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Therapeutic efficacy of silymarin from milk thistle in reducing manganese-induced hepatic damage and apoptosis in rats.

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Abstract

Oxidative stress has been proposed as a possible mechanism involved in manganese (Mn) toxicity. Using natural antioxidants against metal-induced hepatotoxicity is a modern approach. The present study investigated the beneficial role of silymarin, a natural flavonoid, in Mn-induced hepatotoxicity focusing on histopathology and biochemical approaches. Male Wistar rats were exposed orally to manganese chloride (20 mg/mL) for 30 days followed by intraperitoneal cotreatment with silymarin (100 mg/kg). Exposure to Mn resulted in a significant elevation of the plasma marker enzyme activities and bilirubin level related to **liver** dysfunction of reactive oxygen species (ROS) production and hepatic oxidative stress indices. This metal reduced the activities of superoxide dismutase, catalase and glutathione peroxidase and nonenzymatic antioxidant levels such as reduced glutathione, total sulfhydryl groups and vitamin C. In addition, it caused hepatic hemorrhage, cellular degeneration and necrosis of hepatocytes as indicated by **liver** histopathology and DNA fragmentation studies. Coadministration of silymarin alleviated Mn oxidative damage effects by inhibiting ROS generation. Histological studies also supported the beneficial role of silymarin against Mn-induced hepatic damages. Combining all, results suggested that silymarin could protect hepatic tissues against Mn-induced oxidative stress probably through its antioxidant activity. Therefore, its supplementation could provide a new approach for the reduction in hepatic complication due to Mn poisoning.

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