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Oral Communication Abstract
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Anti-apoptotic and anti-fibrotic effect of crocetin against cisplatin-induced acute kidney injury in rats via PI3K/Akt/Nrf2 pathway

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Objectives: Nephrotoxicity is a serious complication observed during the cisplatin chemotherapy and due to this, cisplatin in clinic is restricted in use. Acute kidney injury (AKI) is the serious problem with the kidney that developed the risk for the expansion of chronic renal disease and high risk of death. Crocetin have potential anti-inflammatory and antioxidant effect
and its effect against cisplatin induces renal injury still unknown. In this study we try to explore the nephroprotective effect of crocetin against cisplatin induced AKI via PI3K/Akt/Nrf2 pathway.

Methods: Wistar rats were divided into groups and received the intraperitoneal injection of cisplatin (6 mg/kg) for induction the nephrotoxicity and received the oral administration of crocetin. Body weight and food intake were estimated at regular time interval. Antioxidant, renal parameters, inflammatory parameters and pro-inflammatory cytokines were measured. 4-hydroxynonenal (HNE), phosphorylated-PKC, NF-E2-related factor-2 (Nrf2), heme oxygenase-1(HO-1), and AkT expression were scrutinized.

Results: Crocetin increased the body weight and food intake as compared to cisplatin induced nephrotoxicity rats. Crocetin significantly (P>0.001) reduced the level of BUN (62.5%,) (72.1%); pro-inflammatory cytokines such as IL-1β (69.3%), IL-6 (72.1%), TNF-α (81.2%); inflammatory mediators include COX-2 (65.8%), PGE2 (64.2%) and NF-κB (63.9%). Crocetin significantly (P>0.001) increased the level of SOD (79.3%), CAT (53.2%), GPx(67.3%), GSH (66.9%) and reduced the level of MDA as compared to cisplatin induced nephrotoxicity in rats. Crocetin significantly (P>0.001) suppressed the expression of HO-1 (40.9%), Akt (53.3%), PI3k (64.7%) and Nrf2 (62.6%). Crocetin treatment reduced the size of Bowman capsules and necrosis in the histopathology.

Conclusions: Finally, the result shows that crocetin exhibit an anti-fibrotic and anti-apoptotic effect against cisplatin-induced acute kidney injury via alteration of PI3K/Akt/Nrf2 signalling pathway.