P206

저체온이 ERK 인산화를 통해 신허혈재관류 손상의 완화

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Hypothermia Attenuates Ischemia-Reperfusion Renal Injury Via Preservation of ERK Phosphorylation

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Background: It is known that hypothermia during ischemic period is protective against ischemia reperfusion renal injury. Although it is thought that decrease of metabolic demand and free radical generation may involve in hypothermia, the exact explanation is not known. We evaluate whether molecular pathway might be involved in renal protection against IR renal injury at hypothermia (32 C).

Methods: C57Bl/6 mice were divided into six groups; sham operatative mice, 32 C temperature (cold) IR mice, 37 C temperature (warm) IR mice (reperfusion 27 minutes after clamping of both renal artery and vein) and PD98059 with 32 C temperature IR mice, L-NAME with 32 C temperature IR mice. Kidneys were harvested at 10 min and 27 min after both renal artery ischemia and 24hr after IR injury. Renal ERK, iNOS, eNOS, caspase-3 activation was evaluated by western blot. We examined BUN, serum creatinine (s-Cr), TUNEL and light microscopic findings.

Results: Serum level of BUN and s-Cr in cold IR mice was significantly lower than that of warm IR mice ($p<0.01$). Hypothermia decreased tissue injury score and TUNEL positive cells in IR kidney. pERK and eNOS expression were increased in cold IR mice at ischemic kidney (10 min and 27 min after both renal artery clamping). Inhibition of p-ERK using PD98059 elevated the levels of BUN and s-Cr in cold IR mice than control cold IR mice. Also it increased tissue injury score and TUNEL positive cells that were decreased in cold IR kidney. However, there were no differences of BUN, s-Cr and tissue injury score between cold IR mice and L-NAME with cold IR mice.

Conclusion: In conclusion, the results of the present study suggest that hypothermia has reno-protective effect on IR injured mice. Renoprotective effect of hypothermia may be involved in ERK phosphorylation.

Key Words: 신허혈손상, 신장
Ischemia-reperfusion injury, Kidney