EFFECTS OF NADPH OXIDASE BLOCKADE AND HYPERBARIC OXYGEN PRECONDITIONING ON 4-HNE, NGAL, AND HO-1 TISSUE EXPRESSION IN POSTISCHEMIC ACUTE KIDNEY INJURY INDUCED IN SPONTANEOUSLY HYPERTENSIVE RATS

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Kidney disease represents a serious global health problem. Free radicals and prooxidants produced during ischemic acute kidney injury (AKI) may further aggravate the course of the disease and play a role in the pathogenesis of subsequent complications. The aims of our study were to examine the importance of NADPH oxidase blockade and to determine the effect of hyperbaric oxygen preconditioning on the immunohistochemical analysis of 4-hydroxynonenal (4-HNE), neutrophil gelatinase-associated lipocalin (NGAL), and heme oxygenase-1 (HO-1) tissue expression in postischemic acute kidney injury induced in spontaneously hypertensive rats (SHR). Twenty-four hours before AKI induction, HBO preconditioning was carried out by exposing to pure oxygen (2.026 bar) twice a day, for 60 min in two consecutive days. Acute kidney injury was induced by removal of the right kidney while the left renal artery was occluded for 45 min by atraumatic clamp. NADPH oxidase blockade was performed by Apocynin (40 mg/kg body weigh)t, intravenously, 5 min before reperfusion. We showed increased 4-HNE renal expression in postischemic AKI compared to Sham-operated (SHAM) group. Apocynin treatment, with or without HBO preconditioning, improved creatinine and phosphate clearances, in postischemic AKI. This improvement in renal function was accompanied with decreased 4-HNE, while HO-1 kidney expression restored close to the control group level. NGAL renal expression was also decreased after apocynin treatment, and HBO preconditioning, with or without APO treatment. Considering our results, we can say that 4-HNE tissue expression can be used as a marker of oxidative stress in postischemic AKI. On the other hand, NADPH oxidase blockade and HBO preconditioning reduced oxidative damage, and this protective effect might be expected even in experimental hypertensive condition.

Key words: acute kidney injury, oxidative damage, spontaneously hypertensive rats