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Outcomes and possible mechanism of acute tubulointerstitial nephritis

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Objectives: Acute tubulointerstitial nephritis (ATIN) is an important cause of acute kidney injury, but its outcomes and underlying mechanisms remain unresolved.

Methods: A total of 75 patients who had biopsy-proven ATIN but did not have primary glomerulonephritis were retrospectively reviewed between 2001 and 2017 from two tertiary referral centers. As a primary outcome, renal recovery was defined as a decline $\geq 50\%$ of peak creatinine or ≤ 1.4 mg/dL of subsequent creatinine. Additionally, the risk of end-stage renal disease (ESRD) was tacked after biopsy. Plasma and urine inflammatory cytokines at the time of biopsy were analyzed using a multi-analyte flow assay kit (n=34).

Results: 70 cases (93.3%) were idiopathic ATIN; others were related with autoimmune diseases. At 6 months post-biopsy, 69% of patients achieved renal recovery and 12% of patients needed renal replacement therapy. During the median follow-up period of 3 years (maximum 19 years), the groups with diabetes mellitus, chronic kidney disease, and needing dialysis at the time of diagnosis had a higher risk of low renal recovery and ESRD than the counterpart groups. However, steroid use did not affect the alteration of these outcomes. Among several inflammatory cytokines, monocyte chemotactic protein 1 and interleukin-8 levels were markedly elevated in both plasma and urine; and interleukin-18 levels were high in plasma alone.

Conclusions: Baseline kidney status, not current therapeutic approach, affects the outcomes of ATIN. The present cytokine results will be helpful to develop a novel targeting therapy for ATIN.