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Recent update in the pathogenesis of CKD

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Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function for a duration of more than 3 months with implications for patient health. The prevalence of CKD is estimated approximately to be 8~16% worldwide. Regardless of initial injury, glomerulosclerosis, interstitial fibrosis, and tubular atrophy are common final pathologic features of CKD. Renal fibrosis, a main pathogenic contributor to progressive CKD, is a multi-factorial and multi-cellular process that is orchestrated by many different resident and infiltrating cells in the kidney and by many different mediators and cellular processes. Several epidemiologic studies have revealed that acute kidney injury (AKI) is an important risk for CKD. Moreover, experimental studies have suggested the pathogenic mechanism of AKI to CKD transition which is associated with maladaptive repair of the proximal tubular epithelial cells (TECs) and progressive interstitial fibrosis. The mechanisms include cell cycle arrest and cellular senescence, profibrotic cytokine secretion, pericyte activation with myofibroblast generation, inflammation, loss of peritubular capillaries, and production of extracellular matrix.

Epithelial-mesenchymal transition (EMT), a process in which injured renal TECs undergo a phenotype change, acquiring mesenchymal characteristics and transitioning into fibroblasts, is initially known as a critical mechanism of renal fibrosis underlying CKD. However, controversy on the relative contribution of EMT to the total fibroblast or myofibroblast in the fibrotic kidney lasted for years. On the other hand, pieces of evidence have suggested partial EMT as a new mechanism of renal fibrosis, in which injured renal TECs acquire some of the phenotypic characteristics of fibroblasts rather than converting to a complete fibroblastic phenotype, and dedifferentiated TECs remain attached to the basement membrane after renal injury.

In this lecture, recent update in the pathogenesis of CKD focusing on tubular injury and repair, the origin and activation of renal myofibroblasts, and mediators of CKD progression will be presented.

References

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