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ROS Plays as an Inducer of Remaining Renal Mass Alteration Following Transient Unilateral Ischemic Injury in the Kidney

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Reduction of renal mass causes the compensatory alteration of remaining renal mass, which adapts to functional overload of kidney due to the reduction of nephron. In the present study we investigated that transient and partial renal injury by 30 min of unilateral ischemia induces alteration of contralateral kidney (CLK) and that reactive oxygen species (ROS), a mediator of ischemia/reperfusion injury, is associated with the alteration. Here, we report that 30 min of unilateral renal ischemia (UIRI) in mouse results in the increase in ratio of CLK weight to body weight (BW) with tubular cell proliferation 9 days after UIRI. Superoxide level in the CLK significantly increased along with the increase of NADPH oxidase 2 (NOX2) expression. ERK was activated in the CLK. However, the increase of lipid peroxidation and histological damage in the CLK were not observed. Treatment of Mn (III) Tetrakis (1-methyl-4-pyridyl) porphyrin (MnTMPyP, a superoxide dismutase (SOD) mimetic) on UIRI mouse mitigated the increase in the ratio of CLK weight BW after UIRI along with reductions in NOX2 expression, superoxide formation, ERK activation, and tubular cell proliferation. In conclusion, our findings demonstrate that renal mass reduction by transient I/R injury results in the changes of remaining renal mass through the non-damage inducible levels of ROS production.

Key Words: 허혈/재관류, 신장비대, 활성산소
Ischemia/reperfusion, Kidney hypertrophy, ROS