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허혈성 급성 신부전의 초기 신손상에서 PGI2의 신보호 효과

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Protective Effect of Prostaglandin I2 on Experimental Kidney Warm Ischemia-reperfusion Injury

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Background : Kidney ischemia-reperfusion injury (IRI), a leading cause of acute kidney injury (AKI), is characterized with robust inflammatory response. Prostaglandin I2 (PGI2) is known to have anti-inflammatory function as well as antithrombotic effect. We tested the hypothesis that PGI2 attenuates initial renal injury following warm IRI in mice.

Methods : C57BL/6 mice were randomly allocated into 4 groups: control (IRI without medication, n=8), DMSO-IRI (n=6), PGI2-IRI (n=10), PGI2-sham (n=4). PGI2 was diluted with DMSO and 10 mg/kg was injected into peritoneal cavity right before reperfusion. Serum creatinine was measured for 48 hours and the expression of Na-K-Cl cotransporter (NKCC2) and Na-H exchanger 3 (NHE3) was measured with western blot at 48 hours after IRI.

Results : Renal functional deterioration was attenuated in PGI2-IRI group compared with both control and DMSO-IRI groups (serum creatinine mean±SE, Day 0: 0.52±.035 in control, 0.33±0.031 in DMSO, 0.35±0.033 in PGI2, 0.37±0.014 in sham groups, Day 1: 2.72±0.167 in control, 2.33±0.338 in DMSO, 1.40±0.206 in PGI2, 0.52±0.053 in sham, Day 2: 2.33±0.427 in control, 1.31±0.051 in DMSO, 0.73±0.114 in PGI2, 0.44±0.051 in sham) The expression of NHE3 and NKCC2 was significantly decreased in control, but down-regulation of NHE3 and NKCC2 was attenuated in PGI2-IRI group (NHE3/β-actin on day 2, 29.88±8.787 in control, 116.87±30.933 in PGI2-IRI, P <0.05 ; NKCC2/β-actin on day 2, 77.65±26.787 in control, 364.01±16.189 in PGI2-IRI, P <0.001)

Conclusion : PGI2 treatment attenuated early renal injury and prevented the down-regulation of NHE3 and NKCC2 following IRI.

Key Words : 프로스타글란딘 I2, 허혈성 급성 신부전, 초기 신손상
Prostaglandin I2, Ischemic AKI, Early renal injury