LETTER

A case with acute antibody-mediated kidney transplant rejection and long term follow-up results

Dear Editor,

Antibody-mediated acute rejection (ABMR) occurs in 5-7 % of renal transplant patients; its incidence increases to 40 % in highly sensitized recipients[1]. With optimum treatment, regained renal graft functions could deteriorate, and failure of the graft might occur inevitably in the next years among kidney transplanted patients with acute rejection (AR). Risk factors for AR have been known as presensitization (presence of panel reactive antibody (PRA), donor-specific antibody (DSA), human leukocyte antigen mismatching, blood group incompatibility, long cold ischemia time, and delayed graft function[2].

We present the case of a 50-year-old female with ABMR. Before she received a deceased donor kidney transplantation in June 2014, she had been on peritoneal dialysis for two years due to chronic kidney failure secondary to nephrolithiasis and hypertension. After surgery urine output was immediately seen, her creatinine levels decreased from 6.24 to 1.2 mg/dL on the 5th postoperative day without dialysis. Subsequently, urine output was decreased, and creatinine levels increased while cyclosporine trough levels were 111-240 ng/mL. PRA values increased from 78 % (class 1) and 23 % (class 2) to 90-97 % respectively. DSAs were not searched by then due to lack of kits. Her graft biopsy on posttransplant 8th day revealed ABMR. The patient received three doses of pulse steroid, two doses of anti-thymoglobulin (ATG), and six doses of plasmapheresis with intravenous immune globulin (IVIG). Cyclosporine was switched to tacrolimus (trough level 8-10 ng/mL). Because hemodialysis need was sustained and no recovery was obtained, rituximab was given as rescue therapy. Afterward, urine output increased, and serum creatinine levels decreased to 1.5-1.9 mg/dL without dialysis. Five years she is stable since then and has been followed-up in our clinic with her creatinine levels at 0.9-1.1 mg/dL.

Acute renal allograft rejections are usually presented with fever, fatigue, graft tenderness, oliguric renal failure in the first six months post-transplantation. However, no other clinical finding, except oliguric renal failure, was detected in our case. Also, high pretransplant PRA levels which were probably due to multiple pregnancies increased further after transplantation. Graft biopsy revealed linear C4d accumulation (no C3, C1q, and fibrin deposition) along peritubular capillaries and ABMR was diagnosed. Standard treatment is based on three cornerstones: removal of DSAs from the bloodstream, reduction of DSAs synthesis, and inhibition of the interaction between DSAs and human leukocyte antigen antigens on donor's cells[3]. So treatment strategy includes methylprednisolone 300-500 mg/day for 3-5 days, plasmapheresis plus IVIG (alternate day six doses), change of immunosuppressives to tacrolimus and mycophenolate mofetil. In the literature, reports are demonstrating no improved benefit of rituximab in the treatment of ABMR cases[1]. In our case, both graft and patient survival were obtained with rituximab only. Even though there is lack of evidence to support its routine use in severe ABMR cases, rituximab should be kept in mind for potential rescue therapy.

Key words: Cadaveric kidney transplantation, acute antibody-mediated rejection, rituximab

Conflict of interest

None declared by authors.

References

1. Macklin PS, Morris PJ, Knight SR. A systematic review of the use of rituximab for the treatment of antibody-mediated renal transplant rejection. Transplant Rev (Orlando). 2017; 31: 87-95.
2. Zhao YG, Shi BY, Qian YY, Bai HW, Xiao L, He XY. Clinical efficacy of rituximab for acute rejection in kidney transplantation: a meta-analysis. Int Urol Nephrol. 2014; 46: 1225-1230.
3. Comai G, Ravaioli M, Baraldi O, Cuna V, Gasperoni L, D''Arcangelo GL, et al. Treatment of Acute Antibody-Mediated Rejection. Contrib Nephrol. 2017; 190: 156-167.

Kaynar K[1], Şehit C[2], Ulusoy Ş[1]

[1] Nephrology Department
[2] Internal Medicine Department
Medical Faculty, Karadeniz Technical University, Trabzon, Turkey

Corresponding Author: Prof. Dr. Kubra Kaynar, Karadeniz Teknik Üniversitesi (KTÜ), Tıp Fakültesi, Nefroloji Bilim Dalı, 61080 Trabzon, Turkey, tel: + 905422415879, e-mail: kkaynar@yahoo.com