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돼지 renal sympathetic denervation 모델에서 급성신질환과 inflammasome의 조기 활성화

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Early Activation of Inflammasome in Acute Kidney Injury after Renal Sympathetic Denervation in Pig

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Objectives: Renal sympathetic denervation (RDN) is available and implemented as a strategy for the treatment of resistant hypertension.In the aspect of chronic safety, renal function, as assessed by serum creatinine, eGFR(MDRD), and cystatin C was reported to be unchanged from baseline at 6 months. We investigated whether RDN might cause subtle inflammation and subclinical damage in the early phase of acute kidney injury (AKI).

Methods: Female pigs were divided into 6 groups; normal control (group A), Sham-operated control (group B), contrast media control (group C), and renal sympathetic denervation groups subdivided into 3 groups according to the time of sacrifice; immediately (group D), 1 week later (group E), and 2 weeks later (group F) after RDN. We checked IL-1α, 1β, 18, 6, 10, TNF-α, cystatin C, caspase-1, ASC, and NLRP3 as early biomarkers of inflammation and AKI.

Results: There were no significant changes in group B and C compared to group A. BUN, serum creatinine, cystatin C, urine protein/creatinine ratio, and urine albumin/creatinine ratio showed a tendency to increase in group D and E and then decrease in group F with no statistical significance. Pro-inflammatory cytokines, IL-1β, 18 increased in group D and E ($p<0.05$ vs. A), and decreased in group F ($p<0.05$ vs. D, E) significantly. Casapse-1 activity, ASC, and NLRP3 expressions were also increased in group D ($p<0.05$ vs. group A), and decreased in group E and F ($p<0.05$ vs. group D).

Conclusion: RDN did not cause clinically significant damages on kidneys. However RDN can induce the activation of pro-inflammatory cytokines, caspase-1 and NLRP3 inflammasome, and then transiently self-limited acute kidney injury.

Key Words: Inflammasome, 신장 교감신경절제술, 급성신질환
AKI, Inflammasome, Renal sympathetic denervation