Metastatic pulmonary calcinosis

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To the editor: Metastatic pulmonary calcinosis (MPC) is an uncommon process of unknown etiology associated to a wide variety of both benign and malignant conditions. Pathophysiologically, MPC consists of calcium deposition in the epithelial and vascular basement membrane of alveoli, bronchial walls, and media layer of pulmonary arteries, resulting in a lymphoproliferative interstitial response and pulmonary fibrosis. The significance of early diagnosis of this complication lies in the fact that pulmonary calcifications, in this particular condition, may be potentially reversible with adequate, early treatment.

We report the case of a 37-year-old male, a former smoker with no known drug allergies and a history of kidney transplant for chronic renal failure (CRF) secondary to membranoproliferative glomerulonephritis, systemic arterial hypertension, and acute myocardial infarction. Routine chest X-rays of the patient showed an alveolar-interstitial pattern in both upper lobes, predominately on the left side, with no respiratory symptoms, and was therefore admitted to hospital for diagnostic work-up. Blood and chemistry laboratory test results included: hemoglobin 6.9 g/dL, hematocrit 20.9%, WBCs 9.500/mm^3 with normal differential count, platelet count 195,000/mm^3, ESR 135 mm, iron metabolism and coagulation study within normal limits, except for fibrinogen increase to 766 mg/dL. Basal glucose 86 mg/dL, creatinine 8.6 mg/dL, urea 119 mg/dL, sodium 138 mEq/L, potassium 4.3 mEq/L, chloride 100 mEq/L, calcium 9.23 mg/dL, phosphorus 7.83 mg/dL, parathormone 2,475 pg/mL, normal TSH, C-reactive protein 1.65 mg/dL, beta-2-microglobulin 20.80 mcg/mL, lipid profile, proteinogram, and liver function within normal ranges. Diagnostic tests included a high-resolution CT scan (fig. 1) that showed high-density bilateral involvement of middle and upper pulmonary fields consistent with calcifications. Radiographic bone series revealed advanced vascular calcifications. Functional respiratory test results included: forced vital

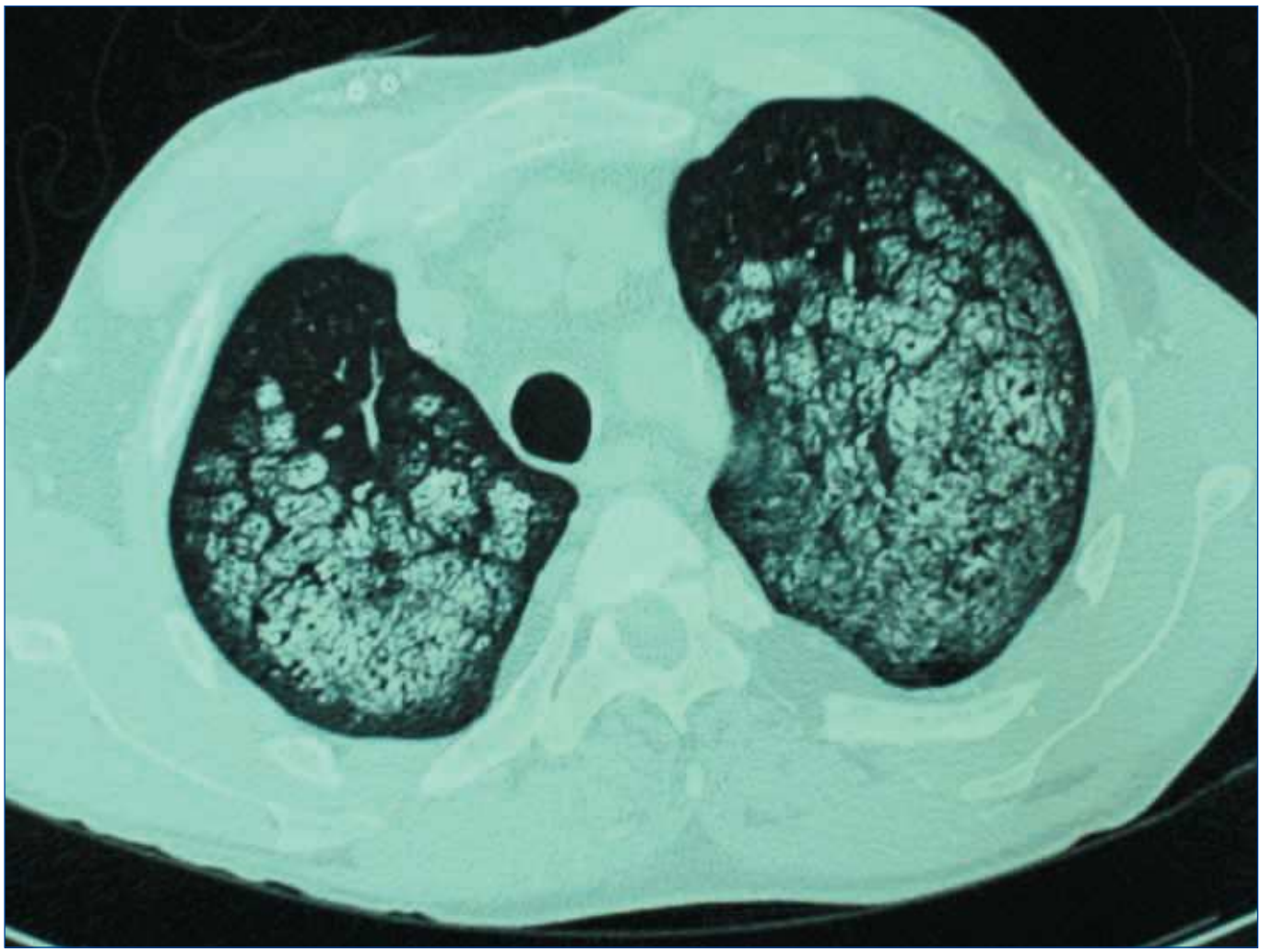

Figure 1.

capacity (FVC), 4.22 L (81.2 % of predicted); forced expiratory volume in the first second (FEV1), 3.60 L (88.4 % of predicted); FEV1/FVC, 85.1 %; RV, 2.04 L (109.4 % of predicted); TLC, 6.23 (91.2 % of predicted). CO diffusion was moderately decreased in absolute values, partially corrected with AV measured, suggesting loss of alveolar units for exchange. Bronchoscopy visualized a bronchial tree with whitish images of a hard consistency, linear and parallel to each other and diffusely distributed in both lungs, consistent with metastatic calcifications. BAL and transbronchial pulmonary biopsy were performed and confirmed diagnosis.

In 1855, Virchow first described metastatic pulmonary calcinosis. This is usually associated to changes in calcium and phosphorus metabolism and to systemic and local pH. The most common cause of metastatic calcifications is chronic renal failure, and other less common causes include primary and secondary hyperparathyroidism, kidney transplant, osteopetrosis, hypervitaminosis D, and malignant diseases such as multiple myeloma, leukemia, parathyroid carcinoma, and others. A high prevalence (up to 60%-80% in certain series) is found in patients with chronic renal failure undergoing hemodialysis. Chest X-rays are poorly sensitive for diagnosis of this condition, while the greater sensitivity of high-resolution CT allows for detecting small calcifications. Clinical course is usually silent, and most patients remain asymptomatic, as occurred in the onereported here. If calcium deposits are extensive, a decreased diffusion, a restrictive functional pattern, and hypoxemia may be seen. Pulmonary fibrosis may occur in most severe cases, leading to respiratory insufficiency and even death.

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Cryptococcosis in a patient with IgA nephropathy treated with corticosteroids

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To the editor: IgA glomerulonephritis (IgA GMN) is the leading cause of primary GMN in the world.[1] Its natural course without treatment is a slow progression to chronic renal failure in approximately 50% of patients.[2] Because of this course, use of corticosteroids has been postulated in recent years in patients with risk factors for progression to chronic renal failure such as increased serum creatinine levels (SCr), proteinuria of approximately 500-1000 mg/day, and hypertension.[1,3-5] We report the case of a 60-year-old male patient with a history of atrial fibrillation, type 2 diabetes mellitus, and dyslipidemia who was referred to our clinic for progressive renal function impairment from SCr 1.1 mg/dL to 2.7 mg/dL in three months with a creatinine clearance (ClCr) of 40 mL/min, microhematuria, and proteinuria. A renal biopsy allowed for diagnosing IgA GMN with significant tubulointerstitial involvement. Treatment was started with ACEIs (enalapril 10 mg/24 h) and a tapering corticosteroid regimen (80 mg/24 h). After two months of treatment, the patient reported fatigue, headache, and tinnitus, and showed renal function impairment (SCr 4 mg/dL and ClCr 16 mL/min). A lumbar puncture was performed, and the subsequent culture showed the presence of *Cryptococcus neoformans.* Imaging tests revealed lesions consistent with cryptococcoma in basal ganglia and the left parasagittal region. Corticosteroids were discontinued, and treatment was started with amphotericin B and flucytosine for two weeks, plus oral fluconazole for one additional month. Lesions disappeared, and the patient is currently asymptomatic, with SCr of 3.3 mL/min, ClCr of 31 mg/mL, and proteinuria of 1.0 g/24 h, and under treatment with enalapril 10 mg/24 h.

In summary, corticosteroids are potent immunosuppressants of both humoral and cell-mediated immunity,[7] which causes patients treated with them to have a 40-fold greater predisposition than untreated patients to suffer infection by opportunistic or atypical microorganisms.[8] Thus, since use of immunosuppressants is a standard therapeutic weapon in our routine clinical practice, we should not forget its potential harmful effects and should watch patients who receive them for the occurrence of symptoms and signs, however trivial they may appear, in order to be able to take any adequate diagnostic and therapeutic actions.

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