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Protective effect of alpha 1-antitrypsin on renal ischemia-reperfusion injury in a mouse model

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Objectives: Alpha1-antitrypsin (AAT) is an important protein in the anti-inflammatory response that functions to regulate the activity of serine proteinases. We aimed to evaluate the protective effect of AAT on ischemia-reperfusion injury (IRI) in a mouse model.

Methods: We investigated the effects of AAT in a C57BL/6 mouse model of IRI by dividing them into four groups: normal control, sham operated, ischemia-reperfusion (IR), and IR after AAT pretreatment (IR-AAT). In the IR-AAT group, mice were pre-treated with AAT (80 mg/kg/day) for 3 days before renal ischemia was induced by clamping the bilateral renal vascular pedicles for 30 min. At 24 hours after IRI, biochemistry, histology, inflammatory cytokines, and apoptosis were assayed.

Results: Blood urea nitrogen and serum creatinine levels were significantly lower in the IR-AAT group than in the IR group. NGAL and KIM-1 protein levels were significantly lower in the IR-AAT group than in the IR group. In addition, there were fewer tubular injuries and less interstitial fibrosis in the IR-AAT group than in the IR group, and the expression levels of TGF-β, IL-1β, and IL-6 were significantly lower in the IR-AAT group than in the IR group. When compared to the IR group, there were fewer TUNEL assay-positive cells, lower caspase-3 activity and Bax, and higher Bcl-2 in the IR-AAT group.

Conclusions: AAT preserved renal function, attenuated tubular injuries and interstitial fibrosis, and inhibited inflammation and apoptosis after renal IRI. Our results suggest that AAT has protective effects against renal IRI by inhibiting inflammatory and apoptosis pathways.