Evolution of Mesangial IgM Nephropathy into Focal Segmental Glomerulosclerosis

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To the Editor:
The significance of mesangial IgM deposits in patients with idiopathic nephrotic syndrome who have no morphologic abnormalities by light microscopy is controversial. Should such cases be considered minimal-change disease, or do these IgM deposits identify a clinically distinct entity? In a clinicopathological exercise detailing the case of a nephrotic child having minimal-change disease with mesangial IgM deposits, Davis and McCluskey [1] indicated that ‘it will be important to determine whether the presence of conspicuous IgM deposits indicates a relatively poor prognosis’. Cohen et al. [2] have suggested that previously reported cases of transition from minimal-change disease to focal segmental glomerulosclerosis may have been examples of glomerular lesions containing IgM evolving into focal segmental glomerulosclerosis. In light of these statements the following case is of interest, even though such an anecdote certainly does not resolve the significance of mesangial IgM deposits in renal biopsy tissue that would otherwise be diagnosed as minimal-change disease.

A 2 5-year-old black male with idiopathic nephrotic syndrome of 1 month duration underwent a percutaneous renal biopsy in April 1979. He was normotensive and had no hematuria or renal insufficiency. By light microscopy, glomeruli had no abnormalities. There were no tubulointerstitial changes. Immunofluorescence microscopy revealed mild granular mesangial deposition of IgM but no IgG, IgA, C3 or C4. Ultrastructurally, visceral epithelial cells had complete fusion of foot processes and microvillus transformation. There was slight segmental mesangial matrix expansion, but no glomerular electron-dense deposits. Therefore, at that time, the patient’s renal tissue had the light and electron microscopic features of minimal-change disease along with mesangial IgM deposits.

The patient was treated with steroids but followed a relapsing and remitting course for 1 year when his nephrotic syndrome became steroid resistant. During this time his creatinine also became slightly elevated.

A second biopsy was performed 16 months after the first biopsy. By light microscopy approximately 20% of glomeruli had segmental hyaline sclerosis with associated glomerular foam cells and adhesions to Bowman’s capsule. There was focally variable interstitial edema and fibrosis. By immunofluorescence microscopy there were mild diffuse global granular mesangial IgM and C3 deposits as well as more intense focal segmental globular IgM and C3 deposition corresponding to sclerotic segments. The two glomeruli examined ultrastructurally did not contain the segmental sclerotic lesion. A diagnosis of focal segmental glomerulosclerosis was made.
The focal segmental glomerulosclerosis could have been missed in the first biopsy by sampling error, or could have evolved from the milder lesion observed at that time. In either event, the presence of IgM might be of value in indicating those cases which otherwise appear as minimal-change disease but actually are missampled or nascent focal segmental glomerulosclerosis. However, a definitive conclusion concerning the significance of mesangial IgM in patients with minimal changes by light and electron microscopy must await clinicopathologic evaluation of many cases with and without IgM deposits.

References