A 36-year-old Senegalese man was referred to the nephrology service for intermittent gross hematuria for several months. He had no significant past medical history and had never smoked. He had resided in France for 5 years but previously had been working in Gabon. The patient denied any history of fever, sweats, pruritus, or abdominal pain. Physical examination was unremarkable. Blood pressure was 125/78 mm Hg. White cell count revealed mild eosinophilia (810 per μl, N < 650). Liver tests and renal function were normal (creatinine clearance was 130 ml/min). Schistosomiasis and HIV serologies were negative. Urine protein was 0.32 g/day. Urine and stool parasitologic examinations did not reveal schistosoma eggs. Chest radiograph, plain film of the abdomen, and renal ultrasonography were normal. Glomerular disease was suspected: patient underwent a renal biopsy that failed to show any glomerular pathology. Immunofluorescence with anti-immunoglobulin A (IgA), IgG, IgM, C1q, C3, λ and κ-chain, fibrin, and albumin was negative. However, abnormalities were seen in some glomerular capillaries (Figure 1) and renal arterioles.

What was the cause of his hematuria?
The Diagnosis | Loiasis

Intravascular forms were identified as loiasis (Figure 2). Fresh blood sample confirmed a massive microfilarial load (8000 Loa loa per ml), whereas filarial serology was negative. The patient was hospitalized to initiate antifilarial treatment. He received 12 mg of ivermectin on day 1, then 25 mg of diethylcarbamazine (DEC) on day 2. Prednisone (1 mg/kg/day) was added to minimize the risk of drug-induced Loa encephalopathy. Nevertheless he experienced fever (39.2°C) and profound encephalopathy (Glasgow Coma Scale = 9/15) within hours following DEC intake. Simultaneously a significant rise in proteinuria (up to 2 g/day) was noted. Loa loa microfilariae (mff) were found in both cerebrospinal fluid and urine. DEC was stopped, prednisone was continued, and the patient was admitted to the intensive care unit for 10 days. The patient progressively recovered without sequelae. Six weeks later, a second course of DEC (beginning with very low doses: 3 mg/day, doubling daily up to 400 mg) was started, resulting in blood sterilization and complete normalization of eosinophilia and urine sediment. The patient did not exhibit further episodes of hematuria.

Visceral manifestations of loiasis, as encephalopathy and renal impairment, are well described and are attributable mainly to antifilarial treatment;1 spontaneous cases of visceral complications are rare.2

Loiasis can be associated with hematuria, proteinuria, and, infrequently, the nephrotic syndrome and renal failure.1 Membranous and membranoproliferative glomerulonephritis, which are associated with the deposition of immune complexes, have been described in this setting. Renal impairment following a single dose of ivermectin3,4 or DEC treatment1 have been reported. These adverse effects are thought to be immunologically mediated and could be the consequence of a massive liberation of filarial antigens following therapy. Encephalopathy is often fatal or leads to serious neurological sequelae. Most patients who develop Loa encephalopathy following treatment have very high microfilarial loads, exceeding 30,000 mff/ml, but a risk exists after ivermectin as soon as the threshold of 8000 mff/ml is reached.1 No specific treatment for the management of these severe adverse effects has yet been identified. Mechanisms remain unclear, but pathological findings in an animal model suggest that the occlusion of cerebral capillaries due to numerous mff damaged or paralyzed by the drug may be the cause of encephalopathy.1 Massive release of antigens or endotoxins, consequent to parasitic destruction, may also be involved in this setting, as well as in kidney involvement.1

Hematuria in a patient coming from an endemic area of loiasis (i.e., Central Africa) should lead to the examination of blood to confirm the diagnosis, as serology may be false negative in this setting. Renal microvascular involvement may be the only manifestation of infestation. A very careful introduction of antifilarial drugs should be performed to avoid life-threatening Loa encephalopathy.

REFERENCES