The Effect of Melatonin on Plasma Markers of Inflammation and on Expression of Nuclear Factor-Kappa Beta in Acetic Acid-Induced Colitis in the Rat

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Abstract:

Background and Aims Melatonin may be involved in gastrointestinal tract physiology and could affect inflammation-related gastrointestinal disorders. Rat models of ulcerative colitis imply melatonin is beneficial. To determine potential pathophysiological mechanisms, we assessed colonic nuclear factor-kappa beta expression and measured serum levels of pentraxin-3, lipid peroxides, and total thiols in an acetic acid model of this disease. Materials and Methods Thirty rats were divided into five groups: a control group, an acetic acid-induced colitis group, a group treated with melatonin before colitis induction, a group treated short-term after colitis induction, and a group treated long-term after colitis induction. After four weeks, blood samples were taken for measurement of pentraxin-3, lipid peroxide, and total thiols. Sections of the colon were taken for histopathological examination and immunohistochemical detection of nuclear factor-kappa beta expression. Results Melatonin administration reduced nuclear factorkappa beta immunohistochemical expression, reduced serum levels of lipid peroxide and pentraxin-3, and maintained serum levels of total thiols. However, in long-term treatment the protective effect of melatonin was not as marked. Conclusion Melatonin is effective in prevention and short-term treatment of the inflammatory process in acetic acid induced colitis whereas the benefit of long-term treatment is unclear. Benefit may be linked to protection mechanisms against inflammatory processes by inhibiting the nuclear factor-kappa beta and conserving endogenous antioxidant reserves of total thiols, thus reducing the level of colonic damage possibly caused by lipid peroxides.

Keywords:

Keywords Melatonin●Acetic acid-induced colitis● Nuclear factor-kappa beta● Pentraxin-3

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