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Renal Tubular GLP-1 receptor is increased in early sepsis which reduced in CKD and sepsis induced kidney injury

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Objectives:

Sepsis is associated with a high incidence of acute kidney injury. In sepsis-related acute kidney injury, the predominant pathogenetic factor is renal vasoconstriction with intact tubular function with oxidative stress. Glucagon-like peptide-1 (GLP-1) has been widely used in various areas including diabetes and shows renoprotective effects. However, renal tubular change of GLP-1 receptor (GLP-1 R) in acute kidney injury is still unveiled. The aim of study is to investigate renal cortical GLP-1 R changes in sepsis related acute kidney injury, and to compare chronic kidney disease related changes.

Methods:

Male SD rats were used, and the experiments were divided into two parts. The first experiment was designed for renal tubular GLP-1 receptor expression in sepsis by dividing into 3, 6, 12, 24, and 72 hours after sepsis induction. The second experiment consisted with control, chronic kidney disease, sepsis, chronic kidney disease and sepsis. CKD was created by 5/6 nephrectomy. The sepsis group had an artificial septicemia by cecal perforation at 8 weeks of the experiment in CKD. After 3 days, blood and urine of each group were collected, and tissues were collected from kidney.

Results:

Renal cortical GLP-1 receptor activity is markedly increased in early stage of sepsis. Comparing 3, 6, 12 and 24 hours of sepsis, the peak GLP-1 R activity is showed in 24 hours after injury. However, the activity is decreased in 3 days after sepsis. Comparing CKD rat model and sepsis related AKI on CKD model, GLP-1 R activity was decreased in sepsis on CKD.

Conclusions:

Renal cortical GLP-1 R is increased in 24 hours of sepsis, then decreased lower than control in 72 hours, which seems to be associated with renal response role in early renal injury. GLP-1 treatment might do renoprotective role in early renal injury.