



Protective Effects of Rosmarinic Acid on Ethanol-Induced Gastritis in Male Rats

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ABSTRACT

Introduction: Alcohol increases superoxide anion and decreases non-protein sulfhydryl groups such as glutathione (GSH) in the mucosal layer. Superoxide anions then react with membrane lipids and produce lipid peroxides. Malondialdehyde (MDA) is a significant product of lipid peroxidation. Today, the main strategies for improving gastric injuries are proton pump inhibitors, histamine type 2 receptor blockers, and anti-acids. Rosmarinic acid (RA) is a synthetic compound commonly found in plants from the Lamiaceae family, including *Rosmarinus officinalis* (rosemary). Studies have revealed that RA decreases MDA as a marker of lipid peroxidation in the cell membrane and increases GSH and GSH peroxidase as antioxidant defenses. This study aimed to evaluate the effects of RA on preserving gastric mucosal layer integrity and glands and protecting gastric ulcers in ethanol-induced gastritis.

Methods and Materials: Animals were randomly divided into seven equal groups, six each, including a control group. After one hour of ethanol gavage, the animals received 50 mg/kg of sodium thiopental (intraperitoneal) and were sacrificed. The abdomen was exposed, and the stomach was removed.

Results: RA increased mucosal thickness, decreased gastric ulcers, enhanced the number of mucosal, chief, and parietal cells, decreased the presence of mucosal and submucosal leucocytes, reduced the number of gastric blood vessels in the mucosal and submucosal layers, increased gastric GSH levels, and decreased gastric MDA levels in ethanol-induced gastritis.

Conclusion and Discussion: Excessive alcohol consumption is considered one of the principal factors in gastric injuries. Investigations have also demonstrated that alcohol weakens the stomach's antioxidant/oxidant balance and prostaglandin (PG) synthesis. Our study demonstrated that RA significantly influences the preservation of gastric mucosal layer integrity and glands and impacts the protection of gastric ulcers in ethanol-induced gastritis. These effects may be due to the high antioxidant activity of RA and its role in raising PG secretion.

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