Does Smoking Increase the Risk of Pulmonary Nocardiosis? A Case Report and Review of The Literature

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ABSTRACT
We are presenting a case of a young male patient who was diagnosed with pulmonary nocardiosis; he was in an immunocompetent state of health, with no apparent risk factors apart from smoking. Initial antibiotics were not helpful. His diagnosis was later identified by microbial culture, which showed Nocardia species in the sputum sample. He responded to a long course of a combination of intravenous co-trimoxazole and imipenem.

Keywords
Pulmonary nocardiosis, Immunocompetent, Smoking.

Background
Nocardiosis is an important consideration and usually occurs in immunocompromised patients, requiring a high index of suspicion due to the non-specific clinical and radiological presentation that may mimic other diseases like TB or fungal infections.

Case Presentation
A 37-year-old male was admitted with dyspnea, cough productive of purulent sputum, and left-sided chest pain. All symptoms lasted for a week prior to the presentation. The cough was intermittent, with a specific pattern, no known relieving or aggravating factors, and no diurnal or day-to-day variation. The sputum was yellowish in color, moderate in amount; no hemoptysis. The chest pain started gradually, intermittent, described as sharp, exacerbated by movement and partially relieved by rest; no radiation; located in the left side of the chest.

He was also complaining of bilateral knee pain, more on the right side; the pain started gradually, contiguous, relieved by acetaminophen, no know exacerbating factors and not radiating.

A month before this presentation he had a fever that lasted for a few days, with no specific pattern, no day to day or diurnal variation, relieved by acetaminophen, which resolved spontaneously after that-also, no history of weight loss, night sweating, or anorexia.

No symptoms suggestive of autoimmune or connective tissue disease, no history of malignancy, no history of transplant before, and no history of chronic lung diseases. No history of receiving any medications before including steroids.

No known past medical or surgical history. His family history has no similar illness. He is married and works as a laborer. He smokes around a pack of cigarettes a day for around 15 years, no history of drinking any alcoholic beverages, no IV drug abuse, no extramarital relations, no herbal medications use, and no risk factors for TB or HIV.

On examination, he was hypoxic upon presentation, with oxygen saturation of 79% on ambient air. His chest examination reveals decreased breath sounds and hyper-resonance on the left middle zone. Cardiovascular, abdominal, and neurological examinations were unremarkable. Knee examinations were also unremarkable, apart from tenderness on palpation.

Labs revealed a WBC count of 17,000 x10^9/L, mainly neutrophils, but normal hemoglobin and platelet counts; C-Reactive Protein
(CRP) was 287 mg/L; ESR was 122 mm/hr; his renal function test was within normal range; his liver function revealed a mild increase in his transaminases; his HIV serology was negative.

The chest x-ray is shown in Figure 1 and the computed tomographic scan in Figure 2. The imaging revealed left lower lobe consolidation, left-sided pneumothorax, and left pleural effusion. The effusion was exudative with LDH at a level of 1586 U/L. The initial sputum culture was negative. Bronchoscopy showed a hyperemic mucosa with thick secretions in the left lower lobe and no endobronchial lesions. Bronchoalveolar lavage (BAL) was negative for malignant cells as well as AFB and fungal stains. A lung biopsy revealed no malignancy and no granuloma. He was treated with piperacillin-tazobactam and vancomycin and was not responding. Later, sputum and BAL samples revealed Nocardia otitidiscaviarum more than a week after they were cultured. He was treated, as per protocol, with a combination of imipenem-cilastatin and trimethoprim-sulfamethoxazole IV for a total of six weeks, then shifted to PO trimethoprim-sulfamethoxazole and doxycycline for another four weeks. He responded very well.

Discussion

Nocardia is filamentous, gram-positive, aerobic, branching, beaded filamentous rods. Human infection occurs by inhalation of airborne bacilli or traumatic inoculation into the skin. The most common presentation is an invasive pulmonary infection, disseminated disease, abscess, and cellulitis. It usually occurs in immunosuppressed patients, such as those suffering from leukemia, HIV, organ transplant, diabetes, or prolonged corticosteroid treatment, which all are known predisposing factors [1,2].

Some studies suggest that 15% of patients have no predisposing conditions [3]. The clinical features of pulmonary nocardiosis are often non-specific and include fever, cough, dyspnea, hemoptysis, and weight loss, so it may mimic TB, fungal infections, or lung cancer [4,5]. The radiological changes are also non-specific and include mass, consolidation, nodules, and pleural effusion [6,7].

The diagnosis of pulmonary nocardiosis requires the isolation of the organism in respiratory secretions. Sputum cultures are positive in nearly all patients, and the diagnostic yield with bronchoalveolar lavage would be higher [8]. In our case, Nocardia was isolated after one week of sampling, though the management strategy could change if isolated earlier.

New molecular testing such as polymerase chain reaction (PCR) and 16s ribosomal-DNA sequencing have enhanced the identification of different Nocardia spp [9]. Such techniques may help accelerate the diagnosis of these cases and the initiation of therapy.

We describe an uncommon presentation of pulmonary nocardiosis as pyopneumothorax in an immunocompetent patient. A literature search showed some reported cases of disseminated Nocardia sp. in an immunocompetent host with different outcomes and unclear risk factors, including pulmonary nocardiosis [10-12]. In these three reported cases of pulmonary Nocardia farcinica in an immunocompetent host with disseminated disease, their treatment included co-trimoxazole with or without a combination of other antibiotics. Outcomes varied. Two of the patients responded to treatment and survived. Other sites of infection were also reported with different Nocardia species, including cutaneous, intravascular, and brain infections. Some of these previous reports have shown the link between nocardiosis and a history of smoking tobacco [13,14].

Kumar and colleagues [13] discussed a 51-year-old male with no past medical history who had presented with a cough and shortness of breath that had been going on for two weeks. A review of symptoms was positive for significant loss of weight and appetite for three months duration. The patient was a weaver by occupation with a smoking history of 12.5 pack years. He was showing significant clinical improvement after two weeks of IV antibiotics. Further investigations showed positive HIV serology. Singh and colleagues [14] reported a case series of four female patients with pulmonary nocardiosis (Nocardia cyriacigeorgica and Nocardia nova) who had chronic lung diseases. A total of 3 out of these 4 cases were smokers, and all four did not have any identifiable cause of immune suppression.

A retrospective study of 59 cases of patients diagnosed with pulmonary nocardiosis showed that smoking status tended to be associated with a higher mortality [15], if the Brinkman index was >428, which is an index of the number of cigarettes smoked every day multiplied by the number of years of smoking; this is used to determine the cumulative dose of smoking.

Pulmonary nocardiosis should not be rolled out of the differential diagnosis among immunocompetent patients. It is essential to recognize the predisposing factors in this patient group and to differentiate infection from colonization when Nocardia is isolated. Clinical assessment, together with close collaboration with the microbiology laboratory, provides a more accurate diagnosis for initiating appropriate treatment with the purpose of reducing morbidity and mortality in these cases.
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