Development of de novo donor specific antibody (DSA) against human leukocyte antigens (HLA) antibodies became a hot issue in kidney transplantation (KT) because it causes not only acute antibody mediated rejection (AMR) but also chronic active AMR which is a major cause of late allograft failure.¹

Jung et al.² screened de novo DSA in 167 KT recipients for 32 months, and found 13 (9.6%) recipients with DSA positivity. These patients were characterized by more acute AMR and less tacrolimus trough level than DSA negative recipients during the first 0–2 months after KT. Therefore, they recommended the regular monitoring of DSA and maintaining proper tacrolimus level during the initial period after KT to prevent AMR.

This paper stressed the importance of de novo DSA on predicting acute AMR, but not all DSAs are harmful to graft function.³ In addition, clinical significance of DSA strength measured with Luminex assay still controversial.⁴ Therefore, presence of DSA should be cautiously interpreted with clinical status, pathology finding and graft function.

Monitoring of DSA becomes routine work in clinical practice, but the real world of DSA is more complex than we think. Further studies for defining precise characteristics and clinical significance of DSA are required in KT.

REFERENCES


