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Selective digestive decontamination prevents renal ischemic injury and distant organ damage via regulatory T cells expansion

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Objectives: Acute kidney injury (AKI) is a huge medical burden worldwide. Little is known about how to prevent AKI. Gut is known as a driver of whole body immune system. Kidney-gut crosstalk has been revealed the connection between gut and kidney. Several studies have been reported that selective digestive decontamination (SDD) reduce mortality in critically ill patients but still controversial. We induced intestinal microbiota alteration by bowel decontamination to identify changes in gut environment whether be beneficial to kidneys and other distant organ or not.

Methods: We used 6 week old C57L/B6 mice to induce AKI using bilateral ischemic reperfusion injury. Bowel decontamination was done by using a mixture of three different antibiotics (Neomycin, Ampicillin, and metronidazole) giving oral garbage once a day for 12 days under specific pathogen-free condition. The renal injury was assessed by serum creatinine level. We analyzed the Foxp3, TNF-α, IL-6, IL-10 by RT-PCR in colon, mesenteric lymph nodes, spleen, kidney. At the same time, we also measured liver function test and lung myeloperoxidase (MPO) to evaluate distant organ injury of AKI.

Results: Bowel decontamination significantly attenuated AKI severity (NL control vs. bowel decontamination control vs. AKI vs. bowel decontamination control, serum creatinine level (mg/dL) 0.12±0.06, 0.1±0.05, 1.12±0.13, 0.47±0.22, p=0.008). It also showed protective effect on liver, as alanine transaminase (ALT) attenuated in bowel decontamination group (25±0.2, 23±0.2, 210±39, 60±27, p=0.017). However, lung injury was not significantly different. Colon, spleen foxp3 was significantly elevated, the kidney IL-6 was decreased in bowel decontamination group (p=0.04).

Conclusions: We conclude that selective digestive decontamination can prevent IRI and distant organ damage following by AKI. Immune modulation effect by FoxP3 expansion could be an important mechanism of kidney-gut crosstalk in AKI, and could be considered as a new therapeutic target to limit renal IRI.