Interleukin-22 inhibits H2O2 induced apoptosis in HK-2 cell through suppression of ER stress.

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Objectives:

Oxidative stress plays a key role in acute kidney injury. Recent studies have shown that endoplasmic reticulum (ER) stress in tubular cell is associated with acute kidney injury. However there is little information about anti-apoptotic cytokine to overcome ER stress in HK-2 cell. In this study, we investigated the effect of interleukin-22 on H2O2 induced apoptosis and ER stress in HK-2 cell.

Methods:

We used HK-2 cell lines. Interleukin-22 toxicity and therapeutic concentrations was examined by XTT test. H2O2 induced injury is known as a oxidative stress model of acute kidney injury. We stimulated HK-2 cell with H2O2 and induced apoptosis in this cell. The protein expression of ER stress markers such as glucose-regulated protein 78 (GRP78) and C/EBP homologous protein (CHOP) were evaluated by western blot.

Results:

In HK-2 cell, H2O2 increased apoptotic protein, cleaved PARP in a dose-depend manner at 4 hours. ER stress marker, C/EBP homologous protein (CHOP) was also increased by H2O2 stimulation. Interleukin-22 pretreatment prevented H2O2 induced apoptosis and decreased expression of CHOP in HK-2 cells on western blotting.

Conclusions:

Our results suggest interleukin-22 prevents H2O2 induced apoptosis HK-2 through suppression of CHOP pathway. Interleukin-22 could be an anti-apoptotic cytokine for acute kidney injury.