IgA Nephropathy and Nephrotic Syndrome

Dear Sir,

We have recently read with great interest the paper by Rambausek et al. [1]. During the period from 1981 to 1985, IgA nephropathy was diagnosed in 63 patients who account for 14.6% of all biopsy-confirmed cases with primary glomerular disease in our hospital. Nephrotic syndrome was present in 8 patients (12.7%). They were sub-classified into two groups according to the response of nephrotic syndrome to treatment (table I). It was obvious that a relatively short history of the renal disease, normal blood pressure, intact renal function on discharge, and minor renal histologic changes were frequently present in the patients with response to steroid therapy (3 cases). In contrast, those without response to steroid and/or other immunosuppressives more often had a prolonged history of the renal disease, hypertension, renal dysfunction on discharge, and relatively severe histologic alterations. The follow-up data during the periods of 4–18 months of these patients are summarized in table II. All 3 patients with response to steroids had only intermittent hematuria and/or mild proteinuria (between 0.2 and 1.0 g/daily urine collection). On the contrary, heavy proteinuria ( > 2 g/daily urine collection) was found in 4 of the 5 patients without response to treatment. Three of them still had hypertension and required antihypertensive therapy. All of them presented with chronic renal insufficiency or chronic renal failure. One case (No. 4) developed chronic renal failure 1 year after the first renal biopsy and continued to evolve from diffuse mesangial proliferative to diffuse sclerosing glomerulonephritis on the second renal biopsy. Many investigators have indicated that the presence of heavy proteinuria at the initial evaluations

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Table I. Comparison of clinical and pathological findings between patients with (responsive) and without (nonresponsive) response to steroid treatment

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<td>Y.-S.</td>
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Table II. Follow-up data during 4–18 months of patients with IgA nephropathy

1 From first onset of symptoms to the time admitted.

was almost always associated with the subsequent development of progressive renal insufficiency [2–4]. However, Mustonen et al. [5] reported that 3 of the 8 cases with IgA

1 Chronic renal insufficiency; CRF = chronic renal failure; all data were recorded at the time of renal biopsy except otherwise indicated.

2 Greater than 15% of glomeruli involved.
nephropathy and nephrotic syndrome had normal blood pressure and were responsive to steroid treatment. Several other studies have supported the response to steroids and indicated that the renal histology of their patients usually showed no change or only mild mesangial alteration [6–11]. Interestingly, Sinniah [12] reported that mesangial IgA as the predominant immunoglobulin was found in 8 of 200 autopsy cases (4%) in whom the

Table II. Renal histology and follow-up of patients with (responsive) and without (nonresponsive) response to steroid treatment

| Nonresponsive | 4 diffuse mesangial hematuria, heavy 12proliferative GN proteinuria2, increasedBP, CRF, and diffuse sclerosing GN |
| GN = Glomerulonephritis; BP = blood pressure; CRF = chronic renal failure; CRI = chronic renal insufficiency. |
| 1 | Between 0.2 and 1.0 g/daily urine collection. |
| 2 | Greater than 2 g/daily urine collection. |

glomerular morphology was essentially normal or revealed only minor changes (compared to 4.76 and 1.49% of the patients with steroid responsiveness to total cases of IgA nephropathy in our investigation and in the report by Rambausek et al., respectively). A recent animal study of IgA nephropathy also showed consistent results with spontaneous IgA deposition in the normal murine glomeruli [13].

It is suggested that some cases with the diagnosis of IgA nephropathy and nephrotic syndrome may be isolated minimal-change glomerulonephritis with nonspecific glomerular mesangial trapping of IgA. At the present time, however, we would conclude that patients with IgA nephropathy and nephrotic syndrome can be further categorized into two clinicopathologically distinct groups. Both the therapeutic response and the histologic evaluation may be quite helpful in determining the prognosis of this association.

References


