Dear Sir,

Sarcoidosis and systemic lupus erythematosus (SLE) are both multisystemic, distinctive disorders of unknown etiology. Sarcoidosis is characterized by an accumulation of monocyte-macrophages to form noncaseating granulomata. In SLE, tissue manifestations are associated mainly with complement activation by autoantibodies and immune complexes, and to some extent by cell-mediated immune complexes, and to some extent by cell-mediated immune mechanisms. Renal involvement may occur in both diseases but is more common in SLE. The coexistence of sarcoidosis and SLE is uncommon [1-4] and the simultaneous involvement of the kidney in this situation is rare. To our knowledge, this is only the second case reported where the kidney is involved in such patients, and the first case was not biopsied [2].

A 43-year-old female presented initially with iridocyclitis, which improved with topical prednisone. A year later, she developed cough with bilateral interstitial lung infiltrates showing sarcoid granulomata and was treated successfully with steroids. Ten months later, she developed recurrent lung disease, fever, generalized arthralgia, hilar and nontender inguinal lymphadenopathy. Laboratory studies were as follows: urine protein 0.8 g/24 h with normal sediment, serum BUN 4.6 mmol/l (15 mg/dl), creatinine 53 mmol/l (0.6 mg/dl), calcium 1.9 mmol/l (7.8 mg/dl), phosphorus 0.90 mmol/l (2.8 mg/dl), albumin 27 g/l (2.7 g/dl), and polyclonal globulins 38 g/l (3.8 g/dl). ANA was 1:320 with a smooth pattern, and anti-double-stranded (ds) DNA was 28% (normal 0-4%). The rheumatoid factor was positive, angiotensin-converting enzyme (ACE) was 115 U/l (normal 8-52), C3 9.8 g/l (98 mg/dl) and C4 1.3 g/l (13 mg/dl; normal 15-45). A 3-day pulse dose of intravenous methylprednisolone produced remission.

By percutaneous renal biopsy, the glomeruli showed mild to moderate mesangial widening, thickened capillary walls and small areas of capillary thrombi and necrosis (fig. 1). Scattered small interstitial, noncaseating granulomata were noted (fig. 2). Immunofluorescence microscopy (IF) revealed diffuse, granular 3 + IgM, 2 + IgG, 3 + C1q and 1 + IgA deposits in the glomerular mesangium and capillary walls. Subepithelial and mesangial electron-dense deposits were found (fig. 3a, b). A diagnosis of membranous and focal glomerulonephritis along with interstitial sarcoid granulomata was made.
The patient responded to prednisone in tapering doses over the next 8 months. Four months after prednisone was discontinued, she noted transient arthralgia in her small joints with persistent ocular blurring and photophobia but no pulmonary symptoms. Renal function remained stable with trace proteinuria. The anti-ds DNA antibody titer had normalized, but the ACE level was still elevated at 97 U/l.

Although the commonest cause of renal disease in sarcoidosis is related to hypercalce-mia and nephrolithiasis [5], other renal com-

Fig. 1. A small interstitial, noncaseating granuloma adjacent to a glomerulus showing slight thickening of capillary walls and mesangium. PAS. × 240.

lications of sarcoidosis such as granulomatous infiltration causing interstitial nephritis [6], granulomatous arteritis, glomerular diseases [5, 7], and functional tubular abnormalities have also been noted. Clinical renal manifestations range from hematuria and varying degrees of proteinuria to reversible renal insufficiency or failure. In some cases, crescentic glomerulonephritis as well as crescents in conjunction with other glomerular lesions have been observed [6]. But none of the glomerular lesions previously reported were accompanied by simultaneous granulomatous lesions in the interstitium as seen in the present case.

Various studies have demonstrated some overlapping clinical features and immuno-logic abnormalities in sarcoidosis and SLE such as hypergammaglobulinemia, autoanti-bodies, altered T-lymphocyte function and circulating immune complexes [2, 3] which play a major role in the pathogenesis of these two entities. Although both diseases are capable of invoking various forms of immune-complex-mediated glomerular disease, the histologic appearance, together with the EM and IF findings in our patient favor lupus nephritis; while the renal interstitial granulomas are part of generalized sarcoidosis.

Both sarcoidosis and SLE have to be considered in the treatment of renal lesions in such patients, depending on their type and severity. Careful monitoring of renal function and urinalysis in patients with sarcoidosis may help to uncover early or subtle renal disease for prompt intervention. Routine periodic serological tests in these patients can identify the development of any other associated autoimmune disorders.

Fig. 3. Electron micrograph of a portion of glomerulus showing subepithelial (a) and mesangial (b) electron-dense deposits. Uranyl acetate and lead citrate a × 6,600. b × 14,700.

References