Lithoptysis in a Patient With Primary Ciliary Dyskinesia

To the Editor: We read with interest the report by García Pachón et al1 of a case of idiopathic chronic lithoptysis. As a complement to their observations, we describe the case of a patient diagnosed with primary ciliary dyskinesia who presented an episode of lithoptysis while being followed by our department. Dyskinetic cilia syndrome or primary ciliary dyskinesia is a recessive autosomal illness that is characterized in its full manifestation by chronic rhinitis, sinusitis, otitis, recurring bronchitis, bronchiectasis, male sterility, and corneal and olfactory abnormalities.2 Infection by Mycobacterium tuberculosis is currently the main cause of broncholithiasis, followed by infection by Histoplasma capsulatum.3 One sign of broncholithiasis, lithoptysis, which consists of expectoration of one or more broncholiths, an event that can resolve the clinical picture in some cases.

A 34-year-old man diagnosed with primary ciliary dyskinesia developed a cough and spontaneously expectorated a stone (Figure 1), with no other associated manifestations. The patient could not recall any similar previous episodes. His medical history included an idiopathic pleural effusion in childhood, repeated episodes of bronchial hyperresponsiveness in the context of respiratory infections, bilateral bronchiectasis, and pulmonary tuberculosis diagnosed in 1994. The patient was therefore prescribed treatment for 6 months. The family history included a sister who had also been diagnosed with primary ciliary dyskinesia. The pneumologist’s examination showed only overall reduced vesicular murmur, with prolonged expiration and some isolated rhonchi. The hemogram, coagulation tests, and biochemistry—including bone metabolism, immunoglobulin study, proteinogram, α1-antitrypsin, and thyroid function—were normal. Hepatitis and human immunodeficiency virus serologies were negative and both urine analyses and kidney function tests were normal. Spirometry at rest showed a forced expiratory volume in the first second of 39.1% of predicted; the ratio between the 2 was 40.98. Computed tomography (CT) of the paranasal sinuses revealed hypogenesis of the frontal and maxillary sinuses and mucosal thickening. A high-resolution CT scan of the chest revealed correctly placed mediastinal structures, with no adenomegaly in the hilar and mediastinal regions; small apical fibrotic areas, bilateral cylindrical bronchiectasis, and slight apical pleural thickening were disclosed. The CT scan also revealed 2 calculi of 3 mm and 5 mm, respectively (Figure 2), inside the right bronchial tree in the upper and middle lobes. Bronchoscopic revealed no broncholiths in the bronchial lumen or abnormalities in the mucosa that might indicate underlying lithiasis. The expectorated broncholith had a hard, irregular, whitish coraliform surface upon macroscopic inspection. Mineralogical analysis showed the stone to be 70% calcium oxalate; the remainder consisted of calcium phosphate (hydroxyapatite). In light of these analyses and because the patient did not present symptoms or complications.

There is no consensus regarding treatment options, which range from mere observation to bronchoscopic broncholithectomy and, finally, surgery.4 In our case, we opted for a wait-and-see approach given the absence of symptoms or complications.

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