KSN 2018
Seoul, Korea · May 17 - 20

Abstract Type : Poster
Presentation No. : PAK 010

Fimasartan attenuates ischemia-reperfusion injury by modulating inflammation-related apoptosis

Jang-Hee Cho, Hye-Myung Ryu, Eun-Joo Oh, Ju-Min Yook, Ji-Sun Ahn, Hee-Yeon Jung, Ji-Young Choi, Sun-Hee Park, Chan-Duck Kim, Yong-Lim Kim
Department of Internal Medicine-Nephrology, Kyungpook National University Hospital, Korea, Republic of

Objectives: Fimasartan, a new angiotensin II receptor antagonist has been shown to reduce myocyte damage and stabilize atherosclerotic plaque through its anti-inflammatory effect in animal studies. We investigated the effects of pretreatment with Fimasartan on renoprotection against the ischemia-reperfusion injury (IRI) in an ischemic renal injury mice model.

Methods:

C57BL/6 mice were pretreated or not with 5 (IR-F5) or 10 (IR-F10) mg/kg/day of Fimasartan for 3 days. Renal ischemia was induced by clamping unilateral renal vascular pedicles for 30 min. Histology, pro-inflammatory cytokines, and apoptosis assay were evaluated 24 hr after IRI.

Results: Compared to untreated group, blood urea nitrogen and serum creatinine levels were significantly lower in IR-F10 group. IR-F10 kidneys showed less tubular necrosis and interstitial fibrosis than untreated kidneys. F4/80 for macrophage infiltration marker and TNF-α expression decreased in IR-F10 group. High-dose Fimasartan treatment attenuated upregulation of TNF-α, IL-1ß, and IL-6 in the ischemic kidney. Lower TUNEL-positive cells were also observed in IR-F10 mice compared to the control. Fimasartan caused a significant decrease in caspase 3 activity and the level of Bax and increased Bel2 level.

Conclusions:

Fimasartan could preserve renal function and tubular architecture from IRI in ischemic renal injury model. Fimasartan also attenuated upregulation of inflammatory cytokines and decreased apoptosis of renal tubular cells. Our results suggest that Fimasartan inhibited the process of tubular injury through the protection of apoptosis induced by inflammatory pathway.