The role of serum C-reactive protein in acute ischemic-reperfusion injury of kidney

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Objective: Blocking early C-reactive protein-mediated inflammatory reaction may have therapeutic implications in improving the prognosis of acute renal failure with severe ischemic-reperfusion injury. Therefore, the role of serum C-reactive protein in acute renal ischemic-reperfusion injury was investigated.

Methods: Fourteen New Zealand albino rabbits were selected and divided into a treated and a control group at random. An acute renal ischemia-reperfusion injury model was induced by clamping the right renal artery for 45 minutes with simultaneous contralateral nephrectomy, followed by right renal reperfusion. The treated group was injected with dexamethasone (1 mg/kg) 2 minutes before renal reperfusion. Serum C-reactive protein, blood urea nitrogen, creatinine, and urine volume were recorded at designed time phases in both groups. Data were expressed as mean ± standard deviation and analyzed using the Student's t test.

Results: In the control group, there was a steady increase of serum C-reactive protein that reached its peak at 6-hour reperfusion, and a positive correlation between C-reactive protein and blood urea nitrogen and creatinine (r = 0.62 and 0.53, respectively); there was a negative correlation between C-reactive protein and urine volume (r = -0.52). Compared with the control group, C-reactive protein values in the treated group remained mainly in the baseline levels after reperfusion, with C-reactive protein peaking at 4-hour reperfusion (p<0.01), whereas urine volume increased significantly (p<0.01).

Conclusions: This study indicates that C-reactive protein is involved in the pathogenesis of acute renal ischemic-reperfusion injury; blocking early C-reactive protein-mediated inflammatory reaction may have therapeutic implications in improving the prognosis of acute renal failure with severe ischemic-reperfusion injury. (Hong Kong J Nephrol 2002;4(1):39-42)

Key words: C-reactive protein, Creatinine/urea, Dexamethasone, Kidney failure, Acute