Prevention Of Reflux Esophagitis After Gastrectomy

Abdurazzak A. Kasymov

Head of the Department of Oncologic Surgery,
PhD (Candidate of Medical Sciences) *Republican Scientific and Practical Medical Center of Oncology and Radiology Tashkent Regional Branch*
Dr_abdurazzak@mail.ru. Orsid.0009-0008-5442-1763

ABSTRACT

Reflux esophagitis is one of the most common and clinically significant complications after gastrectomy. Its development is associated with the loss of the reservoir and barrier functions of the stomach, disruption of anatomical and physiological antireflux mechanisms, and altered gastrointestinal motility. This condition significantly reduces patients' quality of life and may lead to erosive and ulcerative lesions of the esophagus. The article discusses modern approaches to the prevention of reflux esophagitis after gastrectomy, including surgical, pharmacological, and dietary methods. Special attention is given to reconstructive surgical techniques aimed at reducing duodenogastroesophageal reflux.

Keywords: gastrectomy, reflux esophagitis, prevention, gastrointestinal reconstruction, esophagus, duodenogastric reflux, antireflux techniques

Gastrectomy is a radical treatment method for gastric cancer and other severe diseases of the stomach. Despite the high effectiveness of this operation, it is associated with a number of functional complications, among which reflux esophagitis occupies an important place.

After removal of the stomach, the natural antireflux barrier is disrupted, which leads to the backflow of intestinal contents into the esophagus. This causes inflammatory changes in the mucous membrane, which may progress to erosions, ulcers, and esophageal strictures.

The main mechanism of reflux esophagitis development is duodenogastroesophageal reflux, in which bile and pancreatic enzymes enter the esophagus.

Key pathophysiological factors include:

- loss of the lower esophageal sphincter as a functional barrier;
- absence of the stomach's reservoir function;
- altered small intestinal motility;
- increased intraluminal intestinal pressure;
- impaired bile drainage.

A particularly important role is played by the aggressive effect of bile acids, which cause chemical damage to the esophageal mucosa.

Reflux esophagitis after gastrectomy develops as a result of profound anatomical and functional changes in the upper gastrointestinal tract, which disrupt the natural antireflux defense mechanisms. Normally, the lower esophageal sphincter, the cardiac portion of the stomach, and its reservoir function prevent the backflow of intestinal contents into the esophagus. After gastrectomy, these mechanisms are completely or partially lost, creating conditions for pathological reflux.

A key element of the pathogenesis is duodenogastroesophageal reflux. After gastrectomy, bile and pancreatic juice freely enter the small intestine, and in cases of impaired motility may be refluxed into the

proximal parts of the digestive tract, including the esophagus. Unlike gastroesophageal reflux, this type of injury is characterized by a more aggressive effect on the mucosa.

Bile acids and pancreatic enzymes exert a pronounced cytotoxic effect. They damage the lipid layer of the esophageal mucosa, disrupt intercellular junctions, and increase epithelial permeability. This leads to chemical inflammation, which may be more severe than acid-induced injury.

An additional factor is the loss of the stomach's reservoir function. Normally, the stomach acts as a buffer regulating the passage of food and fluid into the small intestine. After gastrectomy, there is accelerated transit of intestinal contents, which contributes to increased intraluminal pressure and enhances reflux.

An important role is played by impaired motility of the small intestine and the reconstructed gastrointestinal tract. Slowed or, conversely, discoordinated peristalsis contributes to bile stasis and increases the likelihood of its retrograde movement. Particularly pronounced changes are observed in less physiologically favorable reconstruction methods.

Anatomical factors are also important. In some types of reconstruction, for example esophagojejunostomy without an antireflux mechanism, there is no barrier between the esophagus and the intestine. This makes the esophagus vulnerable not only to chemical but also to mechanical influences.

As a result of prolonged exposure to aggressive duodenal contents, chronic inflammation of the esophageal mucosa develops. It is accompanied by infiltration, edema, damage to the epithelial layer, and impaired regeneration. With progression, erosions, ulcers, and esophageal strictures may form.

Thus, the pathogenesis of reflux esophagitis after gastrectomy is multifactorial and includes a combination of anatomical, motor, and chemical mechanisms of injury. The absence of physiological antireflux structures and exposure to duodenal contents are the main causes of this complication.

Reflux esophagitis after gastrectomy has significant clinical importance, as it is one of the most common and clinically pronounced functional complications after radical gastric surgery. Its development significantly affects the patient's general condition, postoperative course, and long-term quality of life.

The main clinical manifestation of the disease is damage to the esophageal mucosa caused by aggressive duodenal contents. Patients report heartburn, retrosternal burning, a bitter taste in the mouth, bile regurgitation, and epigastric discomfort. These symptoms are often chronic and persistent, and they worsen after eating and in the supine position.

One of the most significant consequences of reflux esophagitis is a persistent reduction in quality of life. Patients experience difficulty with food intake, sleep disturbances, and may develop anxiety and emotional instability due to constant discomfort. As a result, nutritional status deteriorates, which is particularly critical in patients after oncological surgery.

The clinical significance of this condition is also determined by the risk of progression of morphological changes in the esophageal mucosa. With long-standing inflammation, erosions and ulcers may develop, accompanied by pain and bleeding. In more severe cases, fibrotic strictures of the esophagus occur, leading to dysphagia and marked impairment of nutrition.

Special attention is paid to the potential role of chronic reflux in the development of precancerous changes. Long-term exposure to bile acids and pancreatic enzymes may promote epithelial metaplasia, which is considered a risk factor for subsequent neoplastic processes.

From a clinical perspective, reflux esophagitis also complicates postoperative patient management. It requires additional pharmacological therapy, regular endoscopic monitoring, and in some cases repeat surgical interventions if conservative treatment is ineffective.

Reflux esophagitis after gastrectomy is of major clinical importance, as it not only reduces patients' quality of life but may also lead to serious structural changes in the esophagus. Early detection and prevention are essential components of postoperative follow-up.

The most effective direction of prevention is the choice of an optimal gastrointestinal reconstruction method.

1. Roux-en-Y reconstruction.

It is considered the "gold standard" for reflux prevention.

Advantages:

• reduction of duodenogastric reflux;
• decreased contact of bile with the esophagus;
• improved quality of life in patientsю

2. Small bowel interposition.

Used as an additional antireflux barrier.

3. Formation of esophagoenteric reservoirs

Allows partial compensation for the loss of the stomach and reduces intraluminal intestinal pressure.

Pharmacological prevention of reflux esophagitis after gastrectomy is aimed at reducing the aggressive effect of duodenal contents on the esophageal mucosa, decreasing inflammation, and preventing disease progression. It is considered an important complement to surgical and dietary approaches. One of the main drug groups is proton pump inhibitors. Although acid production is significantly reduced after gastrectomy, these drugs are used to decrease residual acid aggression and protect the esophageal mucosa. They also help reduce symptoms of reflux esophagitis and improve patients' overall well-being.

Prokinetic agents play an important role by improving gastrointestinal motility. Their effect is aimed at accelerating the evacuation of intestinal contents and reducing the likelihood of retrograde reflux of duodenal contents into the esophagus. By normalizing motility, the frequency and intensity of reflux episodes are reduced.

Ursodeoxycholic acid is widely used and has a protective effect on the mucosa. It alters bile composition, reduces its cytotoxicity, and decreases the damaging effect of bile acids on the esophageal epithelium. This is especially important in duodenogastroesophageal reflux, which plays a key role in the pathogenesis of the disease.

Additionally, antacid drugs may be used to provide short-term neutralization of aggressive components of the refluxate. Although their effect is mainly symptomatic, they can reduce heartburn and discomfort in patients.

In some cases, agents that improve the protective properties of the esophageal mucosa are used. They help form a protective barrier, increase epithelial resistance to bile and enzymes, and promote tissue repair.

The effectiveness of pharmacological prevention largely depends on a comprehensive approach and the combination of different drug groups. The best results are achieved with a combination of prokinetics and agents that reduce the toxic effects of bile acids.

Lifestyle plays an important role after surgery.

Recommended:

• small, frequent meals;
• avoidance of fatty and fried foods;
• restriction of acidic and irritating foods;
• avoidance of eating before bedtime;
• maintaining an upright position after meals.

These measures reduce intraluminal pressure and decrease the risk of reflux.

Regular endoscopic examination allows:

• early detection of signs of esophagitis;
• assessment of preventive effectiveness;
• prevention of complications.

Patients after gastrectomy require long-term follow-up.

Prevention of reflux esophagitis after gastrectomy is an important component of comprehensive postoperative management. The most effective methods are reconstructive surgical techniques, especially Roux-en-Y anastomosis. Pharmacological and dietary therapy complement surgical prevention and help reduce symptom severity and complication rates. A comprehensive and individualized approach improves the quality of life of patients after radical gastric surgery.

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