Development of Immunoglobulin A Nephropathy in a Kidney Transplant in a Patient with Previous History of Henoch-Schönlein Purpura Glomerulonephritis

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Dear Sir,

Recurrence of IgA nephropathy in the transplanted kidney is well recognized and occurs frequently [1]. Henoch-Schönlein purpura (HSP) also recurs in the transplant [2,3]. The relationship between HSP and IgA is also recognized as they have identical renal histological features, are associated with raised serum IgA and show the presence of IgA in normal skin capillaries. However, they differ in their clinical presentation as is well known. We describe a case with well-proven HSP which progressed end-stage renal disease who received a live related donor transplant. Two years following transplantation, the patient developed IgA nephropathy in transplanted kidney.

A 20-year-old man was first seen in 1979 with recurrent episodes of purpuric skin rash following attacks of upper respiratory tract infection. This continued for 4 years and was associated with recurrent attacks of frank hematuria and persistent microscopic hematuria in between. He was found to be normotensive with 24-hour urinary protein of 2.4 g and creatinine clearance of 30 ml/min. In January 1983, the patient underwent renal biopsy which showed mesangial proliferative glomerulonephritis, and the immunofluorescence test showed diffuse staining for IgA and focal staining for IgM and C3 in the mesangium. The electron-microscopic examination showed immune complex deposition in the mesangium and paramesangium. He was diagnosed as having HSP. The patient was lost to follow-up from 1985 and then turned up in 1988 with end-stage renal failure. He received a live related renal transplant from his brother

in March 1988 and was immunosuppressed with cyclosporin A and prednisolone. His graft functioned very well, but in March 1991, he was noticed to have hematuria (577,000 glomerular RBC/ml with 100% granular casts), and his renal function deteriorated from 105 mmol/l to 200 µmol/l, no frank hematuria was noted nor was there skin rash. C3, C4, anti-DNA and serum protein electrophoresis were all normal. Serum immunoelectrophoresis showed raised IgA (5.18 g/l, normal range 1-4). His cyclosporin level was kept in the therapeutic range throughout, and he continued to receive a maintenance dose of

prednisolone (10 mg daily). 24-hour urine protein was 0.12 g/day. Ultrasonography and Doppler ultrasonography of the transplanted kidney were normal. Renal biopsy of the transplanted kidney was done and showed mesangioproliferative glomerulonephritis on light microscopy, and immunofluorescence showed heavy deposits of IgA, IgM and C3 in the mesangium. Electron microscopy showed deposits in the mesangium; biopsy of the normal skin showed negative immunofluorescence. We describe a case with HSP proven clinically and by renal biopsy who 2 years after the transplantation presented with IgA nephropathy. This case illustrates the relationship between HSP and IgA nephropathy which has been extensively discussed in the nephrologic literature [4]. It is generally thought that they represent the same or similar pathological process presenting with pure renal manifestation (IgA nephropathy) or renal and extra-

renal manifestation (HSP). The presence of IgA in the mesangium in both conditions as well as the presence of IgA in the skin capillaries of normal skin and raised IgA in the serum in both conditions support the notion that both are related to the activation of *mu*-cosal antibody in response to infection or foreign body, e.g., food.

Both IgA nephropathy and HSP recur following renal transplantation [1, 2]. In a recent interesting study [5], it was shown that of 12 live related transplants in HSP, 9 patients showed histological evidence of recurrence of IgA in the mesangium of the transplanted kidney. Of those 9 cases, 4 had no evidence of glomerulonephritis clinically or on urine investigation and 5 had hematuria and proteinuria but no clinical evidence of HSP. Only 2 patients developed purpuric rash typical of HSP 48 days and 22 months after renal transplantation. In 3 cases [5], biopsy done 1 h after transplantation showed the presence of paramesangial IgA. Other papers also showed that symptomless cases of IgA in the mesangium can occur after transplanting patients with HSP [6, 7]. Our case is further evidence of the relationship of HSP and IgA nephropathy. At the time of this study, he showed no evidence of HSP but he may develop this in the future. It should be added that he was transplanted many years after the transplantation of all evidence of active HSP.

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248

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