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Protective Effects of p-CA Against Acute Liver Damage Induced by LPS/D-GalN in Wistar Albino Rats

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Abstract

Aim: Liver regulates metabolism of biomolecules and injury of liver causes distortion of metabolic functions. This injury may be oxidative or inflammatory induced by numerous factors including alcohol, pathogens and xenobiotics. This scientific study was planned to investigate the anti-inflammatory and anti-oxidant potential of p-coumaric acid (p-CA) on Lipopolysaccharide/D-Galactosamine (LPS/D-GalN) induced liver injury.

Methods: DPPH analysis, reducing power assay and HPLC analysis were performed during in-vitro studies of p-CA. Similarly, in-vivo experiments were performed using Wistar Albino rats. Normal control and intoxicated group received (5mL/kg normal saline p.o), standard treatment groups received ascorbic acid (100mg/kg p.o) and silymarin (25mg/kg p.o), while p-CA treatment groups received (100mg/kg p.o) for 28-days. After completion of 28-days, LPS/D-GalN injection (300 mg D-GalN/kg and 10 µg LPS/kg i.p.) was given at 6th, 12th and 24-hours to all groups except normal control group. Animals were sacrificed; serum and liver samples were harvested and subjected to biochemical and histological examinations, respectively.

Results: The results revealed that p-CA possess strong antioxidant activity. Increased levels of leukocyte infiltration (TLC), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), total bilirubin (TBIL), lipid panel (eg TG, TC, LDL-C, VLDL-C), whereas decreased HDL-C levels noticed in LPS/D-GalN groups as compared to normal control groups. Pro-Inflammatory markers (eg TNF-α, IL-6, IL-1β) and lipid peroxidation marker, eg malondialdehyde (MDA) increased while superoxide dismutase (SOD) and reduced glutathione (GSH) levels were decreased significantly in groups treated with LPS/D-GalN. ANOVA with Bonferroni post hoc analysis was used for statistical analysis of. H&E staining was done to assess architectural abnormalities among liver cells.

Conclusion: In conclusion, p-CA could ameliorate LPS/D-GalN induced hepatic injury via regulation of immune responses, liver function enzymes, lipid profile, oxidative stress and pro-inflammatory markers.

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Biography

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