

LETTERS TO THE EDITOR

Alveolar proteinosis or the importance of identifying concurrent infections

To the Editor: Alveolar proteinosis (AP) is a rare disease which affects the lungs diffusely and is characterized by the deposition of protein-like material rich in phospholipids. This material is characteristically PAS positive and does not cause interstitial alveolar inflammation; thus the degree of fibrosis minimal¹. Signs and symptoms are nonspecific and patients are generally asymptomatic before complications occur. The main complication in AP is concurrent infection with unusual germs like *Aspergillus* sp., *Nocardia* sp., *Mycobacterium* sp., *Cryptococcus neoformans*, *Histoplasma capsulatum*, *Pneumocystis carinii* and certain viruses². We report the case of a woman diagnosed with AP after being infected with *Mycobacterium tuberculosis*.

The patient was a 41-year-old female who smoked 500 cigarettes per year and was a moderate drinker with no relevant medical or occupational history. The patient complained of night sweats, dyspnea upon moderate effort, cough with abundant whitish sputum and wasting syndrome with loss of 6 to 7 kg in weight starting three months earlier. She had presented bloody sputum on one occasion one week before admission. Perihilar ground-glass opacities were observed in the chest radiograph. Physical exploration showed no significant findings. Relevant analytical findings were leukocytes, 6550/ μ l (neutrophils 57%, lymphocytes 34%); hemoglobin, 14 g/dL; hematocrit, 38.6%; platelets, 212 000; and erythrocyte sedimentation rate, 10 mm/h. Biochemistry, proteinogram, coagulation and urine analyses were normal. Nuclear antibodies, anti-smooth muscle and rheumatoid factors were normal. The angiotensin-converting enzyme level was 51 (30-50). The pneumonia panel was negative and no *Legionella* antigen was found in urine. Lung function testing gave normal findings, including a carbon monoxide diffusion capacity of 82.3%. Resting arterial gases were measured, pH being 7.39, PaO₂ 68 mmHg and PaCO₂ 41 mmHg. A computed tomography scan of the thorax showed bilateral ground-glass opacities, mainly in high and mid fields. Bronchoscopy revealed no endobronchial lesions, but PAS-positive material was detected in bronchial aspirate and bronchoalveolar lavage samples. The Löwenstein-Jensen culture was positive for *M. tuberculosis*. A transbronchial biopsy gave no remarkable findings. There was good response to treatment with isoniazid and rifampicin for six months and with pyrazinamide for the first two months. The patient remained asymptomatic one year later. A chest radiograph showed no changes, pulmonary function tests were normal and the biopsy was still PAS positive. Löwenstein-Jensen cultures were negative.

The clinical presentation of AP is variable and nonspecific. The most frequent symptoms are dry cough and dyspnea. Some patients present no symptoms before developing concurrent infections, as in the case we report. Although favorable results have been described after treatment with granulocytic-monocytic series colony stimulating factor³, the only treatment of proven efficacy is whole-lung lavage⁴. This treatment is only

prescribed for patients with dyspnea or respiratory insufficiency^{5,6}. Nevertheless, before considering whole-lung lavage, it is necessary to rule out specifically treatable concurrent infections. Our patient experienced symptoms while the *M. tuberculosis* infection persisted and remained asymptomatic after being treated; therefore, whole-lung lavage was not considered. With the exception of patients with severe respiratory insufficiency, identifying and treating concurrent infections is the best way to improve quality of life and prognosis for AP patients. While these infections were the main cause of death in the past, the prognosis has currently improved considerably with their appropriate treatment.

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