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Urinary Biomarkers at 1 Year Post-Transplant Identify Subclinical Structural Injury and Long-Term Graft Function

Hojin Lee, Jin Young Yu, Minhyung Kim, Tai yeon Koo, Se Won Oh, Sang-Kyung Jo, Myung-Gyu Kim
Department of Internal Medicine-Nephrology, Korea University Anam Hospita, Korea, Republic of

Objectives : Protein tyrosine phosphatase 1B (PTP1B) has recently been identified as a central regulator of the ER stress–oxidative stress–inflammatory axis in ischemic kidney injury. However, its clinical relevance in kidney transplant recipients (KTRs) remains unknown. We investigated whether urinary PTP1B measured at 1 year post-transplantation reflects subclinical graft injury and predicts long-term graft function. Liver-type fatty acid-binding protein (L-FABP) and uromodulin were also measured for comparison.
Methods : Urine samples were collected at 1 year post-transplant from 80 KTRs enrolled in the KNOW-KT cohort. Urinary PTP1B, L-FABP, and uromodulin were quantified by ELISA and normalized to urine creatinine. Recipients were stratified by median biomarker levels. eGFR was assessed annually up to 9 years post-transplant. Graft outcomes and histopathologic findings from protocol or indication biopsies were compared.
Results : Recipients with high urinary PTP1B levels had significantly lower eGFR at year 2. Although differences attenuated thereafter, this early divergence suggests that elevated PTP1B reflects ongoing inflammatory injury preceding overt graft dysfunction. Consistently, the PTP1B high group showed increased interstitial inflammation on biopsy. Recipients with high urinary L-FABP at 1 year demonstrated persistently lower eGFR at years 2, 4, 6, and 8, and exhibited greater tubular atrophy on biopsy, consistent with proximal tubular structural injury. Urinary uromodulin was not associated with longitudinal eGFR differences but was linked to higher glomerulosclerosis scores, suggesting an association with chronic structural remodeling. Kaplan–Meier analyses showed no significant differences in graft failure or overall survival between groups.
Conclusions : Urinary PTP1B measured at a clinically stable time point identifies ongoing inflammatory injury and early functional decline in kidney transplant recipients. Together with L-FABP and uromodulin, PTP1B reflects different aspects of graft injury. These findings extend mechanistic insights on PTP1B to the transplant setting and support its potential as a non-invasive biomarker of subclinical graft injury.