

PREVENTION OF ACUTE EROSIVE AND ULCERATIVE LESIONS OF THE UPPER GASTROINTESTINAL TRACT IN PATIENTS WITH EXTENSIVE THERMAL BURNS

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Abstract

The article explores the prevention of acute erosive and ulcerative lesions in the upper gastrointestinal tract of patients suffering from extensive thermal burns. It discusses the high incidence of these complications, often leading to severe outcomes like bleeding and multiple organ failure. Through endoscopic research, the early development of ulcers post-burn is detailed, along with their treatment using conservative methods primarily, including the use of proton pump inhibitors like omeprazole. The study also highlights the importance of understanding the pathogenesis of these ulcers, emphasizing the role of increased acid production and delayed gastric motility in their development.

Keywords: Acute erosive and ulcerative lesions, Upper gastrointestinal tract, Thermal burns, Hemodynamic disorders, Gastric motility, Proton pump inhibitors.

Introduction

Acute erosive and ulcerative lesions of the gastrointestinal tract quite often occur in the form of complications in patients after burns. Complications of acute ulcers in the form of bleeding occur in 18-23% of patients [1]. In burn patients, even low-intensity bleeding dramatically worsens the general condition, which is manifested by general weakness, lethargy, pallor of the skin, and hemodynamic disorders. Some acute ulcers and erosions are capable of independent scarring and epithelialization, but their clinical picture is poorly expressed [1, 2, 3].

In the absolute majority of cases, uncomplicated acute erosions and ulcers, without specific clinical manifestations, remain unrecognized and are detected only at autopsy. According to pathologists, acute ulceration of the mucosa is detected in 20-50% of those who died after extensive burns of the body, as a result of developing multiple organ failure.

Materials of endoscopic research

Based on the data of endoscopic examination, we found that in patients with extensive burns, acute ulceration of the gastric and duodenal mucosa develops within the next 3-5 days. In the early period, mucosal changes were alternated between foci of pallor and hyperemia. After one day, petechiae and surface erosions with a diameter of up to 1-2 mm occurred, and after 46-48 hours, the size of damage to the gastric mucosa became more significant. Under favorable conditions, the gastric mucosa was restored after 10-14 days, and the erosions disappeared.



However, in some severe patients, they persisted for up to 20-24 days and were often complicated by bleeding.

Signs of ongoing bleeding were observed in 20-25% of patients. At the same time, multiple erosions of hemorrhagic type up to 1-2 mm in diameter were most often detected.

When analyzing the results of endoscopic studies, erosive and ulcerative lesions were most often (67%) localized in the stomach, somewhat less often (43%)—in the duodenum and esophagus (18%). The question of the timing of the formation of stress ulcers is of interest, since the definition of a "critical period" allows doctors to focus their efforts on the effective diagnosis and prevention of bleeding. It is established that the time of ulceration depends on the causes and mechanisms that cause it. A comprehensive assessment of the acid-forming function of the stomach showed that the first 10 days show maximum stimulation of the acid-forming function of the stomach, while its "peak" occurs on the 3rd-5th day, i.e. during the period of the most likely ulcer formation.

The data of daily pH-metry significantly deepened the understanding of the state of acid-forming function of the stomach in patients with extensive burns. On day 1 after the injury, in 81% of cases, $\text{pH} < 1$ was recorded in the stomach body, i.e. there was an extremely high production of free hydrochloric acid.

The study of gastric motor activity in the early postoperative period in burn patients showed that in the 1st-2nd day of gastric motility is absent in all patients. On day 3, low-amplitude waves appear with a frequency of 3 contractions per minute, which do not provide adequate evacuation from the stomach. On day 4, periodic motor activity appears, but the amplitude of the contraction waves does not reach the normal level. Clinically, patients are determined by intestinal peristalsis and gas discharge. By the 6th-8th day, the evacuation function of the stomach was restored with an uncomplicated course of burn disease.

Studies have shown that an increase in acid production occurs against the background of inhibition of gastric motility.

Violation of evacuation of acidic gastric contents, which depends on peristaltic activity, leads to prolonged exposure of the acid-peptic factor on the gastric mucosa, which, together with other causes, significantly increases the risk of ulceration.

The marked reaction of the digestive system to traumatic stress underlies the formation of early true stress ulcers, which account for approximately 80% of all ulcers of the upper gastrointestinal mucosa. In the remaining 20% of patients, ulcers occur in the phase of mucosal dystrophy in a more distant period with a complicated course of burn disease in the form of cardiovascular, renal and respiratory failure, as well as purulent and septic complications leading to the development of multiple organ failure, one of the manifestations of which is ulcers.

According to our data, up to 92% of patients with erosive and ulcerative lesions of the upper gastrointestinal tract were treated with conservative methods. Surgical treatment was performed only for complications in the form of perforation of the hollow organ, profuse or persistent recurrent bleeding with ineffectiveness of endoscopic hemostasis methods. We have widely used proton pump inhibitors, in particular omeprazole. Omeprazole, which is a weak base, is ineffective at neutral pH. However, in the acidic environment of the tubules of parietal cells, it is converted to the active metabolite sulfenamide, which irreversibly inhibits the membrane $\text{H}^+ - \text{K}^+ - \text{ATPase}$. The conversion of omeprazole to sulfenamide occurs quickly (after 2-4 minutes), it effectively suppresses basal and irritant-induced HCl secretion, reduces the total volume of



gastric secretion and inhibits the release of pepsin. Currently, drugs of this group have antisecretory activity, incomparable in strength with any other drugs. The maximum concentration of omeprazole in plasma occurs 1-3 hours after oral administration. By suppressing enzymatic synthesis for 18 hours, the drugs provide a 24-hour duration of the acid-suppressing effect, regardless of the dose [4, 5, 6].

New ideas about the pathogenesis of acute ulceration of the gastric mucosa in patients with burn injuries can effectively affect the protective properties of the mucous membrane, in particular, correct microcirculatory and metabolic disorders in it, as well as eliminate post-traumatic immunosuppression. In this regard, a drug with antioxidant and antihypoxic properties - actovegin-attracts our attention.

The drug has a multifunctional effect, which makes it possible to eliminate metabolic disorders in tissues, in particular in the gastric mucosa, associated with endotoxemia and blood loss under conditions of hypoxia. It is used, as a rule, in the form of intravenous drip infusions of up to 250 ml of a 10% solution per day. This drug dramatically increases the resistance of gastric mucosal cells to proteolytic aggression of gastric juice.

Thus, erosive and ulcerative lesions of the upper gastrointestinal tract are one of the most common complications of burn disease. Complications of acute ulcers in the form of bleeding are accompanied by high mortality. One of the leading factors underlying the pathogenesis is a significant and persistent increase in acid-peptic aggression. The leading component of prevention of erosive and ulcerative lesions of the stomach in burn disease is the early, combined use of modern antisecretory drugs.

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