KSN 2016 Abstract Submission

Acute Kidney Injury

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AICAR, AMPK activator, protects cisplatin-induced acute kidney injury

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Background: Cisplatin causes acute kidney injury (AKI) through proximal tubular injury. We investigated protective effect of the adenosine monophosphate protein kinase (AMPK) activator 5-aminoimidazole-4-carboxamide ribonucleotide (AICAR) against cisplatin induced AKI.
Methods: Male Sprague-Dawley rats were randomly divided into four groups: control, AICAR, cisplatin and cisplatin + AICAR. SD rats were injected with single dose of cisplatin (7 mg/kg, i.p.). AICAR was administered to rats at 100mg/kg i.p. daily for 5 days. The kidneys were harvested on the day 5. Blood urea nitrogen (BUN) and the serum creatinine were measured. Renal damage was analyzed in sections stained with Hemotoxylin and Eosin (H&E).
Results: A single injection of cisplatin caused marked increase of the serum creatinine and BUN levels on day 5. BUN and serum creatinine levels decreased in cisplatin-treated rats after treatment with AICAR. Compared to cisplatin group, acute tubular necrosis score decreased in rat treated with cisplatin + AICAR.
Conclusion: This study suggests that activation of AMPK activator AICAR might ameliorate the cisplatin induced AKI, partially by inhibition of the proximal tubular injury. We will continue to evaluate for JAK/STAT/SOCS pathway or RAS/MAPK signaling pathway.

Keywords: None