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Microvascular Inflammation Predicts Poor Graft Survival in Kidney Transplant Recipients with HLA-DR/DQ Antibodies

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Objectives : The presence of donor-specific antibodies (DSAs) against HLA class II molecules, particularly HLA-DR and HLA-DQ, is a well-established risk factor for antibody-mediated rejection (ABMR) in kidney transplantation. However, the prognostic impact of microvascular inflammation (MVI) in this subgroup remains incompletely understood
Methods : We retrospectively analyzed 270 kidney transplant recipients with confirmed HLA-DR and/or HLA-DQ DSAs. The purpose of our study is to compare graft survival, histological findings, and clinical outcomes between the presence and absence of microvascular inflammation, defined as a g + ptc score of 2 or greater according to the Banff 2022 criteria.
Results : Patients with both HLA-DR/DQ DSAs and MVI demonstrated significantly poorer graft survival compared to those without MVI (0% vs. 95.3%, $P < 0.01$). The presence of MVI was independently associated with a higher incidence of chronic active ABMR and a progressive decline in graft function, compared to patients with no ABMR or with non-classical ABMR phenotypes (probable ABMR or isolated MVI) ($P < 0.01$). Notably, HLA-DR and HLA-DQ antibodies were significantly associated with MVI-positive cases, whereas HLA class I antibodies were not. Identified risk factors for MVI included: Nadir tacrolimus (FK) level < 6 ng/mL ($P = 0.012$), Mean tacrolimus level < 7 ng/mL ($P = 0.006$), and high intrapatient variability (IPV > 13%) ($P = 0.039$). In contrast, high total eplet mismatch (>17) and high class II antibody titer (>7 MFI) were not significantly associated with MVI.
Conclusions : Microvascular inflammation is a strong predictor of adverse graft outcomes in kidney transplant recipients with HLA-DR/DQ antibodies. These findings underscore the importance of integrating histopathological and immunological data for effective risk stratification and personalized management in clinically stable kidney transplant recipients. Our study also suggests that distinct MVI phenotypes may reflect different underlying pathophysiology, warranting exploration of novel therapeutic strategies for ABMR.