Airway Calcium Deposition and Broncholithiasis in Disorders of Mucociliary Clearance

To the Editor: We read with interest the report by García Pachón et al[1] regarding idiopathic chronic lithoptysis and the letter by Roig Vázquez et al[2] on lithoptysis in a patient with primary ciliary dyskinesia (PCD). Complementing these observations, we recently completed an analysis of lithoptysis and airway calcification in a large cohort (n=142) of well-characterized PCD patients.[3] We identified 4 patients reporting lithoptysis with calcium deposition on radiographic imaging (computed tomography scan and chest x-ray), 1 patient with lithoptysis without radiographic calcium deposition, and 2 patients with radiographic evidence of calcium deposition without lithoptysis. In all but 1 patient, the distribution of calcium deposition was similar to the PCD patient reported,[2] that is calcium deposition involving the airways without involvement of hilar or mediastinal lymph nodes or abdominal calcification. Calcium deposition was present in lobes with severe bronchiectasis and all patients were over the age of 40 years, suggesting chronic inflammation is required. Broncholiths were available for analysis in 2

of these patients with calcium deposition involving the airways. Samples stained for fungi and mycobacteria were negative. Energy dispersive x-ray (EDX) coupled with morphological analysis identified calcium chloride (calcite). In 1 patient, calcium deposition was focal and parenchymal on radiology and resembled calcium deposition associated with granulomatous inflammation. EDX coupled with morphological analysis of a broncholith available in this patient identified calcium phosphate. No patient had a history of pulmonary mycobacterial or mycotic infection and purified protein skin testing was negative in all patients.

We agree with the hypotheses suggested by both authors as regards the pathogenic mechanism of calcification in these patients.[1,2] We surmise that airway calcification is a biomineralization response to chronic inflammation and retention of airway secretions. However, if this is indeed the mechanism of calcium deposition, why do patients with cystic fibrosis (CF), a more common genetic disorder of mucociliary clearance, not present with broncholithiasis and airway calcium deposition? Possibilities include a milder PCD phenotype, altered calcium metabolism in CF, or a lower pH of CF airway surface liquid not conducive to calcium deposition. Further analysis of calcium deposition as an endpoint of inflammation in pulmonary disorders including patients with broncholithiasis may lead to new insights into the mechanisms of inflammation and biomineralization in other disease processes such as atherosclerotic heart disease.

Marcus Peter Kennedy MD,[a] Peadar G. Noone,[b] and Michael R. Knowles[b]
[a]MD Anderson Cancer Center, Houston, Texas, USA
[b]University of North Carolina, Chapel Hill, North Carolina, USA

1. García Pachón E, Grases F, Padilla Navas I, Gallego JA, Valero B, Romero V. Litoptisis crónica idiopática. Arch Bronconeumol. 2005;41:468-70.
2. Roig Vázquez F, Ansotegui Barrera E, Martínez Albiach JM. Litoptisis en paciente con discinesia ciliar primaria. Arch Bronconeumol. 2006;42:417.
3. Kennedy MP, Noone PG, Carson J, Molina PL, Ghio A, Zariwala MA, et al. Calcium stone lithoptysis in primary ciliary dyskinesia. Respir Med. 2007;101:76-83.