Effect of Plasmapheresis on Angiotensin II Type I Receptor Antibody

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The presence of angiotensin II type I receptor (AT1R) antibody has been associated with renal allograft rejection. We determined the presence of AT1R antibody in an ELISA assay (CellTrend GmbH Luckenwalde, Germany) in sequential samples from 16 patients who underwent pre-emptive, alternate day plasmapheresis and low dose anti-CMVIg desensitization to allow transplantation across donor HLA specific antibody (DHSA).

Nine patients were treated under a standard desensitization protocol consisting of 1 to 5 plasmaphereses (PP) pre-transplant and 1-5 pp post-transplant (total range = 3-7 PP). DHSA decreased with PP in the standard treatment group and remained below a level consistent with a positive flow cytometric crossmatch during the study period. Seven patients received extended treatments (>8 PP) due to DHSA rebound between the PP treatments.

Tests of samples collected prior to PP treatment showed 4 patients had very high levels of AT1R antibody (>40unit/ml); 2 had intermediate levels (17-40units/ml); 3 were borderline positive (10-17units/ml) and 7 were negative (<10units/ml). Tests of samples collected immediately post-treatment showed that AT1R antibody decreased with PP in 15/16 (94%) patients. However, in the standard treatment group, the AT1R antibody rebounded by 2-6 months in all 3 patients whose initial AT1R antibody was >40unit/ml. At rebound, for 2 of these 3 patients, the AT1R antibody returned to the initial level (>40unit/ml), while the DHSA remained low. These two patients experienced AMR, and 1 graft was lost due to rejection.

In the extended treatment group, continuous PP depleted both DHSA and AT1R antibody and the AT1R antibody remained negative (<10 units/ml) in 2-6 months after desensitization for all 7 patients. Of 3 patients who had sera obtained prior to increase of DHSA, all 3 showed an increase in AT1R antibody prior to the increase of DHSA.

These data indicate that plasmapheresis is effective in depleting AT1R antibody; however, among patients with initial high levels of AT1R antibody, a rebound may occur after treatment and can potentially lead to graft rejection. The risk of AT1R antibody rebound correlates with the initial antibody strength prior to desensitization treatment. Further, these preliminary data suggest that a rebound of ATIR antibody may precede a rebound of DHSA