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Tertiary lymphoid tissues: unique microenvironment in injured kidney

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Acute kidney injury (AKI) is a common clinical condition associated with high mortality and expensive medical cost. Although kidney function recovers in approximately half of cases of AKI suggesting the existence of intrinsic repair capacity, 10 % of remaining cases die within one month, and the other 40% of cases progress to chronic kidney disease (CKD) or end-stage renal disease.

Recently, we identified a unique microenvironment orchestrating kidney injury and repair after AKI. Tertiary lymphoid tissues (TLTs), which mainly consist of lymphocytes and fibroblasts, are formed in the chronic phase after AKI in aged kidneys, contributing to prolonged inflammation and delayed regeneration (Sato Y et al., JCI Insight 2016). Resident fibroblasts in the kidney play critical roles in the formation of TLTs by acquiring the abilities to produce retinoic acid and homeostatic chemokines, and to support B cell area formation. Notably, therapeutic strategy targeting TLTs attenuate histological injury and fibrosis in mouse models.

Furthermore, TLTs are commonly found in human kidneys, and the presence of advanced-stage TLTs correlates well with the severity of kidney injury and inflammation (Sato Y et al., Kidney Int 2020). Specifically, TLTs are commonly found in protocol biopsies of transplanted kidneys, and advanced-stage TLTs are associated with progressive graft dysfunction (Lee YH et al., in revision).

Understanding the mechanism of TLT formation may provide clues to the development of therapeutic strategies to halt the progression of kidney diseases.