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ARTICLE



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ABSTRACT

Sodium arsenite (NaAsO_2) is one of the major environmental toxicants with severe toxicological consequences in some developing and developed countries. Rats in Group A received normal saline. Genotoxicity and apoptosis were induced by single intraperitoneal injection of 10 mg/kg sodium arsenite to rats in Groups B–F. Rats in Groups C and D had earlier been pretreated with *Azadirachta indica* (100 and 200 mg/kg) or E and F with vitamin E (50 and 100 mg/kg), respectively. Markers of oxidative stress, inflammation, hepatic damage, genotoxicity, and apoptosis were assessed. Pretreatment of rats with either *Azadirachta indica* or vitamin E led to a significant ($p < .05$) increase in the activities of glutathione-S-transferase (GST), catalase (CAT), superoxide dismutase (SOD), and reduced glutathione (GSH) in the liver compared to the group that received NaAsO_2 alone. Markers of oxidative stress and inflammation, malondialdehyde (MDA), hydrogen peroxide (H_2O_2) generation, nitric oxide (NO), and myeloperoxidase (MPO), were significantly ($p < .05$) lowered in rats pretreated with *Azadirachta indica* or vitamin E. The frequency of micronucleated polychromatic erythrocytes (MNPCEs) and expression of caspase-3 were significantly ($p < .05$) reduced in rats pretreated with either *Azadirachta indica* or vitamin E compared to rats intoxicated with arsenite. Histopathology of the liver showed areas of infiltration of inflammatory cells with deaths of numerous hepatocytes in NaAsO_2 -intoxicated rats, and these were reversed by *Azadirachta indica*. Together, we report for the first time the genoprotective and antiapoptotic effect of *Azadirachta indica* by a significant reduction in the frequency of micronuclei-induced apoptosis and oxidative stress by arsenic intoxication.

KEYWORDS

apoptosis; chemoprevention; genotoxicity; liver; sodium arsenite

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