Bronchopneumonia Due to *Nocardia asteroides* in a Man With Chronic Obstructive Pulmonary Disease

To the editor: Nocardiosis is a rare disease that can affect the lung, soft tissue, and brain. The incidence of nocardiosis is higher among immunocompromised patients—such as those who have diabetes or are positive for human immunodeficiency virus—and in those receiving immunosuppressant drugs. It has occasionally been described in immunocompetent individuals, however. The use of techniques for earlier detection in recent years has made this a more common diagnosis. We report the case of a man with chronic obstructive pulmonary disease (COPD) and bronchiectasis who developed severe pneumonia due to *Nocardia asteroides*. Outcome was satisfactory.

A 57-year-old man with a medical history of smoking, alcoholism, bronchiectasis, severe COPD diagnosed 17 years earlier, and numerous admissions for exacerbations was hospitalized with an increase in his usual breathlessness, a low-grade fever, and a productive cough with purulent discharge that had started 10 days earlier. Chronic treatment included home oxygen therapy, bronchodilators, inhaled corticosteroids, theophylline, occasional use of antibiotics, and oral corticosteroids; he had not used the last drugs for the past 2 months. Upon physical examination the patient was conscious, oriented, and not cyanotic. Heart rate was 100 beats/min and respiratory rate was 25 breaths/min. The only remarkable lung sounds were coarse ronchi and disseminated wheezes. Noteworthy laboratory findings were a white cell count of 10 700 with a left shift and an erythrocyte sedimentation rate of 77 mm/h; arterial blood gas analysis revealed a pH of 7.35, PaO_2 of 85 mm Hg, $PaCO_2$ of 51 mm Hg, and HCO_3 of 28 mmol/L. A chest radiograph showed pulmonary air trapping, residual lesions in both pulmonary apices, including increased linear markings (tram tracking), signs of precapillary pulmonary hypertension, and a slight alveolar pattern in the lingula. After starting supplemental oxygen therapy, inhaled bronchodilators, and empirical intravenous antibiotic therapy (amoxicillin–clavulanic, 875/125 mg/8 h), the patient's condition worsened, with increased dyspnea, persistent purulent sputum and fever. Ciprofloxacin (500 mg/12 h) was added to the regimen on the fourth day of admission. The fever peaked at 39ºC on the 8th day of hospitalization. Blood cultures were negative and the sputum culture grew the usual flora. A chest radiograph showed bilateral patchy alveolar infiltrates in the left lower lobe, the lingula, and both upper lobes (Figure). On the 9th day, fiberoptic bronchoscopy revealed signs of inflammation and purulent secretions but no endobronchial lesions. Microbiological tests and cultures of the telescoping catheter and bronchial aspirate indicated the presence of branching gram-positive bacilli that were weakly acid-fast by Ziehl-Neelsen staining; they were identified as *N asteroides*, sensitive to amoxicillin–clavulanic acid, cefuroxime, cefotaxime, gentamicin, vancomycin, and trimethoprim–sulfamethoxazole. With a firm diagnosis of bronchopneumonia due to *N asteroides*, intravenous treatment with cefotaxime (1 g/6 h) and imipenem (500 mg/6 h) was prescribed. The patient responded slowly, with radiographic improvement, although he had intermittent fever throughout the next 16 days. Thirty days after admission, the patient was discharged under treatment with trimethoprim–sulfamethoxazole.

Nocardia species, in the Nocardiaceae family, are weakly acid-fast, aerobic, gram-positive bacilli. *N asteroides* is the main

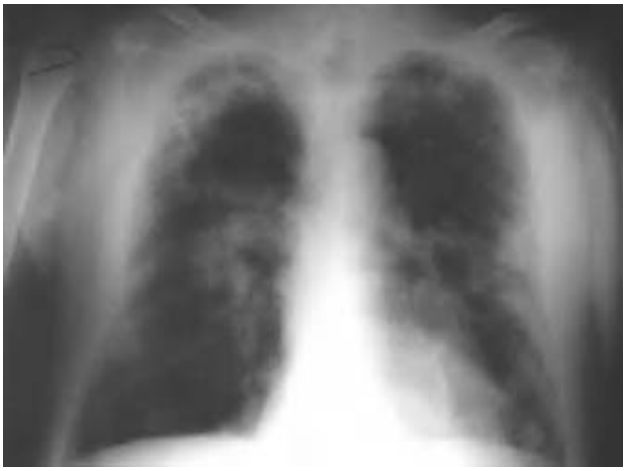

Figure. Bilateral patchy alveolar infiltrates and cystic bronchiectasis.

respiratory pathogen in the genus, whereas *N braziliensis* is responsible for skin diseases.[1] The incidence of infection by *Nocardia* species in Spain is unknown. In larger series reported in the literature,[2-4] the predisposing factors that are most often identified are immunodepression secondary to neoplastic disease or HIV infection, whereas the presence of underlying COPD ranges from 10% to 30%. However, most COPD patients were receiving corticosteroids chronically and at the time the infection appeared, a factor that has also been associated with increased mortality. Our patient was not receiving corticosteroids at the time of admission, although the presence of mucociliary dysfunction in COPD and bronchiectasis can be a predisposing factor, aggravated by the possibility of prior silent colonization, as has been described in asymptomatic carriers.[5] Symptoms mentioned in the literature are very nonspecific: coughing (100%), purulent secretions (70%), fever (70%), and dyspnea.[2-4] That was the clinical picture in our patient's case. Radiographic findings are highly variable: lobar and multilobar consolidation, a tendency to cavitation, pleural effusion, solitary masses, and even reticulonodular infiltrates and mediastinal lymph node enlargement may be present.[6] Our patient had bilateral alveolar infiltrates, and we therefore used a more invasive procedure to reach a definitive diagnosis.[7] Trimethoprim–sulfamethoxazole has traditionally been the treatment of choice, although in vitro sensitivity is not always related to results in vivo. That limitation, along with resistance to those drugs has led to the use of combined treatment (imipenem plus third generation cephalosporins or imipenem plus amikacin) in severely ill patients.[2-4] Treatment lasts from 6 to 12 months and is continued at least 6 months longer in immunocompromised patients.

In conclusion, in patients with exacerbated COPD and unusual radiographic findings, *N asteroides* may be implicated. Mortality due to infection by this pathogen is high (30%-50%)[2-4] and because its isolation and identification are difficult, invasive techniques should be considered early so that appropriate treatment can be started. Other approaches are associated with high mortality.

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