Berger's Disease without IgA Deposits?

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Dear Sir,

The accompanying micrograph shows a typical glomerulonephritis with mesangial electron-dense deposits. The kidney biopsy was performed in a 17-year-old girl presenting the clinical picture of Berger's disease but without IgA mesangial deposits on immunofluorescent microscopy; light microscopy showed focal glomerulonephritis and immunofluorescence IgG and C3 mesangial deposits. Moreover we found that the patient had selective IgA deficiency; serum immunoglobulins measured by automated immunoprecipitation using commercial antisera were: IgA 11 mg/dl (range 189 ± 67), IgG 2,340 mg/dl (range $1,116 \pm 206$), IgM 128 mg/dl (range 92 ± 34).

Nowadays IgA nephropathy is accepted worldwide as an entity [1]. Several different clinical conditions share this common immunopathology and variations in immunopathogenesis allow different evolutions of the disease.

The primary IgA nephropathy (Berger's disease) is mainly a disease of young people characterized by recurring episodes of macroscopic hematuria with pharyngitis and the histological features are: (1) glomerular mesangial deposits of IgA usually accompanied by C3 and sometimes by IgG and IgM; (2) mesangial expansion with variable proliferative and sclerotic segmental lesions; (3) electron-dense mesangial deposits in the absence of systemic disease.

Berger's disease is commonly classified as an immune complex glomerulonephritis for several reasons [2]. Identifying the nature and origin of the mesangial immune deposits in this disease, however, still does not adequately explain why patients in whom such deposits develop also manifest glomerulonephritis and progressive renal failure [3]. Indeed, IgA immune complexes are also detectable in nonhematuric patients with mesangial IgA deposits; moreover, in some experimental models of

Fig. 1. Electron micrograph. $\times 4,000$. Evident mesangial electron-dense deposits without pathological findings in the glomerular capillary wall. M = Mesangial cell; MM = mesangial matrix; E = endothelial cell; G = neutrophil granulocyte. The arrows show typical mesangial electron-dense deposits.

IgA nephropathies, IgA deposits can be visualized in the mesangium without the simultaneous occurrence of he-maturia. Finally, mesangial cells might be stimulated by several factors, probably including immune complex deposition, and they could mediate the glomerular damage. These data indicate that IgA immune complexes are not necessarily pathogenetic and that other factors are involved in the development of the glomerulonephritis.

Our patient presented a selective IgA deficiency and the clinical picture and histological glomerular findings of Berger's disease, although without immunofluores-cent IgA mesangial deposits. This clinical case may suggest that in Berger's disease IgA mesangial deposition might be a coincidental result rather than a cause of the disorder.

References

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