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Renalprotective effect of Ganoderic acid against renal dysfunction in type II diabetes via alteration of TGF-β

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Objectives: Various advancement of diabetes mellitus and its complications, still diabetic nephropathy (DN) is still a global health problem for various patients suffering from diabetes, because of the inefficacy and associated side effects of pharmacological drugs. There is an urgent need for new therapeutic drugs that can prevent the expansion of DN via targeting the various metabolic and inflammatory pathways, without inducing the side effects. Current years, various researchers have suggested that the inflammation plays a significant role in the expansion of DN, hence, NF-kB has received more attention. In the current experimental study, we scrutinize the nephroprotective effects of ganoderic acid against high-fat diet/streptozotocin-induced type II diabetes mellitus and explore the underlying mechanism.

Methods: STZ used to induce type II diabetes and rats were received the oral administration of ganoderic acid and glibenclamide post-induction diabetes and estimation the body weight, food intake, blood glucose, plasma insulin, biochemical, antioxidant, proinflammatory cytokines and inflammatory mediators, respectively.

Results: Dose dependently treatment of GA significantly ($P<0.001$) reduced the blood glucose level (78%), food intake (54%), Hb1c (55.5%), HOMA-IR (50.5%) and increased the plasma insulin (65.7%), body weight (18%). GA significantly altered the antioxidant level in renal tissue via down-regulated the MDA (56.7%) and up-regulated the SOD (76.5%), GSH (70.5%), GPx (65.5%) and CAT (60.5%), respectively. GA altered the renal parameters such as BUN (68.5%), creatinine (60.2%), serum protein (60.5%). GA decreased the expression of TGF-β (46.6%), COX-2 (65.3%), iNOS (68.7%) and PGE_2 (59.9%) and ameliorated the structural alteration in renal tissues.

Conclusions: Collectively, we can conclude that ganoderic acid reduced the renal dysfunction in type II diabetic rats via alteration of the inflammatory pathway and TGF-β.