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Change of lung surfactant protein A and D in renal ischemia reperfusion injury

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Objectives:

Many reports suggested that acute kidney injury(AKI) adversely affects the lungs. Some animal study showed that AKI itself affect to endothelial cell injury due to inflammation and apoptosis in lungs. Surfactant protein (SP)-A and SP-D are members of C-type lectin family. In many lung disease including sepsis, SP-A and D in serum could be used as biomarker to predict prognosis. We investigated whether renal ischemia reperfusion injury influence the change of lung specific markers including SP-A and D, which could develop impairment of lung host defense mechanism.

Methods:

Ischemia-reperfusion (IR) was induced in mice by bilateral renal vascular clamp. At 4 and 24 h, sham and IR models were sacrificed. Blood, kidney and lung were collected. Commercial ELISA Kit was used to measure SP-A and D.

Results: BUN, Creatinine, and tubular injury score on PAS stain in IR groups statistically increased compared with sham at each time. (Creatinine 4h Sham vs AKI 0.35 ± 0.06 vs 1.13 ± 0.08 ; 24h Sham vs AKI 0.31 ± 0.03 vs 2.27 ± 0.16). Inflammatory cell infiltration in lung increased at 4, 24 in AKI group compared with sham. Serum cytokines including IL-6, TNF-alpha, MCP-1 increased at IR models. Also lung MCP-1 increased at 24 h IR model. Bax/BCL-2 ratio in lung increased at 4h IR model. Protein in BAL increased at 24h after IR. Serum SP-A increase at 4h and 24h after IR. Serum SP-D increased at 24 h, However, SP-D and A in lung and BAL were not different between sham and IR model at each time. RAGE, pneumocyte I injury marker is not different between sham and IR model at each time.

Conclusions:

Lung inflammation and cytokine increased after renal IR. Also serum SP-D and SP-A increased after renal IR. AKI itself might influence the pneumocyte II and tight junction.