Oral Abstracts

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545 ASSOCIATION BETWEEN DE NOVO DONOR SPECIFIC HLA ANTIBODY, C4D STAINING IN RENAL GRAFT BIOSY, AND GRAFT OUTCOME
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Background: Despite improved early graft survival, long-term kidney graft loss rate has not been reduced. Both HLA antibodies and HLA donor specific antibodies (DSA) have been detected and may be causal in chronic allograft nephropathy (CAN) and, especially, in acute rejection. We evaluated the association between DSA, C4d staining in transplant and graft outcome.

Materials and methods: During Sep 2004-Aug 2007, HLA antibody testing using the Luminex laboratory screen assay system were performed in 348 recipients. Of these, C4d deposition was assessed in the biopsies of 69 recipients with transplant dysfunction.

Results: In the 69 cases, 29 (42%) showed C4d negativity, 27 (39%) were C4d positive, 6 (9%) were equivocal, and 6 (9%) not diagnostic. Forty-nine (71%) with transplant dysfunction had HLA antibodies and 41 (59%) had DSA. The proportion of C4d positivity was significantly higher in patients with DSA (class I only, II only, and I & II) in comparison with patients without post-transplant HLA antibodies (Table). The incidence of graft failure (including current ScC+4.0) in patients with class II antibodies (class II only or I & II) was significantly higher than that in patients without post-transplant HLA antibodies (Table). In figure, grafts of class II DSA group continued to fail beyond 2 years after transplantation when compared with other 2 groups (None/NDSA or Class I only), however, the difference in graft survival between class II and None/NDSA group did not reach statistical significant level (log-rank P=0.32).

Conclusions: Significant association between C4d staining, de novo HLA Class II antibodies and graft failure strongly suggests the importance of post-transplant HLA antibodies. We propose that amelioration of CAN graft loss depends on monitoring and identification of DSA and appropriate immunosuppression of these antibodies.

Table. C4d positivity and graft outcomes according to DSA groups

<table>
<thead>
<tr>
<th>Antibody Category</th>
<th>N</th>
<th>C4d Positive N (%)</th>
<th>Graft Failure N (%)</th>
<th>Graft Failure or ScC+4.0 N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None (ref)</td>
<td>20</td>
<td>1 (5.0%)</td>
<td>3 (15.0%)</td>
<td>5 (25.0%)</td>
</tr>
<tr>
<td>Class I only</td>
<td>15</td>
<td>8 (53.3%) (P&lt;0.001)</td>
<td>2 (13.3%)</td>
<td>5 (33.3%)</td>
</tr>
<tr>
<td>Class I &amp; II</td>
<td>22</td>
<td>13 (59.1%) (P&lt;0.001)</td>
<td>9 (41.1%) (P=0.06)</td>
<td>13 (59.1%) (P&lt;0.03)</td>
</tr>
<tr>
<td>NDSA</td>
<td>9</td>
<td>9 (100%) (P&lt;0.001)</td>
<td>4 (44.4%) (P=0.09)</td>
<td>6 (66.7%) (P=0.03)</td>
</tr>
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</table>

Figure. Graft survival according to DSA groups

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547 CHRONIC TRANSPLANT GLOMERULOPATHY – CLINICAL AND PATHOLOGICAL CORRELATES
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Introduction: Chronic transplant glomerulopathy is one of the leading causes of severe post-transplant proteinuria and graft loss. It may develop in a response to several different injurious processes, such as humoral rejection or thrombotic microangiopathy. In the majority of cases the etiology is obscure, which excludes the possibility for targeted intervention. Our current knowledge about risk factors for the development of TG, as well as factors affecting its dynamics and prognosis is poor.

The aim of the study was to find pathological and clinical risk factors and correlations of TG as well as parameters that influence the survival of grafts with that pathology.

Materials and methods: We retrospectively reevaluated all 86 kidney transplant cases with TG that have been recognized on the basis of indication