Steroid-Responsive Nephrotic Syndrome with IgA Deposits

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Dear Sir,

Several recent reports have focused attention on cases of steroid-responsive nephrotic syndrome (NS) in association with IgA glomerular deposits. The precise classification of these patients is at present controversial [2, 3, 6]. Those previously reported cases have similar clinical and histologic features as most of them were children with minimal change lesions. However, this condition has also been considered a rare variant of IgA nephropathy [4], minimal lesion nephrosis with IgA mesangial deposits [5] or the coincidence of two glomerular diseases where IgA nephropathy has superimposed on minimal change NS [1]. We present a patient with steroid-responsive NS and mesangial IgA deposits showing 'early' lesions of focal glomerular sclerosis.

A 20-year-old man developed edema on October 2, 1985, without a previous history of infection. Blood pressure was 150/85 mm Hg, serum creatinine 1 mg/dl, proteinuria 92 mg/kg/day and urine red blood cells 8,000/min. Serum albumin was 26 g/l and total protein 43 g/l.

Cryoglobulins, antinuclear antibodies and HBsAg were negative. The serum C3, C4 and CH50 were normal. IgA levels were not raised (97 mg/dl), IgG 420 mg/dl and IgM 107 mg/dl. The patient received a salt-restricted diet and diuretics. A percutaneous renal biopsy showed 29 glomeruli. On light microscopy 3 were sclerosed and another 2 showed focal and segmental glomerulosclerotic lesions (fig. 1). The rest exhibited irregular mesangial hypercellularity with a mild increase in mesangial matrix. There were small foci of interstitial fibrosis and tubular atrophy. Immunofluorescence disclosed generalized and diffuse mesangial deposits of IgA (2+) and C3 (1+). Electron microscopy showed no evidence of electron-dense deposits in the glomerular basement membrane or in the mesangium. On October 27, 1985, proteinuria was negative, serum albumin rose to 36 g/l and total serum protein went up to 63 g/l. Three weeks later, a second attack of NS was present with

edema and proteinuria 72 mg/kg/day. Serum albumin dropped to 28 g/l, hematuria was not detected and renal function remained normal. Treatment with 1 mg/kg/day of prednisone was started; proteinuria disappeared on the 15th day. On the last examination, 3 months after prednisone withdrawal, the patient was asymptomatic and proteinuria was not present.

We think that our patient has an idiopathic NS with IgA mesangial deposits and not IgA nephropathy (Berger's disease). It is known that IgM deposits may appear in some cases of nephrosis, most probably as a consequence of secondary trapped proteins in previously injured

tissue. A similar phenomenon resulting in IgA deposition could also explain the rare observation of IgA deposits in this disease.

Fig. 1. a Glomerulus showing segmental sclerotic lesion (arrow). PAS. $\times 450$. b Diffuse mesangial IgA deposits. IF. $\times 250$.

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