USE OF THE DRUG "KOBAVIT" TO PREVENT COMPLICATIONS FROM THE GASTROINTESTINAL TRACT IN BURN DISEASE

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Abstract

The use of cobavit for the prevention of gastrointestinal complications in burn patients demonstrated superior efficacy in 33 patients compared to standard therapy in 30 patients.

Key words: *burn disease, complication, prevention.*

One of the serious complications of severe burn disease is the development of acute gastroduodenal erosions and ulcers. Gastroduodenal hemorrhage (GDH) occurs in 15-25% of patients with severe burns and most often occurs in the burn toxemia stage. The relevance of this issue is due to the frequency of this complication in patients with extensive wound processes of various etiologies, which increases the cost of treatment for this group of patients, prolongs recovery time, and sometimes leads to unfavorable outcomes. In recent years, interest in acute erosive and ulcerative lesions of the gastrointestinal tract in patients with severe burns has increased due to their increasing frequency, the difficulty of timely diagnosis, and the low effectiveness of conservative and surgical treatment [2].

One of the main causes of acute damage to the stomach and duodenum (DU) are critical conditions such as severe thermal injury, sepsis, multiple injuries, etc. [4].

Acute symptomatic ulcers of the upper gastrointestinal tract (ASU) are typically defined as acute, often superficial, and multiple erosive-ulcerative lesions of the upper gastrointestinal tract (EGT) that occur in patients with severe burns. The most common cause of acute upper gastrointestinal bleeding is thermal injuries, accounting for approximately 30-40% of cases. Developing EGTs not only significantly aggravate the course of burn disease but also increase the mortality rate to 70-80% [4].

Conducting preventive antisecretory therapy allows us to reduce the incidence of GDC in victims of this category to 7–11% [5].

A comprehensive approach to the prevention of GDC, including infusion, metabolic and antisecretory therapy, as well as early enteral support, can reduce the incidence of this complication of burn disease to 5–7% [5].

The pathogenesis of developing bleeding remains poorly understood. According to laboratory studies, shockogenic thermal injury leads to a significant increase in cortisol secretion, the level of which remains elevated for 10–14 days of treatment [2].

Excess hydrochloric acid causes erosive and ulcerative lesions of the stomach and duodenum. These changes are multiple and most often localized in areas of the mucosa that directly produce hydrochloric acid and pepsin, that is, in the fornix and body of the stomach [1].

This causes a significant amount of fluid to leak into the interstitial space, leading to hypovolemia, hemoconcentration, and centralized circulation, leading to microcirculation impairment. A common manifestation of developing compensatory vasoconstriction is a 50–60% reduction in blood supply to the gastric mucosa. Persistent spasm of the celiac vessels with impaired arterial perfusion and venous outflow leads to blood stagnation, plasma leakage into the extravascular space, local hemoconcentration, and subsequent microthrombosis, resulting in areas of ischemic necrosis of the mucosa [1].

Burn injury provokes an immediate response from all organs and systems, which are not always able to maintain homeostasis and are often subject to pathophysiological and morphological damage. One such target system is the gastrointestinal tract. After severe burns, intestinal ischemia and hypoxia disrupt the intestinal epithelial barrier and intestinal bacterial translocation, leading to serious complications such as systemic inflammatory response syndrome, sepsis, and multiple organ failure. Peritonitis or gastrointestinal bleeding accounted for 88.2% of deaths among patients with gastrointestinal dysfunction [1-5].

Gastric damage is caused by an imbalance between the aggressive and protective factors to which the gastric mucosa is exposed; when the protective agents are overloaded and therefore unable to provide sufficient protection, damage occurs as a result of histopathological changes [1-3].

Fundamental and current data on ulcers occurring in burn patients has led to widespread recognition of ulcer prevention. Along with antisecretory therapy using proton pump inhibitors (PPIs), a gentle diet, eliminating unhealthy habits, and stress prevention also play an important role [3].

Mixed protein supplementation reduces the systemic inflammatory response induced by burn injury, attenuates inflammatory cell infiltration, and

improves kidney and liver function. Mixed protein supplementation is known to reduce burn-induced inflammatory cell infiltration and protect liver and kidney function. This study suggests a new and promising potential nutritional therapy for burn patients aimed at reducing the inflammatory response and protecting organ function [2,5].

In Uzbekistan, scientists have developed a drug called Cobavit. It is a complex compound of cobalt, glutamic acid, and vitamin U, possessing antioxidant, immunostimulatory, and inductive effects. The drug also stimulates hematopoiesis and has a positive effect on ulcer healing. However, its effect on the protective mechanisms of the mucous membrane has not been sufficiently studied [2-3].

The aim of the study: to study the effectiveness of using cobavit in combination therapy for the prevention and treatment of duodenal ulcer in burn disease.

Materials and methods: Sixty-three patients aged 18 to 55 years who suffered from burns and as a result had pathological changes in the gastrointestinal tract in the Bukhara branch of the Republican Scientific Center for Emergency Medical Care in 2023-2025 were examined in the Burn and Toxicology Resuscitation Department. Of these, 31 were men and 32 were women. The patients were divided into two groups. The first group (30 patients) received proton pump inhibitors (PPIs): omeprazole, pantoprazole (40 mg/day 2 times a day) for 10 days. The second group (33 patients) received cobavit in a daily dose of 20 mg (1 tablet 2 times a day) for 10 days in addition to a traditional PPI.

The diagnosis of pathological changes in the gastrointestinal system was confirmed by clinical, instrumental and laboratory research methods. Inclusion criteria for the study: patients over 18 years of age, esogastroduodenoscopy examination, patient consent to the study.

All patients underwent a comprehensive clinical, laboratory and instrumental examination. Random selection of patients in research groups was carried out taking into account age and gender composition, causes of appearance, clinical appearance, results of laboratory and instrumental research methods. Treatment effectiveness was evaluated based on the following parameters:

1) changes in the patient's subjective complaints and objective disease manifestations;

2) the condition of the mucous membrane of the stomach and duodenum was studied based on the results of endoscopic examination.

Results and discussion: Before treatment, epigastric pain was observed in 24 patients, heartburn in 12 patients, and nausea in 10 patients out of all 33 patients in the second group. The proportion of clinical symptoms in the 30 patients in the first group did not differ significantly from the first group: epigastric pain was observed in 25 patients, heartburn in 15 patients, and nausea in 14 patients. After a 10-day course of treatment, the main symptoms of the disease (pain, heartburn, nausea, bleeding) disappeared in the first and second groups. In addition, as shown in Table 1, the time of disappearance of the main clinical symptoms was observed much earlier in patients receiving combination therapy with Cobavit than in patients receiving PPIs.

Table 1.

Dynamics of disappearance of the main clinical symptoms in patients in the analyzed groups

Patient group	**Patients**	**Epigastric pain**	**Nausea**	**Boiling of the spleen**
Proton pump inhibitor (first group)	30	6,8±0,24*	6,3±0,48	6,1±0,31*
Proton pump inhibitor+cobavit (second group)	33	4,2+±0,16	5,0±0,26	3,4±0,22

* – significance of changes compared to the comparison group at r<0.05.

The study showed that patients in both groups had new ulcers detected by endoscopic examination. Ulcers ranging from 3 to 9 mm predominated in both groups. The average ulcer size in group 2 was 6.58±0.77 mm, and in group 1 it was 6.72±0.49 mm.

Particular attention should be paid to comparing the reparative effectiveness of combination therapy with Cobavit and the treatment regimen with PPIs. After a ten-day course of combination therapy (group 2), ulcer healing was observed in 28 out of 33 patients (84.8%). The remaining 5 patients had a significant positive endoscopic dynamics, which was manifested by a 2-3-fold decrease in ulcer volume (the average residual ulcer volume was 2.2±0.35 mm).

In group 1 patients (PPI was used) during controlled esophagogastroduodenoscopy, 10 days after the start of treatment, only 19 out of 30 patients (63.3%) had complete wound healing. In eleven patients (36.6%), the wound size decreased by 2-3 times (on average to 2.8±0.7 mm). These observations indicate a significant reparative effect of combined therapy in the kobavit group: wound healing was observed in almost 86.6% of patients within 10 days after treatment.

Thus, the results of the study partially confirm the ability of combined gastroprotection against acute ulcers in combination with Cobavit to enhance the differentiation of the newly formed mucosa and stimulate epithelialization of the ulcer defect. In both analyzed groups of patients with acute gastrointestinal ulcers, a direct relationship was observed between the effectiveness of the ulcer defect scar and its initial size. With PPI, small ulcers were scarred in 89.8% of cases, and medium ulcers in 48.2%. Combination therapy with Cobavit led to the same degree of scarring for small and medium-sized ulcers (91.2%). When analyzing the frequency of ulcer scarring by groups depending on the severity of acute ulcers, a progressive decrease in the frequency of scarring was observed with increasing severity of the disease. However, while the incidence of wound scarring was similar in mild and severe cases (97.3% and 2.3%), combination therapy with Cobavit was 2.3 times more effective than PPI therapy in patients with moderate disease (41.3% in the first group and 92.5% in the second group). These observations further confirm the high reparative effect of combination therapy with Cobavit and allow us to predict the course of wound healing.

Therefore, the positive changes observed in the second group, which additionally received Cobavit, are probably due to the presence of glutamic acid and vitamin U in Cobavit, which have a strong antioxidant and reparative effect. The active substance of vitamin U is methylmethionine, which normalizes the secretion of hydrochloric acid and accelerates the healing of the resulting wound. The element cobalt also plays an important role. As a biogenic microelement, cobalt is used to synthesize cobalamin and has an anti-anemic effect, which is important for the treatment of acute wounds. Since the healing wound contained acute wounds caused by burns, 2 types of antibiotics, which are part of the triple therapy for the treatment of ulcer disease, were not added to the healing wound. The inclusion of Cobavit in the combination therapy of patients with peptic ulcer disease has been proven to be effective.

Conclusion: In patients with peptic ulcer disease, the use of PPIs in combination with cobavit helps to reduce the duration of clinical symptoms and

ulcer healing time in the gastrointestinal mucosa compared to standard conventional therapy.

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