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Original Article

Effect of lecithin on oxidative stress in an experimental model of rats colitis induced by acetic acid

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ABSTRACT

Ulcerative colitis (UC) is an inflammatory disease that affects the bowels. Reactive oxygen species (ROS) are involved in the progress of UC.
Objective: Evaluate the antioxidant effect of lecithin in an experimental model of acute UC induced by administration of acetic acid (AA) in rats.
Methods: Lecithin (0.5 mL/kg/day) administered orally 2 days before and after induction of colitis with 4% AA in a volume of 4 mL. Twenty-five male Wistar rats were divided in 5 groups: control (CO); control + lecithin (CO + LE); colitis (CL); colitis + lecithin (CL + LE); lecithin + colitis (LE + CL). Anal sphincter pressure, LPO (TBARS), and antioxidant activity of enzymes superoxide dismutase (SOD) and catalase (CAT) were measured, and a histological analysis with H&E was performed.
Results and discussion: Anal sphincter pressure was significantly smaller in the CO group, lecithin treatment increased it in pre- and post-treated groups. LPO and SOD activity were increased in the CO group and decreased in the lecithin-treated groups. CAT activity was increased in CO group and decreased in lecithin groups. The histological analysis showed damage to the bowels with destruction of crypts, edema, and inflammatory infiltrate. Use of lecithin preserved the crypts and decreased the edema.

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Conclusion: Ulcerative colitis increased lipid peroxidation, and the use of lecithin was effective reducing damage to the bowels in the model of experimental colitis.

Efeito da lecitina sobre o estresse oxidativo no modelo experimental de colite induzida por ácido acético em ratos

Palavras-chave:
Retocolite ulcerativa
Doença inflamatória intestinal
Lecitina
Estresse oxidativo

RESUMO

A retocolite ulcerativa (RCUI) é uma doença intestinal inflamatória. Espécies reativas de oxigênio (ERO) estão envolvidas no progresso da RCUI.

Objetivo: Avaliar o efeito antioxidante de lecitina em modelo experimental de RCUI induzida pela administração de ácido acético (AA) em ratos.

Métodos: A Lecitina (0,5 mL/kg/dia) foi administrada por via oral 2 dias antes e após a indução de colite com AA. Vinte e cinco ratos Wistar machos foram divididos em 5 grupos: controle (CO); controle + lecitina (CO + LE); colite (CL); colite + lecitina (CL + LE); lecitina + colite (LE + CL). Foram avaliadas: pressão do esfíncter anal, lipoperoxidação (LPO), atividade antioxidante das enzimas superóxido dismutase (SOD) e catalase (CAT), e foi realizada uma análise histológica com H&E.

Resultados e discussão: A pressão do esfíncter anal foi significativamente menor no grupo CL, o tratamento com lecitina aumentou a pressão nos grupos pré e pós tratados. A LPO e atividade da SOD aumentaram no grupo CL e diminuiram nos grupos tratados com lecitina. A atividade da CAT foi aumentada no grupo CL e diminuiu nos grupos com lecitina. A análise histológica mostrou danos ao intestino com destruição das criptas, edema e infiltrado inflamatório. O uso de lecitina proporcionou uma preservação das criptas e diminuição do edema.

Conclusão: A RCUI aumenta a LPO, a utilização de lecitina foi eficaz na redução dos danos ao intestino induzido por AA no modelo de colite experimental.

Introduction

Inflammatory bowel disease (IBD) is characterized by chronic inflammation of the gastrointestinal tract, and numerous physiopathogenic mechanisms may be associated with its etiology, such as those of genetic, dietary, immunological, infectious, parasitical, post-radioactive, ischemic and environmental order.[1] Idiopathic ulcerative rectocolitis (IURC) and Crohn's disease are the most common forms of incidence of IBD and its etiology is not fully clarified.[2,3]

IBD poses a serious global health problem, as it affects primarily young people and has severe and chronic clinical presentations, occurring all over the world.[3]

Research suggests that oxidative stress may be important in the activity and development of IBD. Other studies showed that reactive oxygen species (ROS) are generated in excess in individuals with colitis as compared to normal individuals.[2,4]

The experimental model of colitis performed by Fillmann et al.[1] suggests that besides ROS, nitric oxide is involved in this situation, triggering inhibitory action on smooth muscles, promoting relaxation of the anal sphincter and thereby a decrease in anal sphincter pressure levels.

The increase in the generation of ROS in ulcerative colitis triggers an imbalance in the cell redox status and thereby an increase in free radicals (FR). Such increase overwhelms the antioxidant defense capabilities of the cell, thus characterizing oxidative stress (OS), which in turn triggers lipid peroxidation (LPO), leading to disruption of disulfide bridges of lipids by breaking them and loss of cell integrity, destabilizing it and leading to cell death.[5]

The organism has a defense system against oxidant agents composed of enzymatic activity, i.e., enzymes superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx); and non-enzymatic agents, such as glutathione (GSH), vitamins (A, C, E), flavonoids, and other compounds present in food, such as lecithin. The function of antioxidants (AOX) is to maintain the redox balance by keeping ROS levels low, thereby preventing the formation of free radicals such as superoxide anion, hydrogen peroxide, and the most harmful one, hydroxyl radical.[1,2,5]

Many foods have important AOX action, and included in the diet they maintain the balance between oxidant production and antioxidant defenses. Lecithin can be found in soy, peanuts, spinach, wheat and mainly in eggs yolk, so being an important ally for the redox balance.[6] The distinctive characteristic of lecithin is being amphoteric, with a polar and an