Keynote Lecture I

The art of performing ABO-incompatible liver transplantation

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ABO-incompatible (ABO-I) liver transplantation is an alternative to living-donor liver transplantation (LDLT). The ABO barrier in kidney transplantation fell rapidly thanks to Professor Alexandre's pioneering efforts. However, considering the miserable outcomes for liver transplantation in general, ABO-I liver transplantation was initially limited to highly select patients. Many technical innovations were accomplished in the field of living-donor liver transplantation (LDLT) in Japan. As one of the innovations, strategies to prevent antibody-mediated graft rejection (AMR) after ABO-I LDLT were developed and established. Increases in the safety of ABO-I LDLT expanded ABO-I LDLT primarily to Asia, where LDLT is the predominant form of LT owing to the scarcity of brain-dead donors, and where ABO-I LDLT now accounts for approximately 20% of all LDLT procedures. The desensitization protocol consisting of rituximab, plasma pheresis, tacrolimus, and mycophenolate mofetil prior to LDLT, followed by standard immunosuppression, is currently the best option in terms of safety and efficacy. There was no negative impact of rituximab on the recurrence of hepatocellular carcinoma. The feasibility of rituximab for LDLT for acute liver failure and the necessity of desensitization for children older than 1-year-old LDLT have been documented. Immunological accommodation after ABO-I transplantation could be provided by immune factors in both the donor and recipient grafts. Despite effective desensitization protocol including rituximab, once the process of AMR is initiated, rituximab is ineffective. Early diagnosis of AMR through liver biopsy, followed by treatment involving a steroid pulse, plasma pheresis, and IVIG, is practical. Plasma cell–depleting agents, such as the proteasome inhibitor bortezomib, may be used in ABO-I LT recipients with caution. In addition to decreasing the amount of antibody, a treatment for decreasing or eliminating post-transplantation inflammation might ameliorate the epithelial injury that leads to fatal AMR.