Disappearance of Glomerular IgA Deposits in Steroid-Responsive Nephrotic Syndrome

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Dear Sir,

Nephrotic syndrome or nephrotic range-proteinuria in IgA nephropathy is generally accepted as one of the possible indicators of a poor prognosis [1]. However, several descriptions of steroid-responsive nephrotic syndrome associated with glomerular IgA deposits, with a feature like minimal change nephrosis, have been recently recognized by several investigators [2–12], and several hypotheses for those occasions are realized at present, but the precise pathognomonic cognition is controversial. In our series of steroid-responsive nephrotic syndrome with IgA deposits, we report 2 patients in whom serial renal biopsy revealed the disappearance of glomerular IgA deposits.

Case 1. A 30-year-old man developed edema with a prior history of common cold, and was admitted on January 20, 1987. Five years earlier, he had been hospitalized for nephrotic syndrome. Shortly after the diagnosis of IgA nephritis (mesangial proliferative glomerulonephritis) was established (fig. 1), he subsequently received corti-costeroid therapy, just when the nephrotic syndrome was remarkable for complete remission. On this admission, urinalysis revealed 7.0 g of daily proteinuria without hematuria. Total protein was 4.7 g/100 ml and serum creatinine was 0.8 mg/100 ml. The profiles of immunological and serological parameters were essentially normal. IgA levels were raised to 512 mg/100 ml, IgG 585 mg/100 ml, IgM 135 mg/100 ml. Percutaneous renal rebiopsy included 10 glomeruli on light microscopic examination that showed mild mesangial proliferative glomerulonephritis. On immunofluorescence, not only IgA but other immunoglobulins and complements were negative. Electron microscopic examination was not studied. Corticosteroid therapy completely relieved nephrotic syndrome. Subsequently, elevated serum IgA levels returned to normal values accompanied by remission of nephrotic syndrome.

Case 2. A 37-year-old man was admitted on December 10, 1986, due to the fourth relapse of nephrotic syndrome. During the previous 4 years, nephrotic syndrome had recurred on three occasions. In each of those relapses, complete remission was achieved with corticosteroid therapy. In the first onset of nephrotic syndrome, renal biopsy was performed, which showed diffuse mesangial proliferative glomerulonephritis with isolated IgA deposits. Electron microscopic examination revealed mesangial deposits and foot process effacement. On this admission, urinalysis revealed 10.0 g of daily proteinuria without hematuria. Total protein was 3.2 g/100 ml and serum creatinine was 1.6 mg/100 ml. Serological examinations indicative of secondary disorders were all negative. IgA levels were 236 mg/100 ml, IgG 402 mg/100 ml, IgM 130 mg/100 ml. IgE (RIST) levels were high, i.e. 2,100 IU/ml. Percutaneous renal rebiopsy contained 21 glomeruli that on light microscopic examination showed diffuse mesangial proliferative glomerulonephritis with sclerotic lesions (12/21) and dense lymphocytes infiltration in the interstitium. On immunofluorescence only granular depositions of C3 were observed in glomerular mesangium. Electron microscopic examination revealed striking foot process effacement and small amounts of mesangial deposits. Three weeks after the initial corticosteroid therapy proteinuria was decreased and remission of nephrotic syndrome ensued. In the above 2 patients we observed the disappearance of glomerular IgA deposits on repeated renal biopsy. The series of simultaneous unusual findings, such as steroid responsiveness, lack of hematuria, absence of a decline in renal function, elevated serum IgA levels, and histological pictures of mesangial proliferation with predominant IgA deposits, are opposed to classical IgA nephritis or minimal change nephrosis. And still more, the distinctive feature of the disappearance of glomerular IgA deposits substantially raises the possibility that this condition can be considered a variant form of minimal change nephrosis. As described by Nicholls et al. [13] and D'Amico [14],

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Fig. 1. Immunofluorescence of the first renal biopsy in case 1, showing mesangial deposits of IgA. $\times 100$.

mesangial deposition of IgA is thought to persist throughout the course of the disease, and the disappearance of IgA deposits in classical IgA nephritis has not been demonstrated. IgA deposits in glomeruli may not be concerned with the onset of nephrotic syndrome but with the initial proliferation of the mesangial area [15]. This rare occasion, such as that found in our patients, may be due to mesangial hyperfunction in minimal change nephrosis.

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