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Simvastatin attenuates chromium-induced nephrotoxicity in rats

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Hexavalent chromium, cr (vi) is used for various industrial applications. This chemical agent can cause numerous human diseases, including severe damage to the kidney.the wide environmental distribution of this chemical lead to an increase interest of preventive effects of its adverse effects. Simvastatin (simv) is widely clinically used for lowering hypercholesterolemia. It also has anti-inflammatory and anti-oxidant effects. The study of the effect of simv on cr (vi)-induced adverse effects on experimental animals may be useful for better understanding of the clinical pictures following cr (vi) exposure in humans. The present study was undertaken to investigate the potential protective effects of simv on cr (vi)-induced nephrotoxicity in rat. Forty-eight adult male wistar rats (180-220 g bw) were randomly assigned to eight groups (n = 6). Group one received simv 20 mg/kg/day. Group two was given vehicle only. Groups three, five and seven received intraperitoneally (i.p) cr (vi) at doses of 8, 12 and 16 mg/kg body weight. Groups four, six and eight pretreated with the 20 mg/kg simv 30 minutes to prior administration of cr (vi) at doses of 8, 12 and 16 mg/kg respectively. The experiment repeated for eight consecutive days. Twenty-four hours after the last administration, animals were killed with overdose of sodium pentobarbital. Kidney tissues were excised for measuring malondialdehyde (mda), glutathione (gsh) and histopathological examination. Results of the present study indicated that chromium induced a dose dependent elevation of mda and reduction of gsh levels when compared to those in control rats. Histopathological manifestations were observed in cr (vi)-treated rats. Simv administration restored cr (vi) produced biochemical and morphological changes in rat kidney. Simv decreased mda values and increased gsh levels in cr (vi)-treated rats. Simv clearly reversed the microscopic damage, demonstrating its protective effects against cr (vi)-induced kidney injury. The observations support the view that generation of oxidative stress is responsible for cr (vi)-induced nephrotoxicity. Simv may have a protective effect against cr (vi)-induced oxidative stress in rat kidney.

Biography

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