Guillain-Barré Syndrome after Renal Transplantation: A Case of Clinical Success with Intravenous Immunoglobulin Therapy

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Dear Sir,

A 30-year-old man, seronegative for cyto-megalovirus (CMV), received a renal graft from a cadaveric seropositive donor in May 1994. Immunosuppression was prescribed with triple therapy. To prevent primary CMV infection, human cytomegaloglobulin (Cytotect Biotest®) was given, 100 mg/kg i.v. every 2 weeks. He was discharged on day 11 with a serum creatinine of 2.5 mg/dl (221 µmol/l).

The patient remained well until the 20th posttransplant day, when he presented with arthralgias in the lower limbs and malaise. He was afebrile and the physical examination and chest X-ray were normal. The serum creatinine had risen to 11.6 mg/dl (1,025 µmol/l); an ultrasound study of the allograft revealed no fluid collections or evidence of hydronephrosis. A clinical diagnosis of acute rejection was made and a 3-day course of methylprednisolone was administered without any efficacy. On the 7th hospital day a graft biopsy was performed showing typical cytomegalic inclusions in tubular and glomerular epithelial cells, with mononuclear tubulointerstitial infiltration (fig. 1); signs of acute tubular necrosis were also observed. At this time, leukopenia (2,410 cells/mm³) and increased levels of AST (94 U/l, 1.57 µkat/l), ALT (110 U/l, 1.83 µkat/l) and pancreatic amylase (2,351 U/l, 39.2 nkat/l) were detected. Positive titers of IgM CMV antibodies were also observed. Azathioprine was discontinued and the patient was treated with ganciclovir, 1.25 mg/kg/day. The following days there was a drop in creatinine level to the baseline value, but by the 17th hospital day he had developed paresthesias of the left arm, followed by ascending limb weakness. Examination revealed an areflexic tetraparesis and bilateral facial weakness. The spinal fluid was acellular and con-

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Fig. 1. Light microscopy showing typical cytomegalic cells (arrows) in glomerulus. a Severe tubulointerstitial changes are also present. HE $\times$ 125. b HE $\times$ 1,250.

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tained 94 mg/dl protein; glucose and viral titers were normal. Cyclosporine blood levels were within therapeutic range. Weakness progressively worsened and assisted ventilation was required. A peripheral nerve conduction study performed at this moment revealed absence of conduction in fibularis communis, tibialis posterior and medianus nerves.

A diagnosis of Guillain-Barré syndrome (GBS) was made. Intravenous immunoglobulin was started in a dosage of 0.4 g/kg/day for 5 days with rapid improvement of the muscle power, observing spontaneous movements of his feet 5 days latter; a new electro-myograph was then performed revealing improvement of the nerve conduction. Serum creatinine remained stable. After 18 days of ventilatory therapy he was extubated. Finally, he was able to walk unaided 5 weeks after immunoglobulin therapy and was discharged home with a minimal anterior tibial muscle wasting. One year later he remains well and no subsequent relapses occurred.

This patient is of interest because, without receiving OKT3 therapy and in spite of prophylactic cytomegaloimmunoglobulin, primary infection occurred causing a serious CMV disease with leukopenia, hepatitis, allograft dysfunction, pancreatitis and development of severe GBS.

Acute idiopathic demyelinating polyneuropathy, or the GBS, has been reported in renal transplant recipients associated with CMV infection [1, 2] or as a form of cyclosporine A neurotoxicity [3]. The disease can lead to severe quadriplegia, commonly requiring artificial ventilation. Although both plasma exchange and intravenous immunoglobulin are effective therapies in the treatment of GBS, the latter has shown to be as effective as or better than plasma exchange in one randomized trial [4]. This contrasts with results of other uncontrolled series that reported significant GBS relapses after therapy with un-selective immunoglobulin alone [5, 6]. In addition, renal toxicity reported by several authors [7, 8] can be a limiting factor in patients with pre-existing renal impairment. Nevertheless, this approach produced in this patient a rapid improvement in strength, which returned to normal over a 5-week period with no subsequent relapses. Considering the availability of immunoglobulin, its lower price and few side effects compared to plasma exchange, we conclude that it could be the therapy of choice in acute GBS in renal transplant recipients, although further studies are necessary.

References

Bale JF, Rote NS, Bloomer LC, Bray PF: Guillain-Barré-like polyneuropathy after renal transplant: Possible association with cytomegalovirus infection. *Arch Neurol* 1980;37:784.
Donaghy M, Gray JA, Squier W, Kurtz JB, Higgins RM, Richardson AJ, Morris PJ: Recurrent Guillain-Barré syndrome after multiple exposures to cytomegalovirus. *Am J Med* 1989;87:339-341.

Palmer BF, Toto RD: Severe neurologic toxicity induced by cyclosporine A in three renal transplant patients. *Am J Kidney Dis* 1991;12:116-121.

Van der Meché FGA, Schmitz PIM and the Dutch Guillain-Barré Study Group: A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome. *N Engl J Med* 1992;326:1123-1129.

Irani DN, Cornblath DR, Chaudhry V, Bore C, Hanley DF: Relapse in Guillain-Barré syndrome after treatment with human immunoglobulin. *Neurology* 1993;43:872-875.

Castro LHM, Ropper AH: Human immunoglobulin infusion in Guillain-Barré syndrome Worsening during and after treatment. *Neurology* 1993;43:1034-1036.

Barton JC, Herrera GA, Bertoli LF, Work J Koopman WJ: Acute cryoglobulinemic renal failure after intravenous infusion of gammaglobulin. *Am J Med* 1987;82:624-629.

Rault R, Piraino B, Johnston JR, Oral A: Pulmonary and renal toxicity of intravenous immunoglobulin. *Clin Nephrol* 1991;36:83-86.

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