KSN 2020
FULLY VIRTUAL MEETING

Abstract Type : Poster
Abstract Submission No. : PO-1116

Ganoderic acid exert renal toxicity via alteration of inflammation pathway against anti-inflammatory drug induced renal injury in rat kidney

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Objectives: Non-steroidal anti-inflammatory like indomethacin is commonly used drug for clinical therapeutics. However, indomethacin having the limitation due to induction the renal toxicity. These toxic effects were related with not only the reduction of prostaglandin synthesis but also drug elevated oxidative stress. Due to these toxic effects, antioxidant and anti-inflammatory therapy can be used as the alternative and combinational therapies. Hence, the present experimental protocol was used to scrutinize the renal-protective effect of ganoderic acid against indomethacin induced renal injury via anti-inflammatory mechanism.

Methods: Swiss Wistar rats were pre-treated with ganoderic acid (10, 20 and 40 mg/kg) and then treated with indomethacin (20 mg/kg) to scrutinize the renal function such as uric acid, creatinine and blood urea nitrogen. Additionally, antioxidant parameters like superoxide dismutase (SOD), malonaldehyde (MDA), glutathione (GSH), glutathione peroxidase (GPx), catalase (CAT), 8-Oxo-20-deoxyguanosine (8-oxo-dG); pro-inflammatory cytokines viz., tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), interleukin-1β (IL-1β); inflammatory mediators including prostaglandin E_2 (PGE_2), cyclo-oxygenase-2 (COX-2), endothelial nitric oxide synthase (eNOS) and nuclear kappa b factor (NFkB), respectively. Renal tissue sections were also histopathological evaluated.

Results: Ganoderic acid treatment reduced the renal parameters such as uric acid (78%), creatinine (83.4%), blood urea nitrogen (83.5%); antioxidant parameters like SOD (67.5%), MDA (70.3%), GSH (69.4%), GPx (63.4%), CAT (58.5%), 8-oxo-dG (65.5%); pro-inflammatory cytokines viz., TNF-α (81.5%), IL-6 (73.4%), IL-1β (75.5%); inflammatory mediators includingPGE_2(80.3%), COX-2(65.5%), eNOS (74.4%) and NFkB(80.5%), respectively. The biochemical and histopathological analysis suggest the renal protective effect against indomethacin induced renal injury.

Conclusions: Our result clearly showed that ganoderic acid is a renal protective effect against the indomethacin induced renal toxicity.