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Aging and acute kidney injury

Myung-Gyu Kim
Korea University Anam Hospital, Korea, Republic of

As the population ages, the incidence and prevalence of chronic diseases such as diabetes, hypertension, cancer and chronic kidney disease (CKD) are growing rapidly. Several epidemiological studies have reported that acute kidney injury (AKI) is more frequent in the elderly and they often progress to CKD/ESKD. However, this relationship mostly comes from epidemiological studies of elderly patients with comorbid conditions, and the mechanisms responsible for poor renal outcome in elderly have not been thoroughly investigated.

Aged kidneys show various structural and functional changes, and disturbance of autoregulation/hemodynamic. These aging-related phenotypes may contribute to poor renal outcomes in elderly. Chronic low-grade inflammation, referred to as "inflamm-aging" is an important phenotype of elderly, and has been reported to be observed in aged kidneys. Recently Sato Y et al. showed that aged mice developed multiple tertiary lymphoid tissues (TLTs) in the kidney after AKI, suggesting the possible contribution of TLT to persistent chronic inflammation and impaired recovery in aged mice. Kim MG et al. also demonstrated that persistent M1 predominant inflammation partially driven by senescent tubule cells with cell-cycle arrest may lead to an accelerated progression to CKD after AKI in elderly.

As the mechanisms of aging are uncovered, clinical and basic researches related to aging will contribute to clarifying the mechanisms that mediate poor outcomes of AKI in the elderly population. Further, strategies that aim to change aging-induced injury responses, such as inflamm-aging, may be key to the treatment of elderly patients with AKI and CKD in the future.