

Endobronchial Pulmonary Nocardiosis

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Abstract

In recent years, the reported incidence of nocardiosis, a localized or disseminated infection caused by *Nocardia*, has been increasing. This increase can be attributed to the improvements in diagnostic capabilities and the higher clinical index of suspicion, as well as the increased prevalence of immunosuppressed patients.

We report a case of pulmonary nocardiosis in a healthy young female who presented with cough and hemoptysis, which persisted even after empiric treatment for pulmonary tuberculosis. CT scan of her chest showed infiltrates in the right upper, middle and lower lobes with pleural effusion. Bronchoscopy revealed a friable endobronchial mass. Special stains confirmed the diagnosis of nocardiosis. The patient's symptoms and clinical findings improved with trimethoprim-sulfamethoxazole. To the best of our knowledge, this is the fourth case of this illness presenting as endobronchial mass reported in the literature. Pulmonary nocardiosis should be considered in the differential diagnosis of hemoptysis, even in immunocompetent patients.

Key Words: Nocardiosis, endobronchial mass, immunocompetence.

PULMONARY NOCARDIOSIS is a rare infection; however, its reported incidence appears to be increasing. This is probably due to a higher degree of clinical suspicion, increased use of immunosuppressive treatments and the appearance of AIDS (1, 2). The disease presents with a highly variable clinical and roentgenographic picture that can mimic granulomatous disease or neoplasm (3–5). Thus, it is often misdiagnosed as tuberculosis, invasive fungal infection or cancer. Although nocardiosis usually occurs in immunocompromised hosts, it has also been occasionally reported in immunocompetent patients (6, 7). A review of the literature reveals only three previously reported cases of pulmonary nocardial infection presenting with an associated endobronchial mass, as in the case reported here.

This article describes the clinical course of pulmonary nocardiosis in a healthy young female,

who was initially misdiagnosed as having pulmonary tuberculosis.

Case Report

A 25-year-old female Hispanic immigrant was referred to our clinic for evaluation of persistent cough and hemoptysis. The patient reported a 5-year history of persistent cough and pleuritic chest pain. The cough was usually productive of greenish sputum, but occasionally frank hemoptysis was noted, including two episodes of coughing up approximately 250 mL of blood. Initially the patient continued normal activities and denied any fatigue or dyspnea, but she subsequently developed worsening dyspnea on exertion. She denied weight loss, joint pain or fever. Hospitalized multiple times for treatment of recurrent pneumonia, she had repeatedly tested negative for the HIV virus by serology, and sputum staining for acid-fast bacilli was negative on multiple occasions.

The patient was empirically treated for pulmonary tuberculosis (TB) with six anti-TB medications for six months. Her symptoms progressed despite the empiric anti-TB treatment. She is a non-smoker and has no history of substance abuse. In the clinic, physical examination revealed a

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young female in no distress and without adenopathy, clubbing or oral lesions. Her chest was clear to percussion and auscultation. Heart sounds were regular without murmur. There was no hepatosplenomegaly. She was neurologically intact. Her total white blood count was 6.1 mm^3 with lymphocytes 43% and neutrophils 41%. She had a high erythrocyte sedimentation rate of 81 mm/hr. Serum chemistry levels were normal, including an angiotensin-converting enzyme level of 13 U/L. Electrocardiogram showed normal sinus rhythm. A computed tomography (CT) scan of chest with contrast showed infiltrates in the right upper, middle and lower lobes with a right pleural effusion (Fig. 1). Bronchogenic carcinoma with obstructive pneumonitis was suspected, based upon the clinical and radiological findings. A fiberoptic bronchoscopy was performed, revealing a friable right lower lobe posterior segment lesion appearing "pearly white," with irregular borders and completely occluding the entire segment (Fig. 2). Pathology revealed bronchial mucosa with extensive acute and chronic inflammation and numerous eosinophils. Staining with methenamine-silver was consistent with *Nocardia* (Fig. 3). The patient was started on trimethoprim-sulfamethoxazole.

The patient's cough and sputum production gradually resolved. Repeat CT scan a month later showed disappearance of the pleural effusion and partial clearing of the pulmonary densities (Fig. 4).

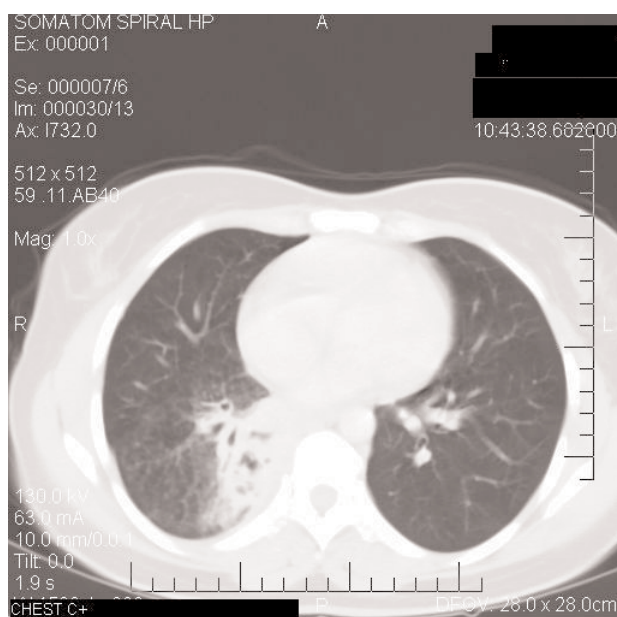


Fig. 1. CT scan of chest with contrast, illustrating infiltrates in right upper, middle and lower lobes with a right-sided pleural effusion.

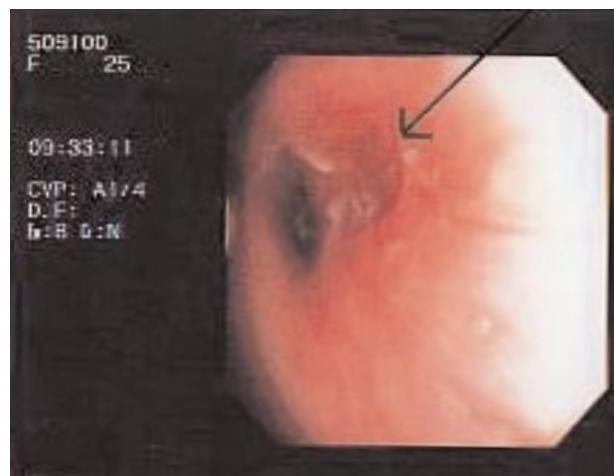


Fig. 2. Photography during bronchoscopy, showing the lesion in the right lower lobe (arrow).

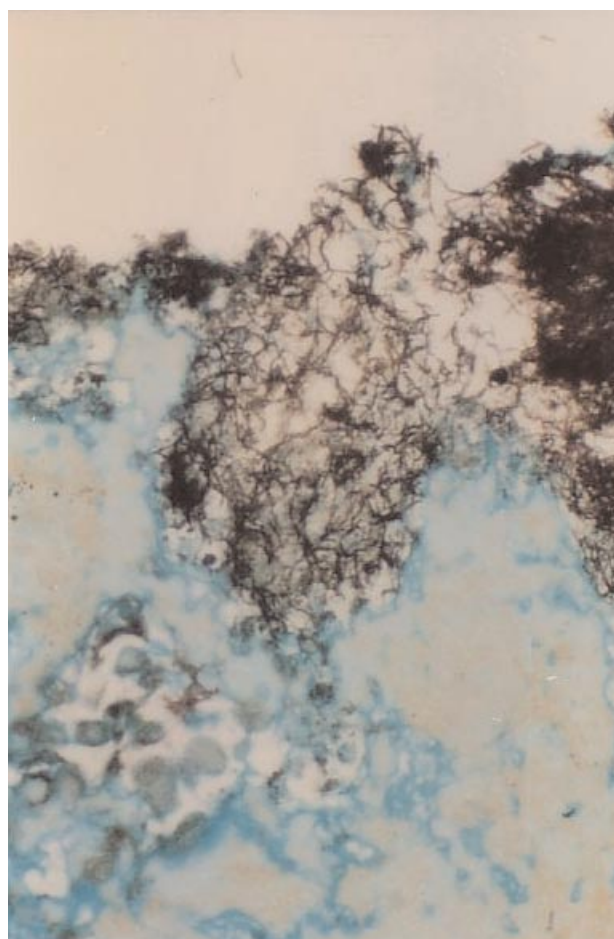


Fig. 3. Gomori methenamine-silver stain, demonstrating black filamentous *Nocardia* (magnification $\times 400\text{X}$).

Discussion

Our immunocompetent patient had pulmonary nocardiosis, which presented as non-resolving pneumonia.



Fig. 4. Repeat chest CT scans, showing disappearance of pleural effusion and resolving pulmonary densities.

Nocardia is characterized by aerobic, gram-positive, variably acid-fast, non-motile, non-spore-forming, branching filaments (6). Found in soil and vegetation, the organism is most virulent when inhaled. As a result, pulmonary infection is the most common form of the resulting disease. Hematogenous spread to other organs is also possible, especially in immunocompromised persons (3, 6). There are no known cases of person-to-person transmission (5). Clinical suspicion is crucial, since most antibiotics commonly used to treat pneumonia are ineffective against it. Delay in diagnosis is common (8). Pulmonary nocardiosis can be acute, subacute or a chronic suppurative infection (1). The clinical manifestations of pulmonary nocardiosis are nonspecific and include productive cough, fever, night sweats, anorexia, generalized malaise, chest pain and hemoptysis (6). Classic pneumonia, lung abscess, or cavitary disease with spread to contiguous structures are also typical clinical presentations.

Pulmonary nocardial infection is not usually considered in the differential diagnosis of an endobronchial tumor. Moreover, expectorated sputum may or may not yield positive smears or cultures even in established pulmonary infection. Various invasive procedures for obtaining specimens, including bronchoscopic biopsy, percutaneous lung aspiration and open lung biopsy, may be needed to

make a definitive diagnosis. Brown et al. (9) described a 28-year-old man who was found, on fiberoptic bronchoscopy, to have a large fungating mass extending into the right main stem bronchus. Two additional bronchoscopies and a mediastinotomy were required before a diagnosis of nocardiosis was made. Even though *Nocardia* will grow in routine bacterial culture media in approximately 2–14 days, bacterial cultures of pulmonary secretions are discarded after 48 hours, and therefore an opportunity for identification of *Nocardia* colonies will be missed. The microbiology laboratory should always be notified, so that culture techniques for the isolation of *Nocardia* are used. Sulfonamides remain the initial treatment and the mainstay of therapy for *Nocardia* infection (1, 10). The optimal duration for therapy is not clear, but most authors recommend 6–12 months of treatment at a minimum (1, 5).

We caution that this potentially treatable disease may be mistaken for tuberculosis, especially in immunocompetent patients, for whom the index of suspicion is low.

This case also underscores the importance of considering infectious processes as a cause of non-resolving lung opacities.

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