IgA Nephropathy with Rapidly Progressive Course after Kidney Transplantation

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Dear Sir,

There are few reports on the clinical course of IgA nephropathy after kidney transplantation [1–3], but graft loss due to recurrent disease seems to be rare. Recently, Diaz-Tejeiro et al. [4] have reported a case of IgA glomerulonephritis with rapidly progressive course and only 4 more cases had previously been reported [5]. We present a new case of IgA nephropathy after kidney transplantation with rapidly progressive renal failure and diffuse crescentic proliferation.

The patient was an 18-year-old male who presented with proteinuria, edema and microscopic hematuria in 1977. Percutaneous biopsy showed a membranoproliferative glomerulonephritis, but immunofluorescence stain was not performed. In 1986 he begun chronic hemodialysis treatment. He was transplanted in September 1987 from a cadaveric donor. The donor HLA antigens were A1, A2, B11, DR4 and DR7, and the patient's were A2, B5, B12 and DR7. Initial oligoanuria was present and diuresis started on day 15 after transplantation. Neither acute rejection nor other complication was noted. The serum creatinine stabilized around 1.3 mg/dl (115 mmol/l). The patient was discharged on day 30 after transplantation with prednisone and ciclosporin as immunosuppressive therapy. In February 1988 an increase in serum creatinine was observed (3.8 mg/dl, 336 mmol/l), together with microhematuria and mild proteinuria (680 mg/day). Allograft biopsy disclosed an IgA nephropathy with mesangial proliferation associated with crescent formation in 80% of the glomeruli. Mild tubulointerstitial and vascular lesions were observed. The patient was treated with methylprednisolone (1,000 mg/day) for 3 days without response. Plasmapheresis was performed every other day for a total of 6 treatments (Plasmaflow separator, 2 liters each time). Simultaneously cyclophosphamide (2mg/kg/day) was administered in addition to low doses of prednisone (0.2 mg/kg/day) and ciclosporin A (4 mg/kg/day). Fifteen days later serum creatinine had decreased from 7.8 mg/dl (690 mmol/l) and stabilized around 3.9 mg/dl (345 mmol/l). The patient was sent back home and periodically reviewed. In June 1988 the patient was admitted again to the hospital. End-stage renal failure had developed and serum creatinine value was 13.1 mg/dl (1,159 mmol/l). The patient returned to regular hemodialysis.

It seems very unlikely that our patient developed a de novo IgA nephropathy in the transplanted kidney, although the donor HLA DR antigens were DR4 and DR7 and HLA DR4 might be associated with the disease [6]. Recurrence of a IgA nephropathy must be considered. More than 30 cases have been reported in the literature [5]. It is possible that the initial biopsy of the patient's own kidney missed an IgA nephropathy so that immunofluorescence stain was not performed. Finally, the inadvertent transplantation of kidneys bearing mesangial IgA deposits into recipients without underlying IgA nephropathy has previously been reported [5,7]; but we think that this possibility can be disregarded because the other donor graft has not shown any sign of IgA nephropathy until now. Nonetheless, resolution of the IgA deposits has been noted in some cases [8].

The development of graft failure has been unusual, only 5 of the previously described patients with IgA nephropathy after kidney transplantation have progressed to graft failure [4, 5]. Three of them carried HLA

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B35, which has been considered in some reports to be a genetic marker of poor prognosis in IgA nephropathy [9], but our patient did not carry this HLA B antigen. Neither plasma exchange nor cyclophosphamide seems to be effective in stopping the course of the disease [4].

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