Hypersensitivity Pneumonitis Associated With *Mycobacterium chelonae*

To the Editor: *Mycobacterium chelonae* is a nontuberculous mycobacterium that is included in the group of rapidly growing mycobacteria.[1,2] The mycobacteria of the *Mycobacterium chelonae-abscessus* group are ubiquitous microorganisms that can cause infection when inhaled, although lung disease caused by *M chelonae* is uncommon. Indeed, in a large series of patients with pulmonary lesions caused by rapidly growing mycobacteria, very few infections were caused by this particular mycobacterium,[3] and we found no cases of hypersensitivity pneumonitis caused by *M chelonae* infection in the literature, whereas we did find cases associated with infection by *M immunogenum*.[4]

A 53-year-old woman with no relevant medical history attended our clinic with a 9-month history of progressive dyspnea, dry cough, and intermittent fever. These symptoms were not associated with asthenia, anorexia, or weight loss. In the systems review, no other associated clinical manifestations were identified. The patient worked as a concierge and had no apparent contact with toxic products. Of note among her personal history was the presence of a parrot in her home and 3 journeys abroad (to Cuba, Sahara, and—6 months earlier—Mexico), although she had not had any contact with exotic animals.

In the physical examination, the patient did not have a fever and her blood pressure, heart rate, and respiratory rate were normal. The chest examination

revealed dry rales at the lung bases but was otherwise normal. The complete blood count was normal, the erythrocyte sedimentation rate was 38 mm/h; coagulation parameters, biochemistry including liver enzymes), and urinalysis (including sediment tests) were all normal. Basic immunological tests for rheumatoid factor, antinuclear antibodies, and antineutrophil cytoplasmic antibodies were negative. No abnormalities were apparent in the conventional chest radiograph and resting arterial blood gas analysis revealed mild hypoxemia and hypocapnia (PaO_2, 74.1 mm Hg; $PaCO_2$, 33 mm Hg). Serial blood cultures and stool and urine cultures were repeatedly negative although *M chelonae* was repeatedly identified in sputum cultures (6 samples obtained on different days). According to the antibiotic susceptibility tests, the microorganism isolated was sensitive to clarithromycin, imipenem, amikacin, and linezolid. The results of serology for common viruses and precipitin tests of budgerigar droppings (e77) and feathers (e78) were negative. Computed tomography of the chest showed enlarged mediastinal lymph nodes (<1 cm), as well as a bilateral patchy interstitial pattern predominantly in the region of the left lung base. Lung function tests showed a mild decrease in forced vital capacity (74% of predicted) in dynamic spirometry, a decrease in tidal volume (77% of predicted) in plethysmography, and a moderate decrease in single-breath carbon monoxide diffusing capacity. No macroscopic abnormalities were observed with fiberoptic bronchoscopy. Samples of bronchial aspirate and bronchial lavage were taken and a transbronchial biopsy was performed. The culture of the bronchial aspirate was positive for *M chelonae*. Cytology of bronchoalveolar lavage revealed 49% lymphocytes, 49% macrophages, 3% neutrophils, 2% eosinophils, and 2% mastocytes. According to the phenotypic study of the lymphocytes, the ratio of $CD4^+$ to $CD8^+$ cells was 0.33. In the transbronchial biopsy, an increase in mononuclear cells was observed in the lung interstitium and a small non-necrotizing granuloma with giant cell infiltrate was identified.

The patient received treatment with clarithromycin and linezolid for 6 months. The outcome was favorable and the cultures were negative. At present, she is asymptomatic and the findings of the complementary tests have returned to normal.

The syndromic diagnosis of hypersensitivity pneumonitis was based on a combination of clinical and radiographic findings, lung function tests, bronchoalveolar culture, and histopathologic study of the lesions.[5] In the case reported here, the patient met all the criteria that are characteristic of hypersensitivity pneumonitis. The syndromic diagnosis of this disease should be completed with studies aimed at identifying the causative agent. Certain nontuberculous mycobacteria, specifically *Mycobacterium avium*, *Mycobacterium terrae*, and *M immunogenum*, have been reported as the causative agents in isolated cases or outbreaks of hypersensitivity pneumonitis. However, in an exhaustive literature search, we were unable to find any cases of this disease caused by *M chelonae*. The case described meets, in addition to the aforementioned criteria of hypersensitivity pneumonitis, the criteria of the American Thoracic Society for nontuberculous mycobacterial infection.[6] Despite an exhaustive medical history, it was not possible to locate the source, although an imported infection is likely given the temporal relationship between the onset of the clinical manifestations and the trip to Mexico. The findings from the imaging studies were indicative of hypersensitivity pneumonitis and unusual in disease caused directly by nontuberculous mycobacteria, which usually lead to such findings as nodules, bronchiectasis, and cavitation.

Clinical improvement in hypersensitivity pneumonitis depends essentially on eliminating the antigenic stimulus. If this is not found, corticosteroids should be used. In the case described, treatment of mycobacteriosis was associated with clinical resolution. This outcome indicates there was no permanent exposure, distinguishing this case from other cases of hypersensitivity pneumonitis related to nontuberculous mycobacteria.[4] In addition, the clinical course, without application of corticosteroids, suggests that treatment of the causative agent can be curative in hypersensitivity pneumonitis due to mycobacteria if the disease is not severe.

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