Dear Sir,

The presence of Congo red negative fibrillar deposits in the glomeruli was first described in 1977 by Rosenmann et al. [1]. Ever since this entity has been reported with increasing frequency [2] under a variety of names such as ‘fibrillary glomerulonephritis’ [3], ‘Congo red negative amyloidosis-like glomerulopathy’ [4] or ‘immunotactoid glomerulopathy’ [5]. The lesion is characterized histologically by the presence of glomerular extracellular microtubules which have been shown to contain immunoglobulins and are negative for amyloid by Congo red stain [1-7]. Clinically these patients are usually adults, with proteinuria sometimes in the nephrotic range, microscopic hematuria, hypertension and progressive renal insufficiency [2, 7], lacking clinical or serological evidence of disorders associated with fibrillar deposits [5, 8-10] such as systemic lupus erythematosus, cryoglobulinemia, amyloidosis or light chain deposit disease. Renal transplantation has been performed in some of these patients and disease recurrence has been reported [6, 7].

This entity has been described in association with a variety of clinical conditions such as rheumatoid arthritis [4], leukocytoclastic vasculitis [11], metastatic carcinoma of the liver [12] and malignant lymphoma [13]. We report another case of nonamyloidotic fibrillar glomerulopathy in a patient with rheumatoid arthritis.

A 60-year-old woman was admitted to the hospital because of renal insufficiency and arthritis. She was well until 20 months earlier when she experienced the onset of pain and swelling in knees and ankles with functional incapacity; subsequently arthritis also developed in wrists, fingers and elbows. 1 year later she was diagnosed as having rheumatoid arthritis (Latex 1/320). She was only treated with aspirin and nonsteroid anti-inflammatory drugs (indomethacin and naproxen).
She was admitted to hospital because of generalized articular stiffness so that she was confined to bed, associated in the last days with nausea, vomiting and decreased urinary output. Her temperature was 36.5°C, the pulse was 90 and the blood pressure was 110/60 mm Hg. On examination the patient was a dehydrated obese woman; the lungs were clear and the heart and abdomen were normal. The peripheral pulses were intact and no peripheral edema was present; inflammatory signs in wrists, fingers, knees and ankles were present. The hematocrit was 30%; the white cell count was 18,200 with 7% bands and 84% neutrophils and the platelet count was 698,000. The total proteins were 7.4 g/dl, albumin 2.2 g/dl, uric acid 15.3 mg/dl, sodium 137 mEq/l, potassium 5.7 mEq/l, calcium 8.9 mg/dl, phosphorus 13.2 mg/dl, urea nitrogen 160 mg/dl, creatinine 6 mg/dl, bilirubin 0.3 mg/dl, aspartate aminotransferase 15 U, alanine aminotransferase 12 U, cholesterol 109 mg/dl, triglycerides 132 mg/dl and glucose 105 mg/dl. The urine gave 3.2 g/l for protein and the sediment contained numerous red cells. The serum IgG was 2,061 mg/dl, the IgA 600 mg/dl and the IgM 112 mg/dl. The C3, C4, latex, cryoglobulins and antinuclear antibodies were normal or negative. Serum and urine immunoelectrophoresis were negative for monoclonal light chains. An electrocardiogram and an x-ray film of the chest were normal. The ultrasonographic examination of the abdomen revealed two normal-size kidneys with loss of the corticomedullary junction. Bone marrow aspirate was normal.

On admission peritoneal dialysis was started; the nonsteroid anti-inflammatory drugs were discontinued and prednisone was added; articular symptoms ameliorated spectacularly and increasing diuresis was evident after the 4th day, the creatinine being 1.7 mg/dl on the 24th day. On the 25th day a renal biopsy was performed. The histological study showed glomeruli with varying degrees of mesangial expansion by PAS-positive material; staining with Congo red was negative and there was no abnormal birefringence. Ultrastructural examination showed glomeruli with diffuse extracellular infiltration by randomly arranged nonbranching microfibrils, 10-12 nm thick (fig. 1). These fibrils were located in subendothelial space and mesangial matrix and they were absent in tubular membranes. Because of technical problems immunofluorescence study was not available. A diagnosis of nonamyloidotic fibrillar glomerulopathy was made. Hemodialysis was started 40 months after discharge.

References
121