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Postconditioning is life saving in an animal model of acute renal failure

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Ischemia/reperfusion (I/R) injury in the kidney accounts for the majority of acute kidney injury and represents an important cause of morbidity and mortality of hospitalized patients. Recently, it has been shown that short, repeated sequences of intermittent ischemia and reperfusion after a prolonged ischemic episode, so-called ischemic postconditioning (POC), attenuate renal I/R injury in animal models. Up to date, there is only one report of applying POC in human kidney transplantation. Although POC appears to be safe and feasible in human, no benefit in terms of reduced DGF or better renal function was observed. The current study aims to evaluate potential mechanisms by which POC protects against I/R injury. We hypothesized that application of the POC strategy could attenuate renal I/R injury by dramatically preventing early mitochondrial free radical generation during reperfusion and ameliorating mtDNA damage. We tested this hypothesis in rats subjected to severe kidney I/R injury. We conclude that POC protects the kidney from I/R at a relatively early time by inhibiting the burst of ROS and by attenuating mtDNA damage and deletions. We further speculate that diminished mitochondrial damage produced by POC was responsible for the lower grade of kidney injuries, as detected by improved serum Cr values, decreased caspase-3 activation, and a decreased number of TUNEL-positive cells. Moreover, opening of mitochondrial KATP channels by POC may play a pivotal role in preventing oxidative stress and attenuating mtDNA damage in renal I/R injury.

Biography

Xiuying Zhang has completed her PhD at the age of 30 years from Jilin University and postdoctoral studies from Johns Hopkins University School of Medicine. She is Professor of Pathology at Jilin University, and now moves to Capital Medical University as a professor and NSFC funded independent Principle Investigator. She has published more than 20 papers in reputed journals.

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