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Nephrology: Losartan treatment diminishes kidney injuries and improves glomerular filtration in hypertensive rats with ischemic acute kidney injury - Milan Ivanov - University of Belgrade

Milan Ivanov
University of Belgrade, Serbia

Renal ischemia-reperfusion (I/R) is a major cause of acute kidney injury. The pathogenetic mechanisms underlying I/R injuries involve oxidative stress and apoptosis. Competitive antagonists of type I angiotensin II receptor (AT1R) are well known antihypertensive drugs with some antioxidative potential. This study aimed to investigate the effects of Losartan on renal I/R injury comorbid with hypertension in an in vivo rat model. Experiments were performed on anaesthetized adult male spontaneously hypertensive rats (SHR). The right kidney was removed and the renal ischemia was performed by clamping the left renal artery for 40 min. SHR groups received losartan or vehicle in the femoral vein 5 min during and 175 min after the period of ischemia. All biochemical parameters were measured and kidney tissue was analysed morphologically and immunomorphologically applying Bax and Bcl-2 antibodies 24 hours after ischemia. Losartan treatment significantly increased creatinine clearance ($p<0,001$; ARF+LOS vs. ARF), attenuated TBARS level ($p<0.01$; ARF+LOS vs. ARF) and increased catalase activities ($p<0.05$; ARF+LOS vs. ARF) after I/R injury. Moreover, bax expression was significantly lower in losartan-treated rats. Tubular dilatation was smaller or even absent in some kidney specimens. In the cortico-medullary zone, tubular necrosis was reduced. Losartan protects SHR kidney from I/R injury by suppressing oxidative stress that results in cell apoptosis reduction and increasing of glomerular filtration. Therefore, blockade of AT1R may have beneficial effects in hypertensive patients who have developed ischemic acute kidney injury.

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