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Anti-fibrotic and anti-apoptotic effect of PLGA loaded nano-formulation of Ganoderic acid against cisplatin-induced acute kidney injury in rats via PI3K/Akt/Nrf2 pathway

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Objectives: High risk of death is associated with conversion of acute kidney injury (AKI) to chronic kidney injury (ACI). Activation of PI3K/Akt/Nrf2 pathway can give lot of protection to cell that might undergo apoptosis. This study is an effort to look into the anti-fibrotic and apoptotic effect of nono formulation of gonoderic on cisplatin induce renal injury via PI3K/Akt/Nrf2 pathway.

Methods: Nanoparticulate systems for ganoderic acid (GA-PLGA) developed by ultra-sonication technique and influential formulation parameters were systematically optimized via Box-Behnkendesign to acquire quality range desired nano size compound. Wistar rats were randomly divided into different group and single intraperitoneal injection of CIS (6 mg/kg) was used for the kidney injury. Biochemical, antioxidant and inflammatory mediator estimated. Renal expression of phosphorylated-PKC, Nrf2, HO-1 and AkT were determined.

Results: GA-PLGA-NPs exhibited the average particle size of 178 nm with poly-dispersity index 0.19, drug loading capacity >91%, eta potential <20 mV and sustained invitro 95% drug release after 48 h. GA-PLGA-NPs significantly reduced the creatinine (42%), blood urea nitrogen (45%), inflammatory mediators such as COX-2 (38%) PGE_2 (32%), pro-inflammatory cytokines TNF-α (45%), IL-6 (42%), IL-1β (36.5%) and antioxidant parameters such as SOD (56%), CAT (45%), GSH (38%) and increased the MDA (74%), respectively. GA-PLGA-NPs reduced the fibrosis and apoptosis through altering the expression of Bax (28%), Bcl-2 (35%) and caspase-3 (37%). GA-PLGA-NPs p-Akt (1.9%), Nrf2 (0.8 %), HO-1 (0.9%), pro-caspase-3 (0.6%).

Conclusions: On the basis of above result, it can be stated that this investigation provides the evidence for anti-apoptotic and anti-fibrotic effect of GA-PLGA-NPs in CIS induced kidney injury in rats via PI3K/Akt/Nrf2 signaling pathway.