Kidney Transplant

신장이식에서 항체매개성거부반응

계명대학교 의과대학 내과학교실

한 승 엽

Update on Treatment of Antibody Mediated Rejection

Seungyeup Han

Department of Internal Medicine, Keimyung University School of Medicine, Daegu, Korea

The widespread use of potent and specific immunosuppressive agents has significantly reduced acute cellular rejection rates and substantially improved 1-year graft survival following kidney transplantation. Substantial improvement of long-term outcomes, however, has not been realized. Acute and chronic antibody-mediated rejections (ABMR) are playing an increasingly critical role in kidney allograft loss and are considered among the most important barriers that limit long-term outcomes.

Since 2003, the Banff Working Group classification system for renal allograft biopsies has differentiated T cell-mediated rejection (TCMR) from ABMR. The most recent Banff 2013 diagnosis of ABMR requires histologic evidence of acute or chronic tissue injury, evidence of current/recent antibody interaction with vascular endothelium and serologic evidence of the presence of circulating DSA. Importantly, C4d staining is no longer a requirement for the diagnosis of ABMR.

Despite the important role of ABMR in patient morbidity and mortality after kidney transplantation, our current understanding of the pathogenesis and pathologic phenotypes of ABMR is limited. Evidence supports an important role for DSA in acute and chronic ABMR. However, not all DSA detected by current assays cause injury in the allograft and not all ABMR phenotypes cause rapid allograft failure. Similarly, C4d has significant limitations as a biomarker of ABMR. Standardized risk stratification strategies are needed to better define preventive and treatment approaches for each ABMR. New assays and molecular tests may be considered as diagnostic and prognostic tools in patients with ABMR.

The management of ABMR is challenging and is associated with poorer outcomes compared with traditional anti-T cell rejection therapy for pure TCMR. Uncontrolled or controlled nonrandomized studies support a role for rituximab, bortezomib, plasmapheresis and IVIG. However, the relative importance of these therapies is difficult to assess because treatment strategies were not standardized, doses and frequencies were not similar, and the specific drugs were combined with other agents. Many of the potential treatment options for ABMR have been imported from other areas of medicine, without appropriate clinical trials in kidney transplantation. Before novel and more effective treatments become available, the close monitoring of high-risk patients and an emphasis on adherence to well-tolerated maintenance immunosuppressants are recommended to minimize the risk of ABMR.