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The Protective Effect of Vanadium Sulphate on Ethanol-induced Gastric Ulcer

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Objective: To evaluate the protective effect of vanadium sulphate on ethanol-induced gastric ulcer in rats and its mechanism of action.

Setting: Department of Anatomy, King Khalid University, Saudi Arabia.

Design: Experimental animals study.

Method: Except for the control group (group I), gastric ulcer was induced in five groups of rats (II to VI), each consists of six. Ethanol was fed into rats which were pre-treated with distilled water, cimetidine, vanadium, selenium and a combination of vanadium and selenium (group II to VI respectively). The ulcer indices were determined in all these groups. Following macroscopic observations, specimens of the stomachs were taken and processed for histologic examination. Stomachs were then homogenized separately for each group and the supernatants were assayed for the activities of superoxide dismutase (SOD), catalase (CAT), levels of reduced glutathione (GSH) and thiobarbituric acid reactive substances (TBARS). Levels of these compounds from all groups were statistically analyzed for comparison.

Result: Rats pre-treated with the reference drug cimetidine showed more or less normal gastric mucosa. However, mild disruption of the surface mucosa was observed in rats receiving selenium and vanadium. Those receiving combination of selenium and vanadium showed almost normal mucosa. Furthermore, vanadium alone or in combination with selenium demonstrated a significant reduction of tissue lipid peroxidation levels, and potent ameliorative effect of the enzymatic and non-enzymatic components of the endogenous antioxidant systems.

Conclusion: Vanadium sulphate significantly inhibits lipid peroxidation and enhances the effects of enzymes that scavenge free radicals that are implicated in the pathogenesis of ethanol-induced ulcers in rats. Selenium seems to enhance its action and exerts a synergistic effect.

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