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Effect of exogenous IGF-1 administration on acetaminophen toxicity induced liver injury

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For liver toxicity, there is no clear protective drug till date. Here, we investigated the protective effects of insulin-like growth factor 1 (IGF-1) on acetaminophen (APAP)-induced liver injury and the molecular processes underlying APAP-induced liver damage involving oxidative stress and endoplasmic reticulum (ER) stress. Forty male Wistar rats were randomly divided into four groups. Group I that had only saline served as the control. Group II received APAP (300 mg/kg body wt.) and saline, Group III & IV received APAP as in Gr. II, plus 1 and 2 mg/kg/day of IGF-1, respectively for three days. Liver histopathology, biochemical analysis and ELISA assays were performed to evaluate the protective effect of IGF-1 against APAP-induced liver injury. Significant cellular damage and necrosis were observed in the liver in the APAP and saline groups. Treatment with IGF-1 resulted in a dose-dependent reduction in cellular damage and necrosis. ALT levels, indicative of liver damage, were significantly decreased in the IGF-1-treated groups. MDA levels, a marker of oxidative stress, were reduced with IGF-1 treatment. GSH levels, an antioxidant, increased with IGF-1 treatment. ATF6 levels were reduced with IGF-1 treatment, while TNF-alpha levels were decreased in a dose-dependent manner. IGF-1 treatment protects against APAP-induced liver injury by reducing cellular damage, oxidative stress and ER stress markers. These findings suggest that IGF-1 may have therapeutic potential in mitigating APAP-induced hepatotoxicity.

Keywords: Hepatotoxicity, Inflammation, Oxidative stress, Paracetamol