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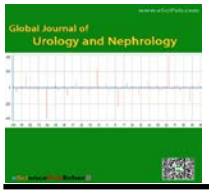

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Renal Ischemia-reperfusion Induces Renal Collagen Deposition Through Nadph Oxidase Activation in Rats

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ABSTRACT

Introduction:Acute kidney injury (AKI) may predispose the onset of chronic kidney disease (CKD) and hypertension through changes in renal NADPH oxidase and renal fibrosis. Objectives: To investigate, in rats, whether AKI induces later renal fibrosis through NADPH oxidase activation. Methodology: This protocol was approved by the Committee of Ethics in Animal Experimentation of UFPE (nº 23076.060473/2014-91). Male Wistar rats (120 days-old; n=8) were submitted to AKI through bilateral renal ischemia-reperfusion injury (IR; 45 min ischemia followed by 30 days of reperfusion). One group (n=8) was submitted to IR in the presence of oral apocynin administration (100 mg/kg) 24 hours before and after IR, to inhibit NADPH oxidase activity, while other (n=8), received daily oral treatment with apocynin (100 mg/kg) 24 hours after reperfusion until the end of experiment. These groups were compared to a control group submitted to simulation of IR procedure. Thirty days after reperfusion, it was evaluated creatinine clearance (ClCreat) and systolic blood pressure (SBP), as well as kidney collection, to evaluate lipid peroxidation, NADPH oxidase activity and collagen deposition. Statistical analysis was performed using one-way ANOVA followed by Newman-Keuls test (P<0.05). Results: Twenty-four hours after IR, serum creatinine was 4 times higher (P<0,001) than control group, while apocynin-treated rats presented similar values. After 30 days of IR, it was observed lower ClCreat (50%, P <0.01) and higher SBP (10%, P<0,05), in parallel, to increased renal lipid peroxidation, and NADPH oxidase activity. Finally, collagen deposition in renal cortex was 170% higher (P<0,01) in the IR group than control rats. Both protocols of apocynin administration prevented IR induced changes. Conclusion: These data suggest that AKI induced by IR promotes renal fibrosis and favors the establishment of CKD and hypertension, through activation of NADPH oxidase-dependent superoxide production.

Keywords: Fibrosis; Ischemia-reperfusion; Oxidative stress; NA

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