

Early change in estimated GFR and renal outcome in patient with chronic kidney disease

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Background/Aims: Many studies have evaluated the usefulness of creatinine-based eGFR and cystatin-C-based eGFR at a time for predicting the risk of chronic kidney disease (CKD) progression. However, there are few data comparing prognostic power between creatinine-based eGFR slope (eGFRcre slope) and cystatin-C-based eGFR slope (eGFRcys slope). This study compared the performance of eGFRcre slope with that of eGFRcys slope in identifying a high-risk group of progression to end-stage renal disease (ESRD) in patient with CKD. **Methods:** From October 2012 to November 2016, patients who had simultaneous measurements of serum creatinine and cystatin-C more than 3 times for 1 year were identified. We calculated baseline eGFR values and first-year eGFR slopes using CKD-EPI equation and linear regression analyses. The patients with baseline eGFR ≥ 60 ml/min/1.73m² were excluded. We defined a rapid progression as eGFR slope < -5 mL/min/1.73m²/year. Primary outcomes were progression to ESRD (defined as initiation of dialysis or kidney transplantation). **Results:** Total 1330 patients were included. Thirty-four% of subjects had diabetes. Baseline eGFRcre were 39 (28 - 48)ml/min/1.73m² and eGFRcys were 38 (27 - 50) ml/min/1.73m². During follow-up of 2.4 (1.5 - 3.2) years, 94 (7%) events occurred. Both eGFRcre slope and eGFRcys slope were associated with higher risk of ESRD independently of baseline eGFR (HR=0.986 [0.980 - 0.992], HR=0.989 [0.983 - 0.996], respectively). Both creatinine and cystatin-C based rapid progression were associated with increased risk of ESRD (HR=2.58 [1.71 - 3.90], HR=1.99 [1.33 - 2.98], respectively). In subgroup analyses of rapid progression group by creatinine (N=509), eGFRcys slope was not associated with risk of ESRD (HR=0.97 [0.88 - 1.08], $p=0.62$). Whereas, eGFRcre slope contributed to further discrimination of higher risk of ESRD in subjects with rapid progression by cystatin-C (N=469) (HR=0.90 [0.83 - 0.97]; $p=0.007$). **Conclusions:** These findings demonstrated that both eGFRcre slope and eGFRcys slope were associated with future ESRD risk. eGFRcre slope can be a better predictor of renal outcome than eGFRcys slope in patients with CKD.

Clinical Significance of De Novo Donor-Specific anti-HLA Antibodies after Kidney Transplantation

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Background: The goal of this study was to investigate the impact of de novo donor-specific anti-HLA antibodies (DSA) on antibody-mediated rejection (AMR) and characterize DSA leading to AMR in kidney transplant recipients (KTRs). **Methods:** We included 174 KTRs without pretransplant anti-HLA antibodies. All the enrolled KTRs were prospectively screened for the development of de novo DSA every 3 months before 1 year posttransplant and annually thereafter. DSA were determined by Luminex assays and expressed as mean fluorescence intensity (MFI). AMR was diagnosed by indication biopsy of allograft. **Results:** Of 174 KTRs, 17 KTRs (9.8%) developed de novo DSA during a mean follow-up of 32.3 \pm 13.5 months. The average time to first detection of de novo DSA was 28.2 months after kidney transplantation. All of de novo DSA were against class II antigens (11 DQ, 1 DR, and 5 both). The mean number of DSA was 1.8 \pm 1.2, ranging from 1 to 5 and mean MFI of DSA was 7277.6 \pm 5320.4. Acute AMR occurred only in KTRs with de novo DSA compared to KTRs without de novo DSA (17.6% versus 0%, $p=0.001$). In the KTRs with acute AMR, allograft biopsy was performed 108.3 \pm 95.5 days after development of de novo DSA. Allograft survival was not different between two groups. There were no differences in the number of DSA and mean MFI of DSA between de novo DSA positive KTRs with AMR and de novo DSA positive KTRs without AMR. **Conclusions:** Regular immunological monitoring is needed in KTRs to detect DSA because de novo DSA increased incidence of acute AMR. Close clinical monitoring and prompt interventions are mandatory in KTRs with de novo DSA, regardless of the number and strength of DSA, to reduce allograft damage induced by AMR. **Key words:** Antibody-mediated rejection, de novo donor-specific antibodies, kidney transplantation, survival