Recurrent IgA Nephropathy in a Renal Allograft Presenting as Crescentic Glomerulonephritis

C.P.		Streather
J.E.		Scoble

Renal Unit, Kings College Hospital (Dulwich), London, UK

Dr. C. P. Streather, Renal administration, Dulwich Hospital, East Dulwich Grove, London SE22 8PT (UK)

Dear Sir,

IgA nephropathy is acknowledged to be a common cause of recurrent glomerulonephritis in renal allografts. Recurrence rates of 38-50% have been described in different morphological series [1-3]. However, there is general agreement that it is rare for such histo-logical disease to be clinically important [4,5]. Diaz-Tejeiro et al. [2] reported a case of recurrent IgA nephropathy having a rapidly progressive course and proceeding to graft loss. We report a second case of crescentic IgA nephropathy in a renal allograft and suggest that this complication may not be as unusual as originally thought.

700

600

500

400

300

200 -

Plasma exchange

Methylprednisolone

Case Report

A 43-year-old man presented with hypertension, proteinuria and haematuria in 1983. A renal biopsy showed a fairly advanced glomerulopathy with some glomerulosclerosis; immunofluorescence confirmed the diagnosis of IgA nephropathy. No specific treatment was instituted and end-stage renal failure was reached by 1988. After a short period of renal replacement therapy, he received a cadaveric renal allograft. Standard immunosuppression with cyclosporin A, Azathioprine and prednisolone was used and the creatinine was stable at 160 $\mu\text{mol/l}$. Four years after transplantation, he presented in outpatients with signs of an acute abdomen. At this point, his urine contained > 100 red cells/mm³ and casts. At laparotomy, he was found to have a localised peritonitis around the entrance to the femoral canal but no detectable perforation. The day of surgery his creatinine rose to 205 $\mu\text{mol/l}$ but had fallen to baseline by the fourth post-operative day. Over the next 2 weeks, as his abdomen recovered, his creatinine rose to a peak of 630

100

Fig. 1. Creatinine versus time, in days, since this presentation.

$\mu\text{mol/l}$ and his urine volume fell to 500 ml in 24 h. Investigations at this time showed his anti-neutrophil cytoplasmic antibody, anti glomerular basement membrane antibody and rheumatoid factor to be all negative. Serum immunoglobulins were normal but C3 was reduced at 0.41 g/l (normal range 0.55-1.2 g/l). An ultrasound-guided renal transplant biopsy was performed, which showed a proliferative glomerulonephritis with 40% of the glomeruli containing cellular crescents. There was also marked acute tubular necrosis, and immunoperoxidase was strongly positive for IgA. The patient was treated with three consecutive days of plasma exchange 3-litre exchanges of fresh frozen plasma and 4.5% albumin. It was decided to use this mode of treatment because the patient had had a prolonged post-operative ileus, was hypoalbuminaemic and we were concerned about his fitness to withstand immunosuppression with cyclophosphamide. The day after the biopsy, the urine volume increased and the creatinine rose more slowly, polyuria lasted for several days and the creatinine fell steadily to $234 \mu\text{mol/l}$ a week after the biopsy. At this stage, a second biopsy was performed to plan further immunosuppression. This showed a less active glomerulonephritis with 25% crescents some of which had become fibrous, in marked contrast to the first biopsy where all the crescents were active and cellular. The acute tubular necrosis was resolving but there was now clear evidence of cellular rejection. This responded to our standard treatment of 3 successive days of intravenous methylprednisolone and on discharge 4 days later, the creatinine was $218 \mu\text{mol/l}$. The continuing fall in creatinine to baseline can be seen in figure 1.

©1994 S.Karger AG, Basel 0028-2766/94/ 0661-0113\$2.75/0

Discussion

End-stage renal failure is reached in about 20% of patients with IgA nephropathy [4]. The prognosis in renal transplants has been widely held to be better, and transplant failure secondary to IgA nephropathy has been considered a rarity. However, some reservation was expressed about this view in a series of transplant biopsies and a review of the literature in 1986 [6]. Over a 14-year period 13 transplant patients with IgA nephropathy were biopsied and the disease had recurred in 6. One of these lost his graft from recurrent IgA nephropathy, but not with an acute crescentic course. Further attention as drawn to the possibility of clinically important recurrence by Diaz-Tejeiro et al. [2] and despite an initial honeymoon period after plasma exchange and cyclophosphamide treatment, the graft was lost 5 months later. In the patient we have described, the acute glomerulonephritis appeared to have started to resolve prior to the initiation of plasma exchange, so no additional immunosuppression other than routine transplant medication was used. Reviews of recurrent glomerulonephritis in transplant patients have necessarily included only small numbers of IgA nephropaths [3,6,7], and it is therefore quite possible that the incidence of significant disease has been underestimated. We would therefore like to draw attention to the possibility that IgA nephropathy can recur in transplants with a rapidly progressive course.

References

- Cameron JS: Glomerulonephritis in renal transplants. *Transplantation* 1982;34:237-241.
Diaz-Tejeiro R, Maduell F, Diez J, Esparza N, Purroy A, Pardo J: Loss of renal graft due to recurrent IgA nephropathy with rapidly progressive course: An unusual clinical evolution. *Nephron* 1990;54:341-343.

Honkanen E, Tomroth T, Petterson E, Kuhlback B: Glomerulonephritis in renal allo-grafts: Results of 18 years of transplantations. *Clin Nephrol* 1984;214:210-219.

Glasscock RJ: The glomerulonephropathies; in Schrier RW (ed): Renal and electrolyte disorders. Boston, Little, Brown, 1992, pp 727-806.

Berger J, Yaneva H, Nabarra B, Barbanel C: Recurrence of mesangial deposition of IgA after renal transplantation. *Kidney Int* 1975;7:232-241.

Bachman U, Biava C, Amend W, Feduska N, Melzer J, Salvatierra O, Vincenti F: The clinical course of IgA nephropathy and Henoch-Schönlein purpura following renal transplantation. *Transplantation* 1986;42:511-515.

Mathew TH, Mathews DC, Hobbs JB, Kincaid-Smith P: Glomerular lesions after renal transplantation. *Am J Med* 1975;59:177-190.

114

Streather/Scoble

Crescentic IgA Nephropathy in a Renal Transplant