

Carvedilol Ameliorates Potassium Dichromate-Induced Acute Renal Injury in Rats: Plausible Role of Inflammation and Apoptosis

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Abstract : Environmental and occupational exposure to hexavalent chromium [Cr(VI)] via textile manufacture, metallurgy, spray paints, stainless steel industries, drinking water containing chromium are often known to cause acute renal injury in humans and animals. Nephrotoxicity is the major effect of chromium poisoning. In the present study, we investigated the potential renoprotective effect and underlying mechanisms of carvedilol using rat model of potassium dichromate (K₂Cr₂O₇)-induced nephrotoxicity. Exploration of the underlying mechanisms of carvedilol revealed that carvedilol attenuated nuclear translocation and DNA binding activity of NF-κB (p65), restored antioxidant and mitochondrial respiratory enzyme activities and attenuated apoptosis related protein expressions in kidney tissues. The serum levels of TNF-α, the renal iNOS and myeloperoxidase activity were significantly decreased in carvedilol pre-treated K₂Cr₂O₇-induced nephrotoxic rats. These results were further supported and confirmed by histological findings. In conclusion, the findings of the present study demonstrated that carvedilol is an effective chemoprotectant against K₂Cr₂O₇-induced nephrotoxicity in rats.

Keywords : apoptosis, carvedilol, inflammation, potassium dichromate-induced nephrotoxicity, applied pharmacology

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