Abstract

Antibody-mediated rejection (AMR) is the main cause of the kidney graft dysfunction and its failure after transplantation. Antibodies lead to vascular damage that is either acute or chronic and manifests as sudden or progressive graft dysfunction. Risk factors for development of AMR are time spent on haemodialysis, retransplantation, previous sensitisation against HLA antigens, and persistence of panel-reactive antibodies. Diagnosis is based on detection of deposits of C4d component of complement in peritubular capilaries and presence of donor-specific antibodies (DSA). We can also observe injury caused by antibodies against non-HLA antigens without detection of anti-HLA DSA. Use of „molecular microscope“ can be beneficial in diagnosis and stratification of the risk of graft failure. High expression of ENDAT (endothelial activation and injury transcript) improves prediction of kidney graft failure more than C4d staining. Based on gene expression, the AMR scoring system correlates with the diagnosis of AMR and predicts graft loss in the future.

The main goal of our work was to recognize patients at risk of AMR. In our study, we confirmed the efficacy and safety of acute AMR therapy with plasmaphereses and administration of intravenous immunoglobulins for improving outcomes of kidney transplantation. In case of acute resistant AMR, we used a protocol with administration of bortezomib and rituximab, whereby we improved mid-term outcomes of kidney graft survival.

In our study, we have shown that patients with low levels of HGF and higher levels of sCD30 before transplantation have high risk of developing AMR. We further evaluated the significance of soluble BAFF in concurrent antibody and cell-mediated rejection, where BAFF could be a beneficial marker of ongoing AMR.