Effect of Curcumin on Inflammation and Oxidative Stress in Cisplatin-Induced Experimental Nephrotoxicity

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Abstract:

Nephrotoxicity is a major complication and a dose limiting factor for cisplatin therapy. Recent evidence suggests that inflammation and oxidative stress may contribute to the pathogenesis of cisplatin-induced acute renal failure. Curcumin is claimed to be a potent anti-inflammatory and antioxidant agent. The present study was performed to explore the effect of curcumin against cisplatin-induced experimental nephrotoxicity. Curcumin in the dosages of 15, 30, and 60 mg kg^{-1} was administered 2 days before and 3 days after cisplatin administration. Renal injury was assessed by measuring serum creatinine, blood urea nitrogen, creatinine, urea clearance, and serum nitrite levels. Renal oxidative stress was assessed by determining renal malondialdehyde levels, reduced glutathione levels and enzymatic activities of superoxide dismutase and catalase. Systemic inflammation was assessed by tumor necrosis factor-alpha (TNF-α) levels. A single dose of cisplatin resulted in marked inflammation (486% rise in TNF-α level) and oxidative stress and significantly deranged renal functions as well as renal morphology. The serum TNF-α level was markedly reduced in curcumin-treated rats. Curcumin treatment significantly and dose-dependently restored renal function, reduced lipid peroxidation, and enhanced the levels of reduced glutathione and activities of superoxide dismutase and catalase. The present study demonstrates that curcumin has a protective effect on cisplatin-induced experimental nephrotoxicity, and this effect is attributed to its direct anti-inflammatory and strong antioxidant profile. Hence, curcumin has a strong potential to be used as a therapeutic adjuvant in cisplatin nephrotoxicity.

Keywords: Cisplatin; curcumin; inflammation; nephrotoxicity; oxidative stress; TNF-α.