

Renoprotective Effects of Isoliquiritigenin against Glycerol-Induced Myoglobinuric Acute Kidney Injury in Rabbits

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ABSTRACT

Acute kidney injury (AKI) caused by myoglobinuria associated with rhabdomyolysis is a severe clinical condition with limited effective therapies. This study evaluates the nephroprotective effects of isoliquiritigenin (ISO) in a glycerol-induced model of AKI in male rabbits. Sixty healthy male albino rabbits were divided into five groups: control, induction (glycerol-induced AKI), vehicle, ISO 30, and ISO 50. Renal function was assessed by measuring serum urea, creatinine, and creatine phosphokinase (CK) levels. Electrolyte balance, oxidative stress markers malondialdehyde (MDA), antioxidant enzyme levels glutathione peroxidase (GPx), and pro-inflammatory cytokine levels (IL6, IL1 β , and TNF- α) were also evaluated. Histopathological examination of kidney tissues was performed to assess structural damage. The induction group exhibited significantly elevated urea, creatinine, and CK levels, alongside electrolyte imbalances, increased malondialdehyde MDA and decreased GPx levels, indicating severe oxidative stress and inflammation. ISO treatment, particularly at a 50 mg dose, significantly reduced serum urea, creatinine, CK levels, and improved electrolyte homeostasis. ISO also demonstrated potent antioxidant effects, reducing MDA and increasing GPx levels, while lowering pro-inflammatory cytokines. Histopathological analysis showed marked protection against kidney damage in the ISO-treated groups, with near-normal renal architecture observed in the 50 mg dose group. ISO exhibits significant nephroprotective effects against glycerol-induced AKI by improving renal function, restoring electrolyte balance, reducing oxidative stress, and suppressing inflammation. These findings suggest that ISO could be a promising therapeutic agent for managing AKI, particularly in cases associated with rhabdomyolysis.

Keywords: Acute kidney injury, Myoglobinuria, glycerol, oxidative stress, inflammation.

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