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Comparison of murine renal ischemia-reperfusion injury models for identifying adequate model of repair phase of ischemic acute kidney injury

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Objectives: Murine models are essential for unraveling mechanisms of both injury and repair of ischemic acute kidney injury (AKI) as well as AKI to chronic kidney disease (CKD) transition. Adequate murine models for investigating the repair process of ischemic AKI are yet to be determined. We aimed to identify the most adequate murine ischemia-reperfusion injury (IRI) model for investigating the recovery phase of ischemic AKI.

Methods: Sham, bilateral IRI (BIRI), and unilateral IRI (UIRI) operations were performed in nine-week-old male C57BL/6 mice. Serial changes in renal function, infiltration of kidney mononuclear cells, and intrarenal cytokines / chemokines were analyzed for 4 weeks.

Results: Plasma creatinine and BUN increased in the BIRI group and remained elevated compared to the control (sham operation) and UIRI groups until seven days after surgery. In contrast, plasma creatinine and BUN of the UIRI group were comparable with those of the control group. Effector memory $CD4^+$ / $CD8^+$ T cells and activated $CD4^+$ / $CD8^+$ T cells markedly increased in the postischemic kidneys in both IRI groups. Expressions of proinflammatory cytokines and chemokines were enhanced in the postischemic kidneys of both IRI groups. Intrarenal proinflammatory micromilieu was persistent until four weeks after BIRI, even though renal function was fully recovered after two weeks. The postischemic kidneys of the UIRI group showed more prominent earlier proinflammatory condition than the BIRI group and atrophic changes at 4 weeks after surgery.

Conclusions: Our study showed that the degree and timing of proinflammatory process were different depending on the IRI models, suggesting that BIRI and UIRI models may be adequate for studying the repair phase of ischemic AKI and AKI to CKD transition, respectively.