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Acute Kidney Injury

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Autophagy protects against contrast induced tubular epithelial injury

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Background: Radiocontrast-induced nephropathy (RCN) is common cause of acute kidney injury in hospital. However, preventing and treating strategies against developing RCN were very limited. The role of autophagy in the pathogenesis of RCN remains undetermined, therefore we investigated its role in RCN.
Methods: We examined the expression of autophagic and apoptotic proteins during progression of contrast (idoxanol) induced injury to renal tubular epithelial cells (RTEC). For determine protective role of autophagy against contrast injury, we inhibit autophagy with small interference RNA (siRNA) for ULK1, and measured the changes of cell viability and induction of apoptotic and autophagy protein for 48hr.
Results: Following contrast exposure to RTEC, cell viability was reduced for 3hr, but it was increased at 24hr and 48hr. Apoptosis was detected as early as 1hr after contrast exposure as indicated by induction of caspase 3 and 8 and they were increased for 48hr. Otherwise autophagy, indicated by LC3 and autophagy-related gene protein 7 (ATG7), was detected at 3hr after contrast exposure, and induction of LC3 and ATG7 were further increased up to 48hr. After inhibiting autophagy by ULK1 siRNA, survived RTEC was not increased at 24 and 48hr after contrast exposure.
Conclusion: Autophagy plays cytoprotective role in contrast induced RTEC injury and it may occur independently of apoptosis.

Keywords: autophagy, contrast induced nephropathy