Sir,

Since Shribman et al. [1] reported the case of a man with miliary tuberculosis and focal proliferative glomerulonephritis (GN), no more than 5 well-documented cases of GN associated with systemic or pulmonary tuberculosis have been described. The purpose of this work is to report a new case.

A 66-year-old previously healthy man, who had suffered for the last 2 months of constitutional syndrome, edema of the lower extremities and abdominal swelling, was admitted. His physical examination revealed edema of the legs and ascites as the only positive findings. Laboratory investigations disclosed nephrotic syndrome: urinary protein output 3 g/24 h and serum albumin 2.8 g/dl. Mantoux test was strongly positive. He had neither hematuria nor renal insufficiency. The following tests were negative or within normal limits: antinuclear antibodies, rheumatoid factor, cryoglobulins, ASLO, serum complement and virus B markers. His plain chest film was normal. A percutaneous renal biopsy was performed, which revealed a membranous nephropathy. An extensive study to rule out tumoral cause yielded negative results. He received diuretics and prednisone (40 mg daily p.o.) and after 2 months stay he was released from the hospital with mild pretibial edema and persistent proteinuria. Two months later he was readmitted because of generalized edema. He presented proteinuria (2.9 g/24 h) and mild renal insufficiency (serum creatinine 1.9 mg/dl). Chest Xray examination revealed three nodular infiltrates with cavitation involving both of the lungs. The search for acid-fast bacilli in urine and sputum was negative. Blood culture was negative for fungi, aerobic and anaerobic microorganisms. The fiberoptic bronchoscopy and cytological examination of the bronchial aspirate were negative. Bronchial washing was negative for Mycobacterium tuberculosis. The patient refused transthoracic fine needle aspiration and open lung biopsy. Upper gastrointestinal series, barium enema, intravenous urogra-phy and tumoral markers (β-HCG, CEA, α-fetoprotein) were normal. Abdominal echography revealed ascites as the only positive data. An embolism site was ruled out by means of echocardiography and digital angiography of the renal veins.
The patient was treated with rifampin (600 mg/day) and isoniazid (300 mg/day). After 2 months of tuberculostatic therapy his chest X-ray film became normal, and 6 months later he had no proteinuria, with a normal renal function. Two years later he was still in a good condition. Until now, the association between military tuberculosis and focal proliferative GN [1] has been described as well as the association between membranoproliferative GN in the course of disseminated tuberculosis [2] and the association of pulmonary tuberculosis, cryoglobulinemia and membranoproliferative GN [3]. In all cases tuberculosis infection and glomerulopathy improved after tuberculostatic therapy. It has been proposed that renal lesions might be the consequence of immune-complex deposition [1]. To our knowledge the association between pulmonary tuberculosis and membranous nephropathy has not been reported previously. Two features must be considered. First, as for the diagnosis of pulmonary tuberculosis in this case, it is well known that in some cases an open lung biopsy is required to reach the diagnosis [4], but we could not perform this procedure in our patient. Second, a coincidence between a common disease, such as tuberculosis, and a frequent type of GN such as membranous nephropathy, should be considered. Nevertheless, we think that in this patient tuberculosis might be the cause of glomerulopathy as both respiratory and renal involvement improved after tuberculostatic therapy, and there was no response to corticosteroid treatment.

References