

Effects of Oral Zinc Sulfate on Induced Colitis in Rabbits

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Abstract

Back ground: The failure of current treatment strategies to control many cases of IBD makes a strong stimulus to find out new modalities of treatment.

Aims: to study the effects of zinc sulfate on induced colitis in rabbits.

Materials and Methods: Colitis was induced in rabbits by rectal acetic acid-ethanol (model 1), or acetic acid (model 2). The effects of zinc sulfate were compared to distilled water (control), and prednisolone regarding changes in body weight, colon segment weight, and gross and microscopical scores. Plasma zinc and copper concentrations were measured in control and zinc sulfate groups in both models.

Results: In model 1, severe gross and microscopical damage observed in colon. Gross and microscopical scores of zinc sulfate group were not significantly different from that of control and of prednisolone groups.

In model 2, a less severe inflammation occurred; yet, an evident gross and microscopical damage were observed.

Zinc sulfate and prednisolone treatment reduced the loss of body weight of rabbits in comparison to the control. The gross and microscopical damages were significantly lowered in zinc sulfate and prednisolone groups.

In both models (1 and 2), a significant decrement in post induction mean plasma zinc level was detected ($p < 0.05$); however, such decrement could be corrected by zinc sulfate therapy.

Conclusions: Acetic acid -induced colitis in rabbits (model II) is preferred for testing the anti-inflammatory effectiveness of new therapeutic modalities. Zinc sulfate has a valid prophylactic activity in this model.

Keywords: Inflammatory bowel disease, induced colitis, acetic acid, zinc sulfate, prednisolone.

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Introduction

Idiopathic inflammatory bowel disease (IBD) comprises those conditions characterized by a tendency for chronic or relapsing immune activation and inflammation within the gastrointestinal tract ⁽¹⁾, Crohn's disease (CD) and ulcerative colitis (UC) are the two major forms of idiopathic IBD ⁽²⁾. Ulcerative colitis and CD pursue a protracted, relapsing and remitting course, usually extending over years ⁽³⁾.

Recent studies pointed to the important role of free oxygen radicals in the pathogenesis of IBD both in animal models of induced colitis and in human beings.

one of the more commonly used models of Induced Colitis in Rabbits is acetic acid induced colitis ⁽⁴⁾. This experimentally induced colitis is similar to the human condition in certain aspects (e.g., acute inflammation with neutrophil infiltration ⁽⁵⁾, increased concentrations of LT B4, and PG E2 ⁽⁶⁾, superoxide dismutase ^(7,8) and increased production of inflammatory mediators, such as hydrogen peroxide (H₂O₂), nitric oxide (NO), myeloperoxidase

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