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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## Genetics and environment: pathogenetic factors of vasculitis?

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**To the editor:** Etiopathogenesis of vasculitis is not fully understood, and environmental factors have been implicated in genetically predisposed individuals.[1,2] The histological renal expression of systemic vasculitis is a pauci-immune necrotizing glomerulonephritis (PNG).[3] Two cases of familial vasculitis in two brothers living in a rural environment are reported here.

### CASE 1

A 63-year-old male, a shepherd living in a rural environment. He consulted in 1988 for an impaired general status, and laboratory tests revealed oliguric acute renal failure. Histological analysis of renal biopsy found a necrotizing vasculitis with extracapillary proliferative glomerulonephritis. The patient was treated with corticosteroids and oral cyclophosphamide. No renal function improvement occurred, and renal replacement therapy was required until the patient died in 2003.

### CASE 2

A 72-year-old male, a farmer living in a rural environment. In April 2004 he complained of cough, expectoration, fatigue, and anorexia. His family history revealed that the patient reported as case 1 was his brother. His parents had died at an advanced age, and he had two sisters with type 2 diabetes mellitus, one of them with a history of pulmonary tuberculosis.

Based on his personal history, clinical signs, and renal function impairment, a renal biopsy was performed, which confirmed the presence of PNG in the setting of a systemic vasculitis associated to P-ANCA (positive anti-MPO, titer 442 U/mL).

### DISCUSSION

Pauci-immune necrotizing glomerulonephritis with extracapillary proliferation is the renal pathological expression of systemic vasculitis. This group of diseases is characterized by inflammation of small and medium-sized blood vessels, and includes Wegener's granulomatosis (WG), microscopic polyangeitis (MPA), Churg-Strauss syndrome, or vasculitis limited to the kidney.[3]

Their etiopathogenesis is unknown. Occurrence of these diseases in several members of a same family has suggested that genetic factors could contribute to its occurrence.[1] We report two cases of PNG as a renal manifestation of systemic vasculitis in two brothers. In case 2, the disease started when the first patient had already died. The presence on the same disease in two brothers could support the genetic component in the etiopathogenesis of vasculitis.

Some researchers have attempted to find associations of vasculitis with HLA genes.[1] Recent studies found a positive association with HLA DR1, particularly in patients with WG, and negative associations with HLA DR3, particularly in Churg-Strauss granulomatosis and polyarteritis nodosa.[1,4] Our patient had the haplotype A1, B8 B35 Cw4 Cw7 DR3 and DR5 DQ2. Case 1 haplotype is unknown because the patient died before the second patient experienced the disease.

It has also been suggested that environmental factors could contribute to disease development in genetically predisposed individuals.[2] Patients reported here lived in a rural environment, and some environmental component may possibly have contributed to occurrence of the same disease in both patients.

In conclusion, these two cases of PNG as an expression of systemic vasculitis in two brothers living in a similar environment could support the suggested hypothesis of an influence of environmental factors on the etiopathogenesis of vasculitis in genetically predisposed individuals.

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## Intracranyal hypertension as presentation of neurobrucellosis in a patient on hemodialysis

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**To the editor:** Brucellosis is a zoonosis with a high incidence rate in Spain, particularly in rural areas. While neurological involvement is uncommon, it has clinical significance due to the associated morbidity.

We report the case of a 29-year-old male born in Senegal with an unremarkable epidemiological history. His clinical history included arterial hypertension and chronic kidney disease (CKD) from an unknown cause on chronic hemodialysis for one year. Patient reported low grade fever and fatigue for the past 15 days. During hemodialysis, he expe-