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Pathological Tau Accumulation Contributes to Tubulointerstitial Injury and Fibrosis in Chronic Kidney Disease

Seong Woo Lee¹, Se Hun Park², Su Bin Lee², Jina Lee², Eun Young Lee¹

¹Department of Internal Medicine, Soonchunhyang University Cheonan Hospital, Korea, Republic of

²Department of Biomedical Science, Graduate School of Soonchunhyang University, Korea, Republic of

Objectives : Tau, a microtubule-associated protein, implicated in neurodegeneration, undergoes pathological post-translational modifications that disrupt cellular homeostasis. Although tau is expressed in the kidney, its role in chronic kidney disease (CKD) remains unclear. We investigated whether renal tau dysregulation contributes to tubular injury and fibrosis in CKD.

Methods : Publicly available single-cell RNA-seq data (GSE183276) were reanalyzed to assess MAPT mRNA expression across kidney cell types in control and CKD human samples. Tau protein levels in human and murine CKD kidneys were evaluated by immunohistochemistry and immunoblotting. Experimental CKD models included unilateral ureteral obstruction (UUO), folic acid-induced CKD (FA-CKD), and adenine diet-induced CKD (AD-CKD). Renal function and histologic fibrosis were assessed. Total tau (t-Tau), phosphorylated tau (Ser396), and acetylated tau (ac-Tau) were quantified. P301S human tau transgenic mice underwent AD-CKD induction to determine causality. Therapeutic efficacy was evaluated using an ac-Tau-specific antibody (50 mg/kg, intraperitoneally).

Results : Human single-cell RNA-seq data demonstrated that MAPT mRNA was broadly distributed across renal cell types in control and CKD kidneys, with predominant expression in proximal tubular epithelial cells. Renal tau protein levels were increased in CKD patients and in UUO, FA-CKD, and AD-CKD mice compared with respective controls. Elevated t-Tau, p-Tau, and ac-Tau levels were associated with tubular injury and interstitial fibrosis. In AD-CKD mice, tau accumulation increased over time and correlated with tubular dilatation and fibrotic area ($p < 0.001$). Compared with wild-type AD-CKD mice, P301S mice exhibited aggravated renal dysfunction, increased α -SMA expression, and enhanced oxidative DNA damage (8-OHdG) ($p < 0.01$). In contrast, ac-Tau antibody treatment attenuated renal injury, reduced fibrotic markers (α -SMA, COL1A1), decreased oxidative stress, and preserved AQP1-positive tubules ($p < 0.01$).

Conclusions : Renal tau dysregulation contributes to tubular injury, oxidative stress, and fibrotic remodeling in CKD. Targeting pathological tau species may represent a novel therapeutic strategy for CKD progression.