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(FGBOU VO SOGMA MRussian Health Ministry)

DEPARTMENT OF INTERNAL DISEASES №2

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Gastric and duodenal ulcer

METHODOLOGICAL MATERIALS
the main professional educational program of higher education - the specialty program in the specialty 31.05.01 General medicin

Vladikavkaz

Methodological materials are intended for teaching 3-4 year students (6-7 semesters) of the Faculty of Medicine of the Federal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation in the discipline "Faculty therapy".

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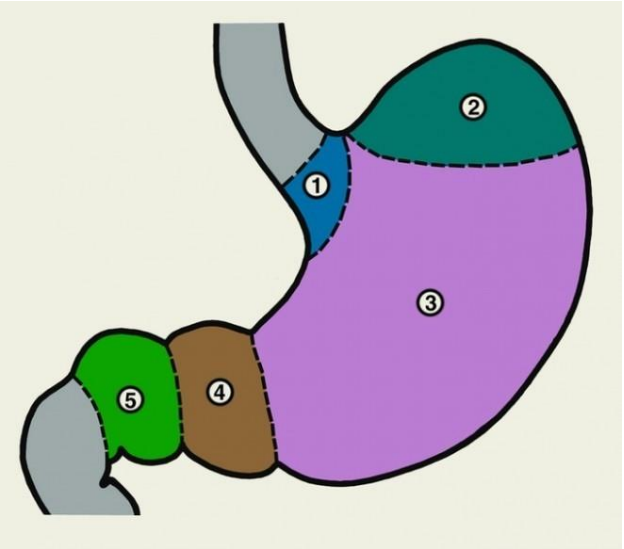
Anatomy and physiology of the stomach

The unique system of the human body is a perfect living mechanism, the well-coordinated work of which depends on the quality of the "fuel", which ensures a clear interaction of all organs. The stomach performs the function of a supplier of nutrition necessary for normal life. Its device describes in detail the anatomy of the human stomach.



Schematic representation of the stomach: 1 - cardiac part; 2 - the bottom of the stomach; 3 - the body of the stomach; 4 - gatekeeper cave; 5 - gatekeeper channel.

Anatomical picture of the stomach

The organ is located in the anterior abdominal region. At the top, it connects to the esophagus, and at the bottom it borders on the duodenum, representing a widened, bag-shaped part of the digestive tube covered with muscles. The stomach is conventionally divided into several main parts with different anatomical and functional features.

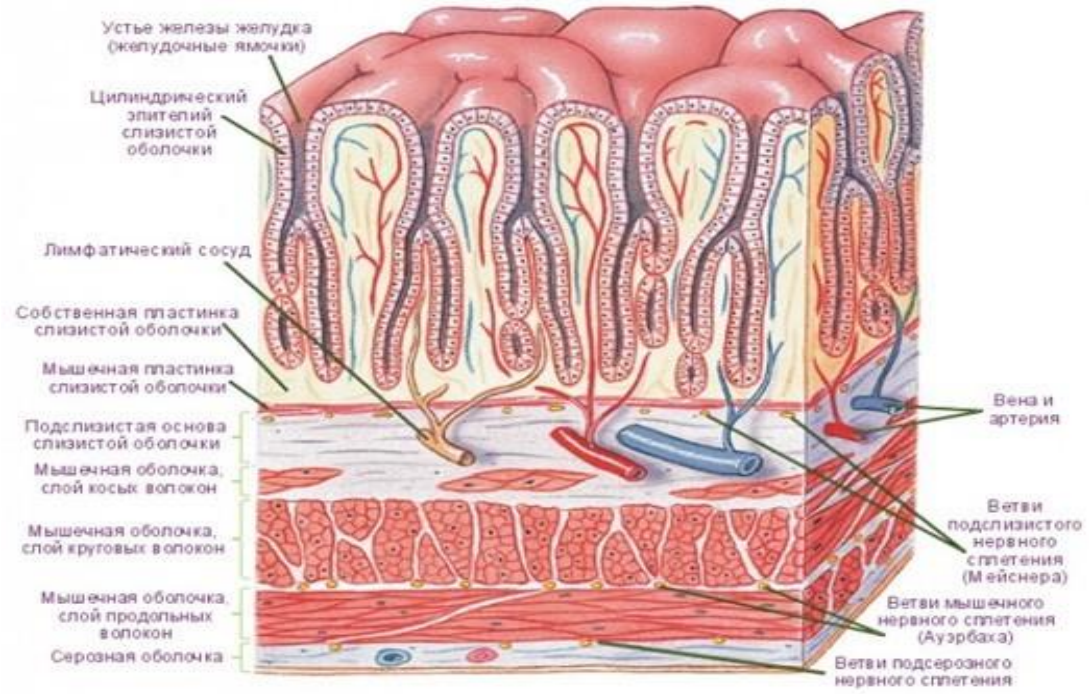
Cardial part.The area located at the junction of the organ with the esophagus. Two adjacent organs interact through a cardinal hole. The muscles in the cardia allow food to move in only one direction, forming an obstruction called the sphincter. Its function is to prevent food from getting back into the esophagus.

Bottom (vault).The part of the organ that occupies the place to the left of the cardia above the horizontal line that passes through the cardinal foramen. It is a dome-shaped bulge filled with air.

Body. The most extensive section, located in the middle of the organ between the fornix and the pyloric part.

Gatekeeper (pyloric) part.The end sector of the stomach, bordering the duodenum. It includes a wide gatekeeper's cave, which merges into a narrow gatekeeper's channel, ending in a circular groove with an opening.

The structure of the walls of the digestive organ



Mucous membrane contains cells of the epithelium, glands and muscle fibers:

- epithelial cells produce mucus containing bicarbonate, which envelops the walls of the stomach, protecting them from the destructive effects of acids and enzymes;
- the glands form pits with walls consisting of cells, some of which produce pepsin and hydrochloric acid, and the other synthesizes substances important for the regulation of digestion;
- muscle fibers participate in the formation of folds of the mucous membrane, thereby increasing its area.

Submucosal layer

Due to the presence of many blood vessels and nerve endings located in the loose connective tissue layer, it contributes to the continuous restoration of epithelial cells of the mucous membrane. And its constituent autonomic nerve fibers are involved in the regulation of digestive processes.

Muscular membrane

Includes three layers of smooth muscle fibers directed in different directions. Their task is to support the motor function of the organ, which allows it to stir and push food.

Serous layer

The outer layer is represented by a thin film covered with epithelium, constantly secreting fluid. Natural lubricant prevents internal friction. In addition, the sheath contains sensitive nerve fibers, when irritated, pain syndrome appears.

The size and shape of the stomach depends on changes in the state of the surrounding organs, the amount of contents and body type. The length of a full stomach reaches 26 centimeters, and an empty stomach does not exceed 20 centimeters.

Systemic anatomy describes the stomach as a distinct system. Its location in relation to other systems of human organs is determined by the topographic anatomy of the stomach.

Definition

Stomach ulcer and / or duodenum - a chronic, cyclical disease, which is based on the expression of the mucous membrane of the stomach and / or duodenum, during an exacerbation.

The mucosal expression is of peptic origin, i.e. local destruction, necrosis of the tissue of the gastric wall due to the aggressive, digestive action of gastric juice with the formation of a peptic ulcer.

A peptic ulcer is defined as a defect in the mucous membrane, covering all layers of the mucosa up to and including the tunica muscularis mucosae and even deeper,

healing by epithelial and fibroelastic proliferation with the development of connective tissue and scar formation.

Peptic ulcer disease is a chronic disease from the moment of its onset, and is characterized by a recurrent (periodically renewed ulcer) course.

Peptic ulcer disease is conventionally divided into two clinical and pathogenetic forms: with the localization of the ulcer in the duodenum and the localization of the ulcer in the stomach. Each of the forms has features in pathogenesis, clinic, prognosis and treatment. A combination of both localization of the ulcer is possible.

Peptic ulcer disease mainly affects men (4-7 times more often than women). The maximum age of patients with peptic ulcer with ulcer localization in the duodenum is 30-40 years, with localization in the stomach - 50-60 years. For 1 case of gastric ulcer, there are 4-5 cases of peptic ulcer disease in the duodenum.

Over the past 15 years, the number of patients with newly diagnosed peptic ulcer disease has a pronounced tendency to increase, with the expansion of the age range of the disease: peptic ulcer disease "got younger" and "aged" at the same time, i.e. the number of "young" and "senile" ulcers increased, and the number of ulcers between the ages of 20-60 also increased.

History of the issue

Peptic ulcer disease is ancient, probably known from prehistoric times.

In many countries, peptic ulcer disease is often called Cruvelier's disease, because historically it is he who is credited with clinically describing gastric ulcer, he gave it an excellent morphological basis.

Thanks to his work, he made it possible to clearly distinguish between benign ulcers and malignant ulcers. Probably, the lack of understanding that there is a benign ulcer, and there is an ulcer against the background of a cancerous lesion of the stomach, characterizes the first stages of the study of peptic ulcer disease.

French physician, anatomist Jean Cruvelier

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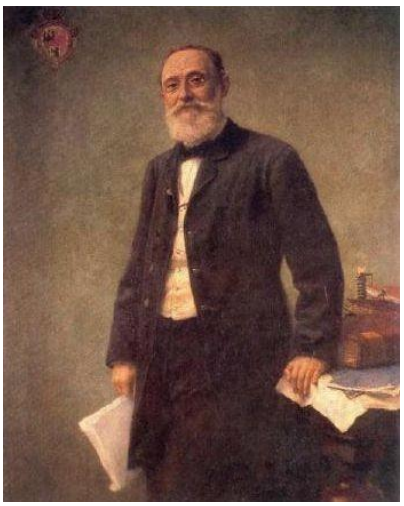
Prout clearly showed that it is hydrochloric acid that is contained in gastric juice. Without acid, there is no ulcer.

The victory over peptic ulcer disease was forged by the hands of surgeons. At some point in history, these were the first successful gastrectomies, gastroenterostomy and meticulous work on modifying vagotomy. Of course, this is the first Latarje operation (1920s), 1940s - Dragsted operation, 1960s - 1970s - highly selective vagotomy.

If we characterize the 2nd part of the XX century, our ideas about the pathogenesis of peptic ulcer were, in general, on the one hand, quite complete, but they were based on numerous theories of the pathogenesis of peptic ulcer, which arose at different times and were confirmed by numerous data.

Rudolf Virchowand his famous vascular circulatory theory of the onset of peptic ulcer disease. In fact, malnutrition of the stomach wall due to impaired circulation has been postulated as the basis for ulcer formation.



Rudolf Virchow

The remarkable study of Claude Bernard "The Peptic Theory of Peptic Ulcer Disease", Aschoff, who, through the analysis of observations, where ulcers are mainly located, positioned the mechanical theory of peptic ulcer disease.

Now it is easy for us to say that there is pyloric Helicobacter, its importance in the pathogenesis of peptic ulcer disease is very great. Of course, there were infectious, historically proposed theories of the origin of peptic ulcer disease. But that was a slightly different infectious theory. These were theories that proceeded from the fact that a stomach ulcer occurs, for example, in patients with purulent appendicitis, on observations of ulcers in patients with suppurative processes. There were years when streptococcus was positioned as the infectious agent that led to the formation of peptic ulcer disease.

Probably the theory that has now been confirmed by time is the gastric theory, the theory of Konjetzny, who brilliantly proved that in all cases of peptic ulcer, inflammation of the gastric mucosa is found. Moreover, this is a chronic active gastritis mainly in the antrum. Naturally, there were critics, and very serious critics, of any theory of the origin of peptic ulcer disease. Including the gastric theory of the origin of peptic ulcer disease.

The years passed, and, of course, the development of pharmacological control over the acidity of gastric juice made an outstanding contribution to our victory over peptic ulcer disease. Understanding of both the nervous and humoral mechanisms

that regulate the functioning of the stomach first led to the creation of such a class of drugs as H2-histamine blockers. They revolutionized approaches to the treatment of acid-related diseases. And then led to the creation of proton pump inhibitors, which largely describe the present day of the course of peptic ulcer disease.

So, how do we assess the diversity of the pathogenesis of peptic ulcer disease. Having the idea that there are factors that protect the mucous membrane, and factors of aggression of the mucous membrane, they have come to the point that such an important pathogenetic factor as infection of the pyloric H. pylori is put on the scales.

It is, of course, a magical story associated with the names of Australian explorers Barry Marshall and Robin Warren. It began in 1983, when the most authoritative magazine Lancet published tiny notes, each of which fit literally one and a half columns. These were letters to the editor.

“Dear Editor! In our hospital (in fact, in the central district hospital) in the city of Perth, we sowed a new type of microorganism from biopsies of the antrum of the stomach of patients with peptic ulcer disease. We believe that this microorganism

is similar to Campylobacter. We associate this microorganism with chronic active inflammation of the gastric mucosa and peptic ulcer disease. "

As the years passed, Warren and Marshall were honored with the pinnacle of their scientific careers. This is the presentation of the Nobel Prize to them. Moreover, pay attention to the wording of the Nobel Committee: "The Nobel Prize in Physiology and Medicine in 2005 was awarded for their discovery of the bacteria Helicobacter pylori and its role in gastritis and peptic ulcer disease."

The mechanism of ulcer formation

Two important components of digestive juices are hydrochloric acid and the enzyme pepsin. Both substances are critical in the breakdown and digestion of starches, fats and proteins in food. They play different roles in ulcers.

- Hydrochloric acid. It is a common misconception that excess hydrochloric acid secreted in the stomach is solely responsible for the production of ulcers. Patients with duodenal ulcers tend to have higher than normal levels of hydrochloric acid, but most patients with gastric ulcer have normal or below normal acid levels. Stomach acid is actually important in protecting against H. pylori, the bacteria that causes peptic ulcers in most cases. The exception is ulcers that stem from Zollinger-Ellison syndrome, a rare genetic condition in which a tumor in the

pancreas or duodenum secretes very high levels of gastrin, a hormone that stimulates the secretion of hydrochloric acid.

- Pepsin. This enzyme breaks down proteins in food. It is also an important factor in the formation of ulcers. Since the stomach and duodenum are made of proteins, they are sensitive to the action of pepsin.

However, the body has a defense system to protect the stomach and intestines against these two potent substances:

- a layer of mucus that covers the stomach and duodenum (first line of defense);
- bicarbonate, which secretes a layer of mucus that neutralizes digestive acids;
- hormone-like substances prostaglandins, which help to dilate the blood vessels in the stomach to ensure good blood flow and to protect against injury. Prostaglandins can also stimulate the action of bicarbonate and mucus.

The destruction of these defense mechanisms makes the lining of the stomach and intestines susceptible to the action of acid and pepsin, increasing the risk of ulcers.

Causes of stomach and duodenal ulcers

The bacteria appear to cause ulcers in this way: Helicobacter Pylori's corkscrew shape allows them to penetrate the mucous layer of the stomach or duodenum so that they can attach to the lining. The surfaces of the cells lining the stomach contain protein. The protein breakdown accelerating factor acts as a receptor for bacteria.

H. pylori survives in highly acidic environments. H. pylori stimulates the increase and release of gastrin. Higher gastrin levels promote acid secretion. The increase in acid damages the intestinal mucosa, leading to ulcers in certain individuals. H. pylori also alters certain immune factors that allow these bacteria to escape detection by the immune system and lead to frequent inflammation, even without invading the mucous membrane. Even if ulcers do not develop, it is believed that Helicobacter Pylori bacteria are the main cause of active chronic inflammation in the stomach - gastritis, and in the upper part of the small intestine - duodenitis. H. pylori is also closely related to stomach cancer and possibly other extraintestinal

problems. H. pylori bacteria are likely to are transmitted directly from person to person. However, it is not known exactly how these bacteria are transmitted.

About 50% of the world's population is infected with H. pylori. The bacteria are almost always acquired during childhood and persist throughout life if the person is left untreated. The prevalence of this bacterium in children is about 0.5% in industrialized countries. However, even there, in regions with critically unsanitary conditions, the conditions for infection with infections are equal to those in developing countries.

It is not yet clear how these bacteria are transmitted. Possibly transmission methods include:

- intimate contact, including contact with liquid through the mouth;
- diseases of the gastrointestinal tract (especially with vomiting);
- contact with feces (feces);
- contaminated waste water.

Although H. pylori are fairly common, ulcers are very rare in children - only 5-10% of H. pylori-infected adults. Several factors may explain why some infected patients develop ulcers:

- smoking;
- alcohol intake;
- the presence in communication of relatives with peptic ulcer disease;
- male gender;
- infection with a bacterial strain that contains a cytotoxin associated gene.

When Helicobacter pylori was first identified as the main cause of peptic ulcer disease, it was found in 90% of people with duodenal ulcers and about 80% of people with stomach ulcers. As more people are being tested and treated for bacteria, the rate of H. pylori-induced ulcers has decreased. Currently, H. pylori is found in about 50% of people with peptic ulcer disease;

Factors that cause ulcers in H. pylori carriers

Certain factors can increase the risk for an NSAID ulcer:

- age 65 and older;

- history of peptic ulcer or gastrointestinal bleeding;
- other serious diseases such as congestive heart failure;
- the use of drugs such as: the anticoagulant Warfarin (Coumadin), corticosteroids, Alendronate osteoporosis drug (Fosamax), etc.;
- alcohol abuse;
- Helicobacter Pylori infection;
- other risk factors for ulcers from H. pylori or NSAIDs;
- stress and psychological factors;
- bacterial or viral infections;
- bodily injury;
- radiation therapy;
- smoking. Smoking increases acid secretion, reduces prostaglandins and bicarbonate, and decreases blood flow. However, research results on the actual effects of smoking on ulcers vary.

Only 10-15% of people infected with Helicobacter Pylori develop peptic ulcer disease. H. pylori infections, especially in the elderly, may not always lead to peptic ulcer disease. Other factors must also be present in order to actually cause ulcers:

- genetic factors. Some people have strains of H. pylori with genes that make bacteria more dangerous and increase the risk of ulcers;
- immune disorders. Some people have a compromised intestinal immune response that allows bacteria to damage the intestinal lining;
- lifestyle factors. Although lifestyle factors such as chronic stress, coffee and smoking have long been considered the main causes of ulcers, they are now believed to only increase the susceptibility to ulcers in some H. pylori carriers - and nothing more;
- stress. Although stress is no longer believed to cause ulcers, some research suggests that stress can predispose a person to ulcers or prevent existing ulcers from healing;

- shift work and interrupted sleep. People who work night shifts have a significantly higher incidence of ulcers than day workers. Researchers suspect that frequent sleep breaks may weaken the immune system's ability to defend against harmful bacteria.

- non-steroidal anti-inflammatory drugs (NSAIDs). Long-term use of NSAIDs such as aspirin, Ibuprofen (Advil, Motrin) and Naproxen (Aleve, Naprosin) is the second most common cause of ulcers. NSAIDs also increase the risk of gastrointestinal bleeding. The risk of bleeding continues as long as the patient is taking these drugs, and it can continue for about 1 year after the break. Short courses of NSAIDs to temporarily relieve pain shouldn't cause serious problems because the stomach has time to heal and repair any damage that has occurred.

Patients with ulcers from NSAIDs should stop taking these drugs immediately. However, patients who require these drugs on a long-term basis can reduce the risk of ulcers by taking PPI drugs - a proton pump inhibitor - such as Omeprazole (Prilosec), Famotidine (Pepsid, an H2 blocker), and others.

15-25% of patients taking NSAIDs regularly will have evidence of one or more ulcers, but in most cases these ulcers are very small. Long-term use of NSAIDs can damage the small intestine as well. Even low doses of aspirin (81 mg) may pose some risk, although the risk is lower than with higher doses. The risk is greatest in people using very high doses of NSAIDs for a long period of time, especially in patients with rheumatoid arthritis.

- Medications. Certain drugs other than NSAIDs can also worsen ulcers. These include: Warfarin (Coumadin), an anticoagulant that increases the risk of bleeding, oral corticosteroids, and some chemotherapy drugs Spironolactone and Niacin. Bevacizumab, a drug used to treat colorectal cancer, may increase the risk of gastrointestinal perforation (perforation or perforation of an ulcer is a breakthrough of an ulcer outside the stomach or duodenum and release its contents). Although the benefits of bevacizumab outweigh the risks, gastrointestinal perforations are very serious. If they occur, patients should stop taking the drug.

- Zollinger-Ellison syndrome(ZES) .. Another cause of peptic ulcer disease, although much smaller than H. pylori or NSAIDs, is Zollinger-Ellison syndrome. Large amounts of acid are produced in response to the overproduction of the hormones gastrin, which in turn causes pancreatic or duodenal tumors. These tumors are usually cancerous and need to be removed. Acid production must also be suppressed to prevent new ulcers.

ZES should be suspected in ulcer patients who are not infected with H. pylori and who have no history of NSAIDs. Diarrhea can occur before the symptoms of the ulcer. Ulcers that occur in the second, third, or fourth parts of the duodenum or in the jejunum (the middle part of the small intestine) are signs of ZES. Gastroesophageal reflux disease (GERD) is more common and often more severe in patients with ZES. Complications of GERD include ulcers and narrowing (strictures) of the esophagus.

Ulcers associated with ZES are usually persistent and difficult to treat. Treatment consists of removing the tumor and suppressing the acid with special drugs. In the past, gastric removal was the only treatment option.

Experts don't know what factors actually increase the risk of developing ulcers.

Causing factors:

- Psycho-emotional stress
- Negative long-term emotions
- Medicinal effects
- Bad habits (alcohol, smoking)
- Helicobacter pillory infections
- Violation of duodenal patency
- Local exposure to mechanical, physical and thermal irritants.

Contributing:

- Violation of the nature and rhythm of food.
- Predisposing:
- Hereditary predisposition

- Increase in the mass of parietal cells
- Increased release of gastritis in response to food intake
- Trypsin inhibitor deficiency
- Gastroduodenal motility disorder
- Blood group 0 (I)
- Rh positive (Rh +)
- Nonsecretory status (inability to secrete AVN antigens from saliva)
- Fucomucoprotein deficiency
- HLA genotype: B5, B15, B35
- Impaired production of immunoglobulin A.

Pathogenesis

In the pathogenesis of peptic ulcer disease, the primary importance belongs to the disorder of the mechanism of neurohumoral regulation of gastric secretion.

At the first stagethere is a disintegration of the processes of excitation and inhibition in the cerebral cortex. At the second stage, dysfunction of the hypothalamic-pituitary system is added. At the third stage, there is a violation of the function of the nervous autonomic system.

The leading one is the predominance of the tone of the nervous parasympathetic system (vagotonia), which causes the following effects:

a) increased tone of smooth muscles of the stomach and increased peristalsis

b) an increase in the secretion of hydrochloric acid and gastrin

c) suppression of the activity of antroduodenal inhibition of gastric secretion with the throwing of acidic gastric contents into the duodenum, where it does not have time to alkalize.

d) the development of inflammatory and dystrophic processes in the duodenum.

e) a decrease in the secretion of enterogastrin, secretin, pancreozymin (a decrease in the secretion of bicarbonates of pancreatic juice), a violation of the inhibition of hydrochloric acid production.

The noted violations are aggravated by the doubled number of parietal cells and a decrease in the protective properties of the mucous membrane (chronic type B gastritis).

At the fourth stage dysfunction of the endocrine system, especially the local, gastrointestinal endocrine system of the gastrointestinal tract joins, leading to the disintegration of the functions of various parts of the digestive system.

The production of hormones and biologically active substances that stimulate the production of acid-peptic factor (cortisol, thyroxine and triiodothyronine, insulin, parathyroid hormone, gastrin, neuropeptide, gastrointestinal polypeptide) increases. The activity of local hormones inhibiting gastric secretion (somatostatin, glucagon, sex hormones, calcitonin, endorphins, enkephalins) decreases.

As a result, with the predominance of factors of aggression, an ulcer of the duodenum or stomach develops. In the latter case, sympathicotonia is of greater importance in pathogenesis. Against the background of an increased tone of the nervous sympathetic system, the following occurs: a decrease in the tone of the smooth muscles of the stomach, a slowdown in evacuation, antral stasis develops, which reflexively, due to stretching of the walls of the pyloric section, causes an increase in the production of gastrin, and, consequently, hydrochloric acid, the closure function of the pylorus is disrupted, leading to reflux of duodenal contents, which has a damaging effect on the gastric mucosa with the formation of a peptic ulcer.

Aggressive factors:

- hydrochloric acid
- Helicobacter pylory
- pepsin
- bile acids
- increase in the number of parietal cells
- accumulation of histamine in the stomach wall
- dysregulation of gastric secretion

- glucocorticoids of the adrenal cortex

Protective factors:

- mucus secretion
- prostaglandins
- renewal of the epithelium (good regeneration)
- blood supply to the gastric mucosa
- normal mechanism of inhibition of gastric secretion
- mineralocorticoids of the adrenal cortex

Pathogenesis of stomach ulcers

- entering the stomach, Helicobacter pylory multiplies and colonizes on the mucous membrane;
- the permeability of the epithelial barrier increases due to alteration of the epithelium by factors of microbial aggression (ammonia, cytotoxins, proteases, products of destroyed leukocytes;
- Helicobacter pylory disrupts the composition of the structure of the gel, directly damaging the epithelium of the mucous membrane;
- epithelial cells lose contact with the basement membrane and slough off, forming microdefects on the surface of the gastric mucosa, and the aggressive environment forms the formation of an erosive and (or) ulcerative defect;
- urease produced by Helicobacter pylory is a strong chemotaxis factor; monocytes and leukocytes attracted by it secrete cytokines, produce free radicals, damaging the epithelium;
- in the mucous membrane affected by Helicobacter pylory, the formation of leukotriene increases, which causes a sharp vasospasm, as a result of which trophism is disturbed;
- through the damaged areas of the mucous membrane, the reverse flow of hydrogen ions increases and ulceration is formed, an ulcer is formed.

Pathogenesis of duodenal ulcer

1. an increase in the tone of the vagus nerve, both during food intake and outside it, and the inhibition of gastric secretion is not active enough;
2. a constant increase in the tone of the vagus nerve causes gastric secretion with high peptic activity;
3. under the influence of acidification, the duodenal mucosa undergoes metaplasia;
4. from the antrum of the stomach, Helicobacter pylory move to the duodenum, colonize the metaplastic epithelium, and then damage it as in the stomach.

Rare causes of gastroduodenal ulceration:

1. ulcers caused by drugs (acetylsalicylic acid and other NSAIDs);
2. ulcers resulting from pronounced gastric hypersecretion of HCl (Zollinger-Ellison syndrome in the gastronome, hyperparathyroidism, systemic mastocytosis);
3. a stomach ulcer, which is ulcerated cancer or lymphoma.
4. PUD should also be distinguished from other symptomatic gastroduodenal ulcerations, acute and chronic, arising again against the background of certain diseases and external influences. Acute stomach ulcers, usually superficial, are clinically manifested by bleeding and a low recurrence rate after healing, and can occur in patients with extensive burns, CNS damage, and stress.

Peptic ulcer classification

By etiology

- associated with Helicobacter pylory
- not associated with Helicobacter pylory

By localization

Stomach ulcers:

- cardiac and subcardial departments
- bodies
- antrum
- pyloric canal

Duodenal ulcers

- bulbs

- posterior bulbous section

A combination of a stomach ulcer and a duodenal ulcer (gastrojejunal ulcer).

By type of ulcers

- single
- multiple

By size (diameter of ulcers)

- small, diameter up to 0.5 cm
- medium, diameter 0.5-1 cm
- large, diameter 1.1-2.9 cm
- giant, diameter 3 cm or more for stomach ulcers, more than 2 cm for duodenal ulcers

According to the clinical course:

typical

atypical:

1) with atypical pain syndrome,

2) painless (but with other clinical manifestations),

3) asymptomatic.

By the nature of the flow

- Peptic ulcer disease detected for the first time (acute).
- Recurrent course (chronic):
- with rare exacerbations (once every 2-3 years or less)
- with annual exacerbations
- with frequent exacerbations (2 times a year or more)

By stage of the disease

- exacerbation
- remission
- incomplete remission (fading exacerbation)

Characteristics of the function of the gastroduodenal system:

- increased secretion
- normal secretion
- decreased secretion

Clinical manifestations

Pain syndrome:

1. Epigastric pain is the most important and persistent symptom of peptic ulcer disease. Painful sensations appear in the pathologically altered mucosa. The characteristic of the pain syndrome depends on the localization of the ulcer. The pain is almost always associated with food intake, the later after a meal the pain, the more distal the localization of the ulcer: earlier pain sensations (immediately after a meal) - with a stomach ulcer, late (after 2-3 hours) and hungry (in the morning, on an empty stomach) - with duodenal ulcer. For duodenal ulcer, a seasonal rhythm of pain syndrome is characteristic.
2. Pain is relieved (relieved) by taking anticholinergics. The cause of pain should be considered movement disorders of the stomach and duodenum. Assessment of the sensation of pain, its severity depends on the personality characteristics and mental attitude of the patient. The anamnestically revealed pain syndrome is confirmed by palpation data, in which there is local pain in the epigastrium (positive Mendel's symptom).
3. pain can radiate to the right hypochondrium, behind the sternum, in the back;
4. possible painless form of peptic ulcer disease.

Dyspeptic syndrome:

- heartburn, sometimes excruciating. Heartburn - associated with the throwing of acidic contents from the stomach into the esophagus. It can also be, as a result of a spasm of the pathologically altered lower segment of the esophagus, is the equivalent of pain,
- belching sour, air;

- anorexia;
- dysphagia;
- vomiting is a complex reflex act, it is usually the contents of the stomach, less often with an admixture of blood (fresh or of the type of `` coffee grounds "), occurs mainly at the height of pain, after which there usually comes relief of pain and dyspeptic symptom complexes

Intestinal dyspepsia syndrome: a tendency to constipation.

Physical status: local palpation and percussion pain in the epigastric region, but clinical symptoms are sometimes absent.

Asthenoneurotic symptom complex

Patients suffering from peptic ulcer disease may experience depression of mood with increased excitability and anxiety. Sometimes the ability to concentrate on an activity is lost. As a rule, poor sleep is noted.

In patients with duodenal ulcer, signs of dystonia of the autonomic nervous system are found, increased salivation - in the morning a wet pillow at the corner of the mouth, in the afternoon there are frequent swallowing movements, increased sweating, sharp dampness of the palms, pronounced red, less often white, dermographism (`` vascular game "). Often, with an objective study, tachycardia, pulse lability, and low blood pressure are noted.

Clinical forms of peptic ulcer disease and features of their course

The clinical forms of peptic ulcer disease and the peculiarities of their course are characterized by a significant variety and depend on many factors: on the localization of the ulcer, age, gender of the patient, the nature of complications, and others.

<u>For duodenal ulcers and pyloric ulcers, it is characteristic:</u>

- late, "hungry" and night pains,
- increased acidity of gastric contents,
- hypersecretion.

<u>With ulcers of the pyloric canal:</u>

- there are intense pains caused by the involvement of the neuromuscular apparatus of the gatekeeper in the process with its prolonged spasm and increased intragastric pressure;
- nausea and vomiting, which in some patients may be the only symptom of the disease;
- weight loss.

postbulbar ulcers (extra-bulbous):
- more often located in the area of the upper bend or in the initial segment of the descending part of the duodenum,
- they prevail in men;
- a typical clinic of a duodenal ulcer, but there may be some peculiarity in the nature of the pain: it occurs more often at the end of the day, the frequency, connection with food intake is not always preserved, in some patients the pain becomes paroxysmal; pain can be very stubborn and not relieved by conventional drugs and heat;
- localized pain in the right upper quadrant of the abdomen, sometimes in the back and around the navel;
- a feature of ulcers - a tendency to frequent bleeding, to the penetration of the ulcer into the pancreas with the development of reactive pancreatitis;
- there may be jaundice caused by inflammation that has spread to the sphincter of the large duodenal papilla, compression of the common bile duct and impaired outflow of bile from the biliary system;
- adhesions to the gallbladder and other organs.

There are "mute" ulcers, in which there is no main symptom of an ulcer - pain, the disease can manifest itself as sudden bleeding, perforation.

In adolescence, the course of peptic ulcer disease has a number of features:

1. localization is more often duodenal;
2. accompanied by a sharp pain syndrome, torpid current;
3. severe dyspeptic disorders;
4. high levels of gastric secretion;

5. repeated profuse bleeding occurs more often;
6. there may be an instability of gastric secretion - heterochilia, that is, a change from high acidity indicators to lower ones and vice versa;
7. violation of the motor function of the stomach is manifested in increased peristalsis, pyloric spasm.

Complications of peptic ulcer

- bleeding
- perforation
- penetration
- periviscerite
- obturation of the outlet of the stomach as a result of edema and cicatricial-ulcerative deformation of the walls of the affected organ
- pyloric stenosis
- cancerous degeneration.

Typical signs of profuse bleeding are bloody vomit of the color of coffee grounds (hematemeses) and tarry stools (melena):

1. bloody vomiting is more often observed with localization of an ulcer in the stomach, but it can also be with a duodenal ulcer
2. the brown color of the vomit depends on the chlorhemin impurity.
3. tarry stools are usually observed with a duodenal ulcer, but it can also be with an ulcer of any other localization. The black color of feces depends on the admixture of iron sulfide and indicates a high localization of bleeding.
4. the first symptoms of profuse internal blood loss are sudden onset of weakness, thirst, dizziness, nausea, feeling of lack of air.
5. when bleeding occurs, the pain syndrome disappears or decreases.

Ulcer perforation- one of the most severe complications, it occurs in 5-15% of cases. Distinguish perforation into the free abdominal cavity, accompanied by the development of acute peritonitis, covered perforation and perforation into the retroperitoneal tissue and into the thickness of the lesser omentum.

Periviscerites- the most frequent complications of peptic ulcer disease. Inflammatory adhesions with adjacent organs are a consequence of ulcer scarring and reactive inflammation. In some patients, the presence of periduodenitis or perigastritis does not manifest itself clinically.

Gatekeeper stenosis- pyloric stenosis (stenosis pylori) as a complication of peptic ulcer disease usually occurs with long-lasting ulcers, with it comes cicatricial narrowing of the pyloric canal and bulb. Pyloroduodenal stenoses develop gradually and in the first stages are compensated by the increased work of the stomach muscles.

The transition of the ulcer to cancer. The diagnosis of a stomach ulcer that degenerates into cancer is often difficult. The following changes in the clinical picture of the disease are suspicious of the malignancy of the ulcer: pains lose their frequency and become permanent, the secretory function of the stomach may decrease, occult blood in the feces is constantly determined, hypochromic anemia, neutrophilic leukocytosis and an increase in the number of platelets appear, a decrease in body weight and deterioration general condition, treatment is ineffective. The most reliable diagnostic method in proving degeneration is fibrogastroscopy with targeted biopsy.

Diagnosis of gastric and duodenal ulcers

An ulcer is always suspected in patients with persistent dyspepsia. Symptoms of dyspepsia occur in 20-25% of people who live in industrialized countries, but only about 15-25% of dyspeptic patients actually have ulcers. There are several steps to be taken to accurately diagnose ulcers:

- Medical and family history... The doctor will ask questions about dyspepsia for a detailed response from the patient, and will also check:

- other important symptoms such as weight loss or fatigue;
- current and past drug use (especially long-term use of NSAIDs);
- family members with ulcers;
- drinking and smoking habits;

- Exclusion of other diseases and disorders. Dyspepsia is caused by many other diseases. Symptoms of stomach ulcers - particularly stomach and chest pains - can be similar to those of other illnesses, including:

- Gastroesophageal reflux disease. About half of GERD patients also have dyspepsia. For GERD or other esophageal problems, the main symptoms are: heartburn, burning pain up to the throat. It usually develops after eating and gets better from antacids. The patient may have difficulty swallowing, belching, or heartburn. Older patients with GERD are less likely to have these symptoms, but instead may be: loss of appetite, weight loss, anemia, vomiting, or dysphagia (difficulty or painful swallowing).
- Heart problems. Heart pain, such as from angina or heart attack, most likely comes from exercise and can be transmitted to the neck, jaw, etc. In addition, patients usually have risk factors for cardiovascular disease;
- Gallstones. The main symptom is a persistent attack or an eating pain on the right side under the chest. This pain can be severe and may radiate to the upper back. Some patients experience chest pain. Pain often occurs after fatty or heavy food, but gallstones almost never cause dyspepsia;
- Irritable Bowel Syndrome - May cause upset stomach, abdominal pain, nausea, vomiting, and bloating. It is more common in women than in men;
- Side effects of medications. Dyspepsia can also occur from gastritis, stomach cancer, or as a side effect of certain medications, including NSAIDs, antibiotics, iron, corticosteroids (Theophylline), and calcium blockers;

Research

1. EGDS (esophagogastroduodenoscopy) with targeted biopsy - is performed in all cases to establish a diagnosis. It determines the shape, size, depth of the ulcer defect, reveals motor disorders. In case of duodenal ulcers - once for diagnosis, in case of stomach ulcers - it is repeated to control the healing of the ulcer.

2. Biopsy examination:

- rapid urease test - express diagnostics of Helicobacter pylori;

- morphological examination - for the diagnosis of Helicobacter pylor, the state of the mucous membrane, malignant changes, the exclusion of rare causes of peptic ulcers,

- bacteriological method. Allows you to determine the sensitivity to antibiotics Helicobacter pylori.

3. Test for the presence of Helicobacter pylori is strictly required for every patient with peptic ulcer. One of the research methods is enough: respiratory "C" - urease test, stool - test (determination of Helicobacter pylori antigen in feces).

The results can be false negative under the influence of medication (antibiotics, bismuth preparations, proton pump inhibitors). Tests can only be performed 4 weeks after stopping these drugs.

4. Complete blood count (detection of anemia, inflammatory processes).

5. Analysis of feces for occult blood - diagnosis of acute and chronic blood loss.

6. X-ray examination of the stomach - with suspicion of complications, first of all - with stenosis of the outlet of the stomach.

7. Ultrasound of the abdominal organs - to identify concomitant pathology, complications.

8. Study of the secretory function of the stomach (intragastric pH metry) - is important for choosing the optimal treatment regimen.

9. Coagulogram - a decrease in the factors of the blood coagulation system.

Consultation of specialists.

Mandatory:

- therapist;

- gastroenterologist.

According to indications:

- surgeon - with complicated peptic ulcers (perforation, bleeding, stenosis);

- an oncologist - in case of suspicion of malignancy (malignant degeneration) of the stomach.

Treatment

General principles: patients with a newly diagnosed ulcer, as well as with an exacerbation of a peptic ulcer, regardless of age, location and nature of the ulcerative process, are subject to hospitalization in the therapeutic department; if for some reason hospitalization is impossible and treatment is carried out at home, it should be close to inpatient. Do not smoke or consume strong alcoholic beverages. In complex therapy, therapeutic nutrition is of great importance. The diet of a patient with peptic ulcer should include:

- minimal stimulating effect of food on the main glands of the stomach, a decrease in motor activity,
- preference for products with good buffering properties: animal proteins: boiled meat and fish, milk, cream, soft-boiled eggs or steamed omelets, cottage cheese, butter. Stale white bread, dry biscuits and biscuits, dairy and vegetarian soups are also allowed. The diet includes vegetables (stewed or mashed), various cereals, jelly and fruit juices, baked apples;
- during an exacerbation, the patient must eat at least 6 times a day, between meals it is recommended to drink half a glass of warm milk or a mixture of milk and cream in order to bind the hydrochloric acid released after the evacuation of food from the stomach;
- with a pronounced exacerbation of peptic ulcer with the presence of pain and dyspeptic syndromes, table number 1a is assigned (food is given only liquid or in the form of jelly, mashed potatoes, the amount of salt is limited to 4-5 g. Bread and crackers are excluded. Meat and fish are given in the form of a souffle one once a day in small quantities);
- table number 1a low-calorie (2100-2200 kcal) and physiologically defective. Long-term appointment of table No. 1a may be accompanied by a feeling of hunger, loss of body weight, therefore, it is appointed only if necessary and for a short time (several days);
- in the phase of remission, table No. 5, somewhat enriched with protein products.

Principles of pharmacotherapy for peptic ulcer disease:

1. the same approach to the treatment of gastric and duodenal ulcers;
2. compulsory basic antisecretory therapy;
3. selection of an antisecretory drug that maintains an intragastric pH> 3 for about 18 hours a day;
4. the appointment of an antisecretory drug in a strictly defined dose;
5. endoscopic control at 2-week intervals;
6. the duration of antisecretory therapy, depending on the timing of the healing of the ulcer;
7. eradication anti-Helicobacter pylori therapy in HP-positive patients;
8. mandatory monitoring of the effectiveness of anti-Helicobacter therapy after 4-6 weeks;
9. repeated courses of anti-helicobacter therapy if it is ineffective;
10. maintenance anti-relapse therapy with an antisecretory drug in HP-negative patients;
11. influence on risk factors of poor response to therapy (replacement of NSAIDs with paracetamol, selective inhibitors of COX-2, combination of NSAIDs with misoprostol, ensuring patient compliance, etc.).

Drug therapy for gastric ulcer and duodenal ulcer

Given the pathogenesis of the development of peptic ulcer disease, eradication therapy is in the first place. Each patient with peptic ulcer disease, in whom HP is found in the gastric mucosa, by one method or another (rapid urease test, morphological method, using HP DNA determination by polymerase chain reaction, etc.), is given eradication therapy.

Dosage regimens for the eradication of HP include a proton pump inhibitor or H2-receptor blocker (ranitidine, famotidine), as well as bismuth preparations in combination with two or three antibacterial drugs.

According to the 2000 Maastricht (European) Consensus, first-line eradication therapy includes:

proton pump inhibitor or ranitidine (histamine H2 receptor blocker), bismuth tripotassium dicitrate in a standard dose 2 times a day + clarithromycin 500 mg 2

times a day + amoxicillin 1000 mg 2 times a day or metronidazole 500 mg 2 times a day, at least 7 days.

Psychotropic: Sedatives and tranquilizers - diazepam, seduxen, elenium, bellataminal, valerian root decoction, motherwort herb infusion.

Antacids: Almagel, Almagel A 15-30 ml X 3-4 times a day: preparations containing bismuth - vikalin (vikair), a mixture of Bourget (sodium bicarboate, sodium phosphate, sodium sulfate), dissolve in a glass of water, 1 tbsp. x 3-4 times a day.

Agents affecting various levels of nervous regulation:

1. M-anticholinergics - atropine 0.1% - 1.0. Platyphyllin 0.2% - 1 ml, etc. 1-2 times a day, for 10 days up to 4-6 weeks, with breaks for 2-3 days, every 10 days.
2. ganglion blockers: benzohexonium 0.1% - 1.0 2 times, quameron 0.03 x 3 times; pyrilene 0.005x 3 times with persistent pain syndrome, in combination with hypertension.
3. central anticholinergics (difacil, methyldiazil, aprenal 2% - 1.0x 2 times a day).
4. blockers of central dopamine receptors: metoclopramide (cerucal, raglan, prileperan) and sulpiride (eglonil, dogmatil) - drugs selectively affect the motility of the gastroduodenal system.

Drugs affecting the acid-peptic factor intragastrically:

1. H2 blockers - histamine receptors - cimetidine (cinamet, tagamet, belamet) 400 mg x 2 times a day with the transition to maintenance therapy 400 mg at night for at least 1 month, ranitidine 150 mg - 2 times a day, famotidine 20 mg - 2 once a day.
2. selective blockers of H-cholinergic receptors - gastrocepin (pirenzepine) 50 mg - 300 mg per day for 4-6 weeks: maintenance therapy of 50 mg at night for a long time.
3. inhibitors of sodium-potassium ATPase (proton pump) - omeprazole 30 mg in the morning or in the evening.

Drugs that improve reparative processes: anabolic steroids, methyluracil, potassium orotate, pentoxil, oxyferriscarbon, allonton, solcaseryl at a dose of 8 ml / day, within 2 weeks, with the transition to perroralbic pium of the drug (100 mg x 3 times a day for 1-2 weeks).

Agents affecting the formation of mucus (liquiditon, flacarbon, biogastron).

Means that have a protective effect on mucous membranes (cytoprotectors): sucralfate (Venter) 0.5-1.0 3 times a day for 30 minutes. before meals and 4 times before bedtime.

In cases where it is assumed or revealed the involvement of HP in the onset and recurrence of peptic ulcer disease, the following is used:

de-nol (1t.x 3 times 30 minutes before meals for 2-4 weeks);

metronidazole (1t.x4 times a day);

omeprazole

oxacillin (2 g per day) and others (clarithromycin).

Second line therapy (for repeated treatment) includes a proton pump inhibitor in a standard dose 2 times a day + bismuth tripotassium dicitrate 120 mg 4 times a day + metronidazole 500 mg 3 times a day + tetracycline 500 mg 4 times a day for 7 days.

One of the seven-day regimens is used to ensure the eradication of HP in more than 80% of cases:

one... Rabeprazole or omeprazole 20 mg 2 times a day or lansoprazole 30 mg 2 times a day. Drugs should be taken at intervals of 12 hours.

+ clarithromycin 500 mg 2 times a day at the end of meals

+ metronidazole or tinidazole 500 mg 2 times a day at the end of meals.

2. Proton pump inhibitor 2 times a day (standard dose)

+ azithromycin 1000 mg once a day for 3 days

+ amoxicillicin 1 g 2 times a day at the end of meals

3... Ranitidine bismuth citrate 400 mg 2 times a day with food

+ clarithromycin 250-500 mg or tetracycline 1000 mg or amoxicillin 1000 mg 2 times a day

+ metronidazole 500 mg 2 times a day with food.

Quad therapy:

Proton pump inhibitor 2 times a day (standard dose)

+ bismuth tripotassium dicitrate 120 mg 3 times 30 minutes before meals and the 4th time 2 hours after meals before bedtime

+ metronidazole 250 mg 4 times a day at the end of a meal or tinidazole 500 mg 2 times a day at the end of a meal

+ tetracycline or amoxicillin 500 mg 4 times a day at the end of meals.

In all regimens, metronidazole can be replaced with furazolidone (0.1 g 4 times or 0.2 g 2 times a day). The use of pilobact, which contains omeprazole, clarithromycin, tinidazole, is justified. The combination contains a full set of drugs for 7-day eradication therapy (1st week), and if the bismuth preparation is included in the scheme for 7-10 days, then in terms of effectiveness it will correspond to the backup option (2nd line).

After the end of the combined eradication therapy, it is necessary to continue treatment for another 5 weeks with duodenal and 7 weeks with gastric localization of ulcers with a single appointment (at 14-16 hours) of the proton pump inhibitor that was used in eradication regimens, or one of the following drugs: ranitidine 300 mg 1 time in 19-20 hours; famotidine 40 mg once every 19-20 hours; or bismuth tripotassium dicitrate 240 mg 2 times a day.

With uncomplicated ulcer, prolonged treatment after the end of the eradication course may not be carried out. If only antisecretory drugs are used in the therapy of these patients, especially proton pump blockers, then HP translocation from the antrum into the body of the stomach with the development of pangastritis is noted, which significantly reduces the effectiveness of subsequent eradication therapy and is a risk factor for the development of stomach cancer.

Drug treatment of gastroduodenal ulcers not associated with HP

The goal of treatment is to relieve symptoms of the disease and provide scarring of the ulcer. Drug combinations and regimens include an antisecretory drug in

combination with a cytoprotective agent, mainly sucralfate. One of the following schemes is used:

1. Ranitidine 300 mg per day, mainly once in the evening (19-20 hours) and an antacid drug (almagel, phosphalugel) inside 1-2 doses in the interdigestive period 3-4 times a day and in the evening before bedtime as a symptomatic remedy for quick relief pain and dyspeptic disorders.

2. Famotidine 40 mg per day, mainly once in the evening (at 19-20 h) and an antacid drug as a symptomatic agent.

3. Omeprazole 20 mg at 14-15 hours and an antacid drug as a symptomatic agent. Instead of omeprazole, rabeprazole 20 mg / day, or lansoprazole 30 mg / day, or pantoprazole 40 mg / day can be used.

Sucralfate (0.5-1 g 3 times a day 30 minutes before meals and in the evening before bedtime on an empty stomach) is prescribed simultaneously with antisecretory drugs for 4 weeks, then in a half dose for a month.

The factors that determine the often recurrent course of peptic ulcer disease are:

- contamination of the gastric mucosa by HP;
- taking NSAIDs;
- a history of ulcer bleeding and ulcer perforation;
- low compliance, i.e. lack of readiness of the patient to cooperate with the doctor, manifested in the refusal of patients
- stop smoking and drinking alcohol, irregular medication.

The effectiveness of treatment for gastric ulcers and gastrojejunal ulcers is controlled endoscopically after 8 weeks, and for duodenal ulcers - after 4 weeks.

Indications for surgical treatment of peptic ulcer:strictures in the esophagus, stomach and duodenum. Penetrating and perforating processes in the absence of regression of the pathological process under the influence of drug therapy. Continuous or recurrent bleeding, despite the use of endoscopic and medical methods of hemostasis.

For the prevention of exacerbations of ulcer and especially duodenal ulcer, and, consequently, their complications, two types of therapy are recommended:

1. Continuous(for months and even years) maintenance therapy with an antisecretory drug in half the dose, for example, 150 mg of ranitidine daily in the evening or 20 mg of famotidine. The indications for this therapy are: ineffectiveness of the performed eradication therapy; complications of ulcer (ulcerative bleeding or perforation); the presence of concomitant diseases requiring the use of non-steroidal anti-inflammatory drugs; concomitant ulcer erosive and ulcerative reflux esophagitis; the patient's age is over 60 years old with annually recurrent course of ulcer, despite adequate course therapy.

2. Therapy "on demand",providing for the appearance of symptoms characteristic of an exacerbation of ulcer, taking one of the antisecretory drugs: rabeprazole, ranitidine, famotidine, omeprazole, lansoprazole in a full daily dose for 3 days, and then in a half dose for 3 weeks. If, after such therapy, the symptoms of exacerbation completely disappear, then therapy should be discontinued, but if the symptoms do not stop or recur, then esophagogastroduodenoscopy and other studies should be performed. The indication for this therapy is the appearance of symptoms of ulcer after successful eradication of HP.

Relapse prevention.Seasonal prophylaxis - courses of taking antacids, M-anticholinergics, sedatives, anabolic steroids in the autumn-spring periods. Prolonged use of at least 3-4 years of maintenance doses of H2 blockers - histamine receptors (cimetidine 400 mg at night) or gastrocepin (50 mg at night) - a cholinergic receptor blocker.

Since 2016, a new drug has been registered in Russia - rebamipide (REBAGIT), which has been actively used in previous years in a number of countries as a gastro- and enteroprotector. Its mechanisms of action are to stimulate the synthesis of prostaglandins PGE2 and PGI2 and glycoproteins of the gastric mucosa, as well as inhibition of oxidative stress products, proinflammatory cytokines and chemokines / Rebamipide helps to improve the blood supply to the gastric mucosa,

activates its barrier function, as well as the alkalizing function of the stomach, enhances and replacement of gastric epithelial cells [14].

Rebamipide is the only gastroprotective agent with proven efficacy against regression of gastritis changes associated with H. pylori infection. Persistence of H. pylori infection in the gastric mucosa leads to the induction of the production of interleukin (IL) 8 by epithelial cells and the subsequent initiation of neutrophil chemotaxis. This is especially pronounced in H. pylori strains expressing the CagA cytotoxin. After adhesion, H. pylori using a type IV secretion system translocates the cytotoxin CagA into the intracytosolic space of the epithelial cell [1, 4, 16]. Activation of nuclear factor kappa B (NF-kB) and subsequent transcription of pro-inflammatory genes induce the production of IL-8.

Rebamipide has demonstrated the ability to block signal transduction pathways involved in the activation of IL-8 synthesis [17, 18]. In vitro studies have shown that rebamipide reduces the expression of IL-8 and activates NF-kB in malignant cells of the gastric mucosa (in experimental models), including in response to the translocation of the cytotoxin CagA. Similar results were obtained in an in vivo study: with continued administration of rebamipide after the end of ET, the expression of IL-8 and tumor necrosis factor alpha significantly decreased. Clinically, these changes reflect the results of studies that showed that long-term administration of rebamipide leads to a regression of morphological signs of gastritis, expressed in lymphocytic-neutrophilic infiltration of the gastric mucosa.

The presented data on the potential anti-Helicobacter effect of rebamipide have been confirmed in clinical studies of the drug's efficacy in the framework of ET regimens. A recent meta-analysis of six randomized clinical trials (RCTs) (611 patients) demonstrated good efficacy of rebamipide when included in the ET regimen: 73.3 versus 61.4% in the comparison group. The odds ratio (OR) of successful eradication with rebamipide in ET regimens was 1.74 (95% confidence interval (CI) 1.19–2.53). It should be noted that in the study under consideration, there were no significant differences in the frequency of side effects in both groups (OR 0.69; 95% CI 0.376-1.300; p = 0.329).

A number of researchers have analyzed the efficacy of rebamipide treatment at the end of ET. In RCTs, in 309 patients who completed ET and continued to receive rebamipide, the percentage of gastric ulcer scarring was higher than in patients using placebo - 80 versus 66.1% (95% CI 3.1-24.7; p = 0.013).

A similarly designed RCT comparing the efficacy of rebamipide or omeprazole after ET showed a comparable efficacy in gastric ulcer scarring by week 12 of treatment (81.2% versus 82.5%). An important point: against the background of the use of rebamipide, the minimum incidence of side effects was recorded, which in total was 0.54%.

So, according to the data of experimental and clinical studies, in addition to gastroprotective activity, rebamipide realizes its own anti-Helicobacter potential, which is reflected in a significant increase in the effectiveness of ET. In addition, the drug can be used to continue therapy after the end of ET in order to potentiate the repair of the gastric mucosa.

Please note: the drug is most effective in a dose of 100 mg three times a day (with a little water). The course of treatment is from two to four weeks. However, as the results of the study show, the maximum effectiveness is achieved when the course is extended to eight weeks. Subsequently, according to indications, repeated courses of therapy at the same dose can be used.

Thus, the results of a number of experimental and clinical studies indicate that rebamipide is an effective gastroprotector that increases the effectiveness of ET regimens, and, consequently, the treatment of H. pylori-associated gastric ulcer and duodenal ulcer and the prevention of its recurrence. It is advisable to use rebamipide in the post-eradication period to potentiate the repair of the gastric mucosa.

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