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Abstract.

Rebamipide contributes to the improvement of blood supply of the GI mucosa, activates its barrier function, activates alkaline secretion of the stomach, increases proliferation and metabolism of epithelial cells of the GI tract, cleanses the mucosa from hydroxyl radicals and suppresses superoxides, produced by polymorphonuclear leukocytes and neutrophils in the presence of Helicobacter pylori, protects the GI mucosa from bacterial invasion and the damaging effects of non-steroidal anti-inflammatory drugs (NSAIDs) on the mucosa. Rebamipide, originally developed as a treatment for gastric ulcers, has attracted the attention of researchers as a potential drug for the treatment of UC due to its ability to stimulate mucus production, reduce oxidative stress, and decrease inflammation. Due to the presence of these properties, it is hypothesized that rebamipide may have a protective effect on the intestinal mucosa during prolonged inflammation, making it a promising candidate for inclusion in therapeutic strategies for ulcerative colitis. The results of this study suggest that rebamipide holds potential therapeutic benefits for the treatment of ulcerative colitis.

Key words. Rebamipide, colitis, rats.

Introduction.

Rebamipide is a drug - gastrocytoprotector, which increases the content of prostaglandin E2 (Pg E2) in the gastrointestinal (GI) mucosa and increases the content of prostaglandins Pg E2 and Pg I2 in the contents of gastric juice [1-3]. The drug has a cytoprotective effect on the mucosa of the GI tract under the damaging effects of ethanol, acids and alkalis, acetylsalicylic acid. It promotes the activation of enzymes that accelerate the biosynthesis of high molecular weight glycoproteins and increases the content of mucus on the surface of the GI mucosa walls [2-5]. Contributes to the improvement of blood supply of the GI mucosa, activates its barrier function, activates alkaline secretion of the stomach, increases proliferation and metabolism of epithelial cells of the GI tract, cleanses the mucosa from hydroxyl radicals and suppresses superoxides, produced by polymorphonuclear leukocytes and neutrophils in the presence of Helicobacter pylori, protects the GI mucosa from bacterial invasion and the damaging effects of non-steroidal anti-inflammatory drugs (NSAIDs) on the mucosa [3,4,6,7]. Rebamipide is widely used for the treatment of acute and chronic gastritis and peptic ulcer [1-10]. It is already known that rebamipide has anti-inflammatory properties, reducing the production of free radicals, inhibiting pro-inflammatory cytokine production, inhibiting migration and adhesion of inflammatory cells [1,5,6,9]. To date, indications for the use of rebamipide are limited to the treatment of peptic ulcer disease, chronic gastritis, prevention of mucosal lesions on the background of NSAIDs [1-10]. The study of pharmacological possibilities of using rebamipide in ulcerative colitis is an urgent problem.

Ulcerative colitis (UC) is a chronic inflammatory bowel disease that affects any part of the colon and is characterized by a progressive course with periods of exacerbation and remission [2,8,10]. This disease significantly reduces the quality of life of patients due to pain, diarrhea, and other dysfunctions of the digestive system [1-10]. According to recent data, the prevalence of UC in European countries varies from 2.4 to 294 cases per 100,000 population and tends to increase. Inflammation in UC usually begins in the rectum and may spread to a large part of the colon, leading to erosions and ulcers, disruption of mucosal integrity and, consequently, deterioration of the functional activity of the intestine [2-4]. Common morphological features of UC are thickening of the intestinal walls, reduction in the number of papillae, increased vascularization, and permeability of the mucosa [2-7].

In addition, rebamipide has an antioxidant effect, neutralizing free radicals that often accumulate in tissues during chronic inflammation and can exacerbate mucosal damage. Through this antioxidant effect, rebamipide helps reduce oxidative stress and promotes regeneration processes [8-10].

Another important characteristic of rebamipide is its ability to increase mucosal blood flow, which plays a critical role in maintaining mucosal health and mucosal repair in inflammatory bowel disease. By improving microcirculation, tissues receive more oxygen and nutrients, which speeds up their recovery [5, 9].

Rebamipide may also modulate the immune response by reducing the production of inflammatory cytokines such as TNF-α and IL-6, which are actively involved in the development and maintenance of inflammation in the gut [1-10]. The regulation of these cytokines may reduce the degree of inflammation and reduce the symptoms of ulcerative colitis [5-8].

In animal models such as rats, UC can be induced by a variety of methods, including chemical exposure, such as with dextran sodium sulfate (DSS) or 2,4,6-trinitrobenzene sulfonic acid (TNBS), which lead to the development of symptoms and morphologic changes characteristic of the disease. These models allow us to evaluate the efficacy of new therapeutic agents, study immune responses, and investigate molecular pathways of inflammation and tissue repair [1,5,8].
Modulation of the inflammatory response and mucosal defense may be one of the avenues of therapy for UC [1,6,9]. Rebamipide, originally developed as a treatment for gastric ulcers, has attracted the attention of researchers as a potential drug for the treatment of UC due to its ability to stimulate mucus production, reduce oxidative stress, and decrease inflammation. Due to the presence of these properties, it is hypothesized that rebamipide may have a protective effect on the intestinal mucosa during prolonged inflammation, making it a promising candidate for inclusion in therapeutic strategies for ulcerative colitis [1-10].

Few studies report the therapeutic value of rebamipide in the treatment of ulcerative colitis and proctitis. K. Makiyama et al. (2005), using a sample of 11 patients with mild to moderately active UC who continued conventional treatment of the disease, showed that enemas with rebamipide at a dosage of 150 mg twice daily improved the course of UC in 90.9% of patients. Moreover, complete clinical and endoscopic remission was achieved in 45.5% of UC patients. In another prospective non-comparative study by M. Miyata et al. (2005) including 11 patients with steroid-resistant and/or steroid-dependent UC, 81.8% of patients achieved remission with a 12-week course of treatment with rebamipide enemas. Thus, these clinical data suggest that rebamipide is promising in terms of its potential to repair intestinal damage. Rebamipide has the ability to inhibit neutrophil activity, induce regression of lipid peroxidation, and stimulate regeneration of epithelial cells of the GI mucosa, which makes it a potential agent for the treatment of UC when administered in the form of enemas. However, the detailed molecular mechanism of action of rebamipide against intestinal inflammation remains unclear.

The aim of the study was to experimentally evaluate the effect of rebamipide on the course of ulcerative colitis in adult rats.

Materials and Methods.

A series of experiments were conducted to study the effect of rebamipide on the course of ulcerative colitis in laboratory rats. The tests were conducted on a group of adult male Wistar rats with a total of 60 individuals. All studies were conducted according to the requirements of the decision of the Council of the Eurasian Economic Union in the Sphere of Circulation of Medicines from 03.11.2016 No 81. Animals were kept in vivarium conditions at a maintained temperature of 24-25°C, light regime -12/12.

The animals were divided into 4 groups: control group (n=10), group with induced colitis (n=10), group receiving rebamipide without colitis (n=20), and group with colitis treated with rebamipide (n=20).

Ulcerative colitis was induced in rats of the second and fourth groups by administration of 2% dextran sodium sulfate (DSS) solution in drinking water for 7 days. After induction of colitis, animals of group four were started to receive rebamipide at a dose of 10 mg/kg body weight daily through a tube, once a day for 14 days.

Treatment efficacy and the effect of rebamipide on the course of colitis were evaluated using clinical assessments, histologic analysis of colonic tissue, and biochemical markers of inflammation. Clinical assessments included body weight, degree of diarrhea, and presence of bleeding. Histologic specimens were taken after the end of the experimental period, stained with hematoxylin and eosin and evaluated for morphologic signs of inflammation.

Evaluation of Disease Activity Index (DAI): the disease activity index was used to assess the severity of ulcerative colitis in the rats. The index evaluated three parameters: body weight loss, stool consistency, and rectal bleeding. Body weight was measured daily, and the severity of diarrhea and rectal bleeding were visually assessed and scored on a scale ranging from 0 to 4. The scores of the three parameters were summed to calculate the disease activity index for each rat.

Biochemical Analysis: after 14 days of treatment, the rats were sacrificed, and blood samples were collected for biochemical analysis. The levels of inflammatory markers such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha) were measured using enzyme-linked immunosorbent assay (ELISA) kits. Additionally, myeloperoxidase (MPO) activity, an indicator of neutrophil infiltration in the colon, was assessed.

Statistical processing of the data was performed using the t-criterion of Student's t-test. The criterion of statistical reliability of the obtained conclusions was considered to be the value of $p < 0.05$, which is generally accepted in medicine.

Results.

All rats receiving 2% dextran sodium sulfate (DSS) solution in drinking water for 7 days developed symptoms of ulcerative colitis in 100% of cases: diarrhea (on the 4th day), rectal bleeding, and decreased body weight. The damage index in group 2 and group 4 (before treatment with rebamipide) was 276.8 ± 25.4 mm2 and 287.5 ± 21.2 mm2, respectively.

Rebamipide administration to rats of the fourth group (10 mg/kg) decreased the frequency of diarrhea, the degree of rectal bleeding severity, and decreased the degree of body weight loss ($p<0.05$). Intestinal mucosal damage in this group was 174.5 ± 31.7 mm2.

We evaluated the body weight changes in rats before and after the induction of colitis. The results showed a significant decrease in body weight in the colitis group compared to the control group ($p<0.05$). However, rats treated with rebamipide exhibited less weight loss compared to the colitis group ($p<0.05$), indicating a potential protective effect of rebamipide on body weight loss.

To further assess the severity of colitis, we used the Disease Activity Index (DAI) scoring system, which includes parameters such as weight loss, stool consistency, and rectal bleeding. The DAI scores were significantly higher in the colitis group compared to the control group ($p<0.05$). However, rebamipide-treated rats showed a lower DAI score compared to the colitis group ($p<0.05$), suggesting an improvement in colitis symptoms with rebamipide treatment.

Histologic analysis confirmed less damage and inflammation of the intestinal wall in rats treated with rebamipide. A decrease in neutrophil and macrophage infiltration and preservation of the intestinal mucosa structure were recorded.

Biochemical assays also confirmed decreased levels of inflammatory markers such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), indicating an anti-inflammatory effect of rebamipide.
Conclusion.

The results of this study demonstrated that rebamipide significantly reduced the disease activity index in rats with ulcerative colitis compared to the control group. The histological examination revealed a significant decrease in the severity of inflammation, ulceration, and epithelial damage in the rebamipide group. Additionally, rebamipide administration resulted in a significant reduction in the levels of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6).

These findings suggest that rebamipide has potential therapeutic benefits for the treatment of ulcerative colitis. Rebamipide exerts its anti-inflammatory effects by suppressing the production of pro-inflammatory cytokines, thereby reducing the inflammation and mucosal damage in the colon. This anti-inflammatory action of rebamipide makes it a promising candidate for the treatment of ulcerative colitis, especially in cases where conventional therapies have failed or have limitations.

The implications of these results for the treatment of ulcerative colitis in humans are significant. Rebamipide could potentially offer an alternative treatment option for patients with ulcerative colitis, particularly those who do not respond well to current available therapies.

Moreover, rebamipide may have a favorable safety profile and can be easily administered orally, which makes it a more patient-friendly choice compared to other treatments that may require invasive procedures or have systemic side effects.

However, further studies are required to validate these results and investigate the long-term effects and optimal dosage regimen of rebamipide in ulcerative colitis. Additionally, clinical trials involving human subjects are necessary to determine the efficacy and safety of rebamipide in the treatment of ulcerative colitis. Nevertheless, the findings from this experimental study provide a promising basis for future research and offer hope for improving the management of ulcerative colitis in humans.

In conclusion, the results of this study suggest that rebamipide holds potential therapeutic benefits for the treatment of ulcerative colitis. Rebamipide reduces inflammation, mucosal damage, and disease severity in an experimental model of ulcerative colitis in rats. These findings highlight the need for further research and clinical trials to evaluate the efficacy and safety of rebamipide in human subjects. Rebamipide may represent a promising addition to the current treatment options for ulcerative colitis, offering new possibilities for patients who do not respond well to conventional therapies or experience limitations in their use.

REFERENCES