KSN 2018
Seoul, Korea · May 17 - 20

Abstract Type : Poster
Presentation No. : PTL 007

ABO-incompatibility lower incidence of DSA-induced antibody-mediated rejection.

Dong Ryeol Lee[1], Byung Chang Kim[2], Jung Myung Ahn[1], Mi Young Jeon[3]
[1]Department of Internal Medicine-Nephrology, Maryknoll Medical Center, Korea, Republic of
[2]Department of Laboratory Medicine, Maryknoll Medical Center, Korea, Republic of
[3]Department of Pathology, Maryknoll Medical Center, Korea, Republic of

Objectives: Donor-specific anti-HLA antibody and Anti-ABO antibody are a big challenge for successful kidney transplantation. Limited data are available on how existences of both DSA and anti-ABO antibody have an influence on the development of AMR. A study showed the lower incidence of DSA-induced chronic AMR in ABO-incompatible kidney transplantation which suggested anti-ABO antibody lower DR expression and de novo DSA production. Purpose of our study is to validate clinical effects of ABO-incompatibility on the incidence of DSA-induced AMR.

Methods: We examined 206 stable kidney transplant recipients (175 ABO compatible-KTRs and 31 ABO incompatible-KTRs) for development of DSAs from June 2013 to June 2017. We biopsied 34 recipients who had DSAs on Luminex PRA. We compared clinical outcomes and incidence of DSA-induced AMR in between ABO-incompatible KT and ABO-compatible KT.

Results: 34 of 206 stable KTRs (16.5%) had PRA-DSAs, Median time of DSA occurrence was 5.3(0.1-21.4) year of post-transplantation. Median peak MFI level was 1514.5(151-17453). The incidence of AMR was 6.8 %(14of206). Compared with ABO-compatible KTR, incidence of DSA occurrence was significantly higher (41.9%, 13of 31 vs. 12.0%, 21of 175, P<0.001) and earlier (2.1±2.1year vs. 9.4±6.3year, P=0.001) after transplantation, however, DSA-induced AMR development was significantly lower in ABO-incompatible KTR (15.4%, 2of 13 vs. 57.1%, 12of 21, P=0.018). HLA mismatch number (3.93±1.5 vs. 3.2±1.4, P=0.007), female donor (64.5% vs.39.1%, P=0.008) and living unrelated donor rate (76.7% vs. 30.2%, P=0.001) were significantly higher in ABO-incompatible KTR.

Conclusions: Our study showed that DSA occurrence was earlier and higher rate in ABO-incompatible kidney transplantation, even after ABO-incompatible KTR were desensitized with PP, low dose IVIG, and Rituximab. However, the lower incidence of DSA-induced AMR was significantly in ABO-incompatible KTR suggested that ABO incompatibility may have a possible protective effect against the development of AMR. Our study needs to be verified.