

Abstract Type : Oral

Abstract Submission No. : OR-1319

L-Carnitine Treatment Attenuates Renal Tubulointerstitial Fibrosis Induced by Unilateral Ureteral Obstruction

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Objectives: L-Carnitine (LC) has protective effects against multiorgan damage through its antioxidant capacity and preservation of mitochondrial network. This study examined whether LC treatment would offer nephroprotection in a rat model of unilateral ureteral obstruction (UUO) and an in vitro study.

Methods: Sprague-Dawley rats underwent UUO and treated daily LC for 7 or 14 days. The influence of LC on UUO was evaluated by means of histopathology, gene expression, oxidative stress, apoptosis and autophagy, mitochondrial function, and AKT/PI3K signaling pathway. In addition, human kidney cells (HK-2) subjected to H₂O₂ were treated with LC.

Results: LC suppressed inflammatory mediators (monocyte chemoattractant protein-1 and osteopontin) and inhibited fibrotic cytokine transforming growth factor- β 1 (TGF- β 1) expression, resulting in a significant attenuation of tubulointerstitial fibrosis (TIF) in a time-dependent manner. Increased oxidative stress induced by UUO was associated with impairment of mitochondrial function, and excessive apoptosis and autophagy via AKT/PI3K dependent signaling pathway, and this was reversed with addition of LC. In H₂O₂-treated HK-2 cells, LC improved cell viability and reduced reactive oxygen species production. Moreover, LC treatment decreased H₂O₂-induced upregulation of TGF- β 1 expression and AKT/PI3K activation in HK-2 cells.

Conclusions: L-Carnitine protects against the progression of TIF in obstructed kidney.