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Organ crosstalk and cellular apoptosis during acute kidney injury

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Acute kidney injury (AKI) is a common and often catastrophic complication among hospitalized patients, and despite advancements in renal replacement therapy (RRT), mortality rates remain unacceptably high. Kidney ischemia-reperfusion injury (IRI) activates the host innate and adaptive immune response, releasing cellular and soluble mediators which drive distant organ dysfunction. Organ crosstalk occurs via the activation of pro-inflammatory and pro-apoptotic pathways, and recent experimental evidence has elucidated common mechanisms of death-receptor mediated cellular apoptosis both locally in the kidney and in extra-renal organs during ischemic AKI. These

pro-apoptotic pathways lead to injury in the lungs, heart, liver, brain, and gut and further exacerbate multiple organ failure (MOF) during kidney IRI. While pharmacologic interventions to systemically inhibit inflammation have demonstrated laboratory success, large clinical trials have failed to show benefit in patients. We anticipate that abnormal apoptotic signaling may be a common mechanism of distant organ injury during ischemic AKI with future potential therapeutic implications. The purpose of this manuscript is to systematically review mechanisms of cellular apoptosis in mediating kidney IRI, organ crosstalk and systemic organ dysfunction during ischemic AKI.

Biography

Aisha Khan is a professor in Nephrology at University of Michigan in Michigan, USA.

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