KSN 2017 Abstract

KSN-17-P034

Beneficial effect of α-mangostin on Acute Kidney Injury in rats via PI3K/Akt/Nrf2 Signaling Pathway

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Objectives : Acute kidney injury (AKI) is a common clinical burden with high morbidity and mortality rate. Renal reperfusion/ischemia injury play a significant role in the induction of AKI via initiate the interrelated sequence within the renal which culminate in renal damage and induction the death of renal cells. The intercellular signaling pathways involved in the AKI mechanism still unclear. Few researchers suggest that Nuclear factor erythroid 2-related factor (Nrf2) play a crucial role in the expansion of oxidative stress, via increased the protein kinase C (PKC) pathway activation. α-mangostin is a powerful antioxidant against several renal injury in various animal models. In the current study, we make attempt to scrutinize the α-mangostin against renal ischemia/reperfusion injury in animal model and find the possible mechanism.

Methods : Swiss albino Wistar rats divided into 5 groups. After 30 min the left renal tissue was removed to investigate the renal ischemia/reperfusion (I/R) damage. The renal morphology and serum parameters were estimated, respectively. The renal expression of Nrf2, phosphorylated-PKC, AkT, HO-1 and caspase-3 were also determined, respectively.

Results : The LPO marker (malondialdehyde), creatinine and BUN up-regulated 5-10 times after induction of I/R injury. The dose dependent treatment of α-mangostin significantly down-regulation the MDA, creatinine and BUN content. The treatment also restrained the acute tubular necrosis. I/R group rat showed the reduced expression of p-Akt, Nrf2, HO-1, pro-caspase-3 and enhanced the renal expression of caspase-3, which was significantly altered by the α-mangostin treatment at dose dependently.

Conclusions : In can be concluded that α-mangostin attenuates the renal injury in I/R injury rats via PI3K/Akt/Nrf2 Signaling Pathway.

Keywords : Acute Kidney Injury, α-mangostin, PI3K/Akt/Nrf2 Signaling Pathway.