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Session Title : Update in ABO-incompatible kidney transplantation

Mechanism of Accommodation in ABO incompatible kidney transplantation

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ABO-incompatible transplantation is one of the strategies employed to overcome the shortage of donor organs. However, the production of serum antibodies against the donor's blood group A or B antigens by the recipient is the key factor to the success of this transplantation strategy. Many reports suggest that the immunobiological state after ABO-incompatible kidney transplantation (ABOi KTx) represents an accommodation through which rejection does not occur clinically despite the presence of antigens on the graft's vascular endothelium and the presence of antibodies in the blood of recipients. A number of intertwined factors are responsible, including ABO blood group antigens in the graft, antibody production, complement control, and immunosuppressive drugs. However, the mechanisms are fully unknown. We previously reported that the synthesis of ABO antigens is maintained in the renal grafts for long-periods after transplantation. Although the characteristics of antibody production after ABOi KTx were critical factors for AMR, there were no reports describing the long-term B cell immunobiology after ABOi KTx. Our data showed most of recipients maintained very low antibody titers specifically against donor blood type after ABOi KTx. In vitro study showed that they did not or less respond persistently against the RBCs with the same donor blood group antigens, although they continuously produced antibodies against nondonor blood group antigens in the recipients of type O. These findings suggest that the accommodation after ABOi KTx could occur due to a downregulated B cell response against the donor blood group antigens. However, some patients continued to have antibodies against RBCs of the same donor blood type without developing antibody-mediated rejection (ABMR) after the ABOi KTx. Although the ABO antigens are similar in RBCs and organs, the details are not identical. Recently, we developed a new tool to exam antibody titer, which mimics the ABO blood group antigens on the renal endothelial cells. We found that antibody detected by the isohemagglutinin assay by using RBCs after ABOi KTx with stable graft function were hyporeactive to ABO antigens of renal endothelial cells. Donor-specific down-regulation of the capability to produce antibodies was acquired in ABOi KTx after stabilization of renal graft function.