. Bronchoscopy showed that there was stenosis at the left inferior lobe dorsal section and posterior base section with widen carina and hypertrophied mucous membranes containing large amount of sticky, purulent exudate, especially at the dorsal section. Bronchial alveolar lavage was performed for the left inferior dorsal section. In the bronchoalveolar lavage fluid (BALF), 62.5% was neutrocytes and 33.5% was lymphocytes without eosinophil. When Gram+ *Bacillus filiformis* was noted on the sputum and BALF smears (Figs. 2 and 3), he was immediately suspected of Nocardia infection. The doctor was called and the culture time was extended to confirm the speculation. Chronic inflammation of mucous membrane was detected in the biopsy of the left inferior lobe dorsal segment. Bacteriology tests by two cultures of sputum and BALF samples finally confirmed that the pathogen was *B. nocardiosis* in both samples. Since *B. nocardiosis* is easy to disseminate, cranial CT was performed on the patient even if he did not have headache, vomiting or other neural symptom. Low-density shadow could be seen on the left temporal lobe from the CT image (Fig. 4). Two lesions were found by the brain MRI on the left temporal and right frontal lobes (Figs. 5

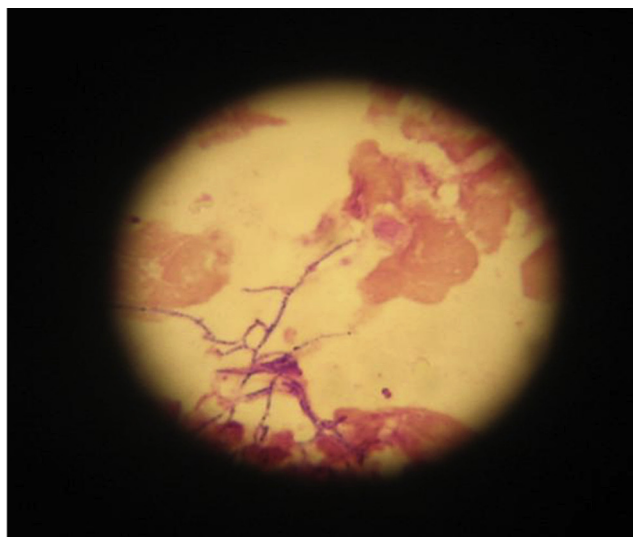


Fig. 2. Sputum smear showing Gram+ *Bacillus filiformis*.



Fig. 3. BALF smear showing Gram+ *Bacillus filiformis*.

and 6), and they were considered to be cerebral abscesses. Lumbar puncture was performed. The cerebrospinal fluid (CSF) was clear and the pressure was 125 mm H₂O. Indian ink staining was negative and no bacterium nor Nocardia was found in the smear and culture. The pathology test did not find any malignant cell. The patient was finally diagnosed pulmonary infection of *B. nocardiosis* complicated with cerebral abscess, and treated with intravenous injection of 0.2 g linezolid every 12 h and 1 g meropenem every 8 h alternatively for 4 months in total (because there was obvious decrease in WBC and PLT counts when the patient was treated with linezolid continuously for a week. So, he was switched to meropenem till the counts became normal). The injection was combined with oral administration of 2 g SMZCo three times a day for 12 months. The chest CT (Fig. 7) and cranial MRI (Figs. 8 and 9), taken 1, 2 and 12 months after the completion of the therapy showed a complete absence of the lesions observed previously.

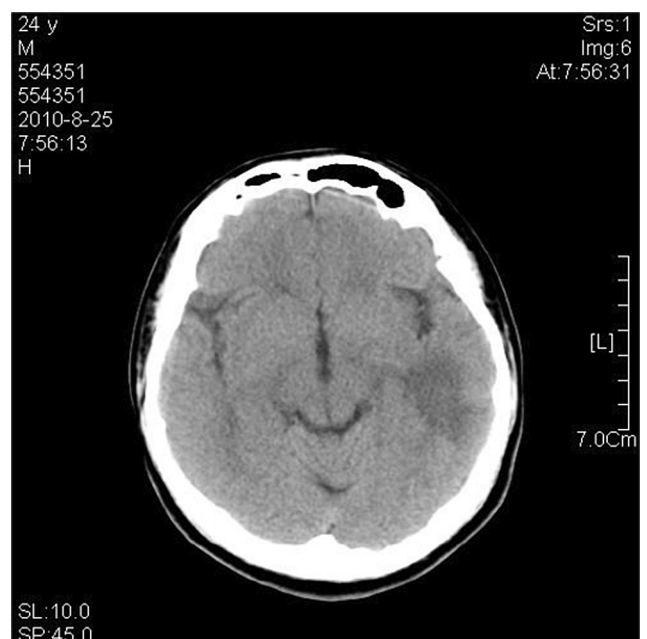


Fig. 4. Cranial CT prior to antibiotic treatment showing low-density shadow in the left temporal lobe.

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