

**CLINICAL, INSTRUMENTAL
AND LABORATORY EXAMINATION
OF PATIENTS WITH GASTRITIS,
GASTRIC AND DUODENAL ULCERS.
BASIC SYMPTOMS AND SYNDROMES**

***Independent study manual
for medical students***

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

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**КЛІНІКО-ІНСТРУМЕНТАЛЬНЕ
ТА ЛАБОРАТОРНЕ ДОСЛІДЖЕННЯ
ХВОРИХ НА ХРОНІЧНИЙ ГАСТРИТ,
ВИРАЗКОВУ ХВОРОБУ ШЛУНКА
І 12-ПАЛОЇ КИШКИ.
ОСНОВНІ СИМПТОМИ І СИНДРОМИ**

*Методичні вказівки
для самостійної роботи студентів
медичних факультетів*

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Упорядники Т. В. Ащеулова
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CLINICAL SYMPTOMS AND SYNDROMES IN GASTROINTESTINAL DISEASES

The main symptoms of the digestive system diseases are: pain of various locations, disturbance of appetite or taste, heartburn, belching, nausea, vomiting, dysphagia, meteorism, diarrhea, constipation, bleeding. However, the combination of these complaints depends on the defeat of a certain part of the digestive tract.

The main syndromes of the digestive system diseases are: pain syndrom, dyspeptic syndrome, maldigestion and malabsorption syndrome, astheno-vegetative syndrome, hemorrhagic syndrome, intoxication syndrome.

Pain syndrom: pain behind the sternum, radiating to the left arm, scapula, interscapular region, lower jaw, not associated with physical activity and in with absence of changes in the ECG – with diseases of the esophagus; pains in the epigastric region (in the center of the epigastrium, to the right or left of the midline, umbilical region), various intensities that appear after eating (after 0.5–1 hours, after 1.5–2 hours), often nocturnal, “hungry” (6–7 h after eating) - with pathology of the stomach and duodenum; pain in the umbilical region, often spreading throughout the abdomen; pain in the flanks (in the colon projection) of different character, can be reduced after flatus, defecation

Dyspeptic syndrome: appetite disturbance (decrease, increase, or its complete loss-anorexia), bad taste in the mouth, nausea, vomiting, belching, weight loss (with slowed motor function of the stomach), taste perversion, food aversion

Maldigestion and malabsorption syndrome: diarrhea, meteorism, weight loss, anemia, multivitamin deficiency (impaired absorption of fat-soluble (A, D, K, E) and water-soluble (B1 and B2) vitamins), electrolyte disorders (hypokalemia, hypocalcemia, hypomagnesemia).

Astheno-vegetative syndrome: low working capacity, greater fatigability, irritability, emotional lability, sweating, palpitations, hand shaking, “lump in the throat” sensation, carcinophobia.

Hemorrhagic syndrome: bleeding due to erosion, peptic ulcers.

Intoxication syndrom: fever, general weakness, malaise

Chronic gastritis

Chronic gastritis (CG) is a chronic recurrent process of the gastric mucosa (GI), which is based on inflammation, cell regeneration, atrophy of the glandular epithelium, secretory dysfunction, motor or evacuation disorders, gastric endocrine dysfunction.

The main etiological factors causing the development of chronic gastritis:

– *Helicobacter pylori* infection (the source of infection is a sick person or a bacteriocarrier, in the saliva, stool and plaque of which *Helicobacteria* can be detected);

– autoimmune (the formation of autoantibodies to the parietal (lining) cells of the gastric mucosa that produce hydrochloric acid and Castle's internal factor – gastromukoprotein);

- duodenogastric reflux (there is a destruction of the mucous barrier and the formation of reflux gastritis);
- food allergy (an allergic reaction to dairy products, eggs, chocolate, fish, etc.) leads to the development of inflammatory changes in the gastric mucosa, an increase in the number of plasma cells synthesizing immunoglobulins E, G, M);
- treatment with gastrotropic drugs (long-term use of certain drugs: acetylsalicylic acid, indomethacin, potassium chloride, reserpine, anti-tuberculosis drugs, etc.);
- Alimentary (violation of the rhythm of nutrition, intake of poor-quality food, abuse of spicy, very cold or very hot food);
- endogenous (chronic infections of the upper respiratory tract, diseases of the endocrine system, metabolic disorders, iron deficiency, chronic pulmonary, cardiac, renal failure);
- smoking (long-term smoking leads to damage to the protective mucous barrier);
- alcoholism (frequent use of alcohol leads to the development of atrophic gastritis).

Classification of Gastritis: updated Sydney system (1996)

- *Non-atrophic gastritis* (synonym - antral, hypersecretory)
- *Atrophic, autoimmune, multifocal* (diffuse, corporal, associated with pernicious (B₁₂-deficient) anemia, type A)
- Special forms:
 - chemical (reactive reflux gastritis);
 - radiation;
 - lymphocytic (associated with celiac disease);
 - non-infectious granulomatous;
 - eosinophilic (food allergy, other allergens);
 - other infections.

Chronic non-atrophic gastritis (Helicobacter pylori-infection associated)

Chronic non-atrophic gastritis accounts about 80 % of all types of chronic gastritis. Almost 100 % relationship was established between Helicobacter pylori infection, chronic gastritis and peptic ulcers. Most often detected in the elderly. Mainly affects the pylorus of the stomach, but can spread to all its areas (pangastritis).

Clinical features of the chronic non-atrophic gastritis depend on the stage of the disease.

In an early stage, the process is localized in the antrum of the stomach without secretory insufficiency. Patients have epigastric pain 1.5–2 hours after eating, often hungry pains, heartburn, sour belching, tendency to constipation, appetite preserved.

In a late stage (pangastritis) atrophy of the gastric mucosa occurs with the development of secretory insufficiency. Complaints: poor appetite, nausea,

belching (air or smell like food and like rotten eggs), feeling of heaviness and fullness in the epigastrium after eating, frequent and loose stools

Physical examination

During a general examination, depending on the stage of the disease, slight or significant weight loss, tongue clean, slightly or densely coated at the root, sores at the corners of a mouth, local or diffuse pain in the epigastrium, rumbling during palpation of the large intestine.

Laboratory and instrumental examination methods:

1. **Clinical, biochemical and immunological** analysis reveal no characteristic changes.
2. Cytological, microbiological, histological, immunological methods are used to diagnose *Helicobacter pylori* infection.
3. **Assessment of the gastric secretory function:** production of the gastric acid and pepsinogen may be normal, increased or decreased, but there is no achlorhydria.
4. **X-ray examination:** rough or sluggish mucosal relief, segmental or sluggish peristalsis, irregular or accelerated evacuation of the contrast agent
5. **EGD:** the gastric mucosa is hyperemic or pale, hemorrhages and erosions, hyperplasia of folds, exudation, antral spasm, stasis, vascular translucence, reflux of duodenal contents are often seen.
6. **Histological examination of endoscopic material:** foci of intestinal metaplasia, atrophy of the glandular epithelium, presence of helicobacteria.

Chronic atrophic gastritis

Chronic atrophic gastritis occurs due to a decrease in the endogenous mechanisms of protecting the gastric mucosa and is characterized by atrophy of the gastric mucosa and secretory insufficiency

It is rare. It is localized in the fundus of the stomach, which leads to a sharp decrease in the secretion of hydrochloric acid, pepsinogen, an internal factor and causes the development of B₁₂-deficient anemia.

Clinical features: weakness, dizziness, sweating, the feeling of heaviness and fullness in the epigastric region or upper abdomen shortly after eating which has a monotonous character, often belching (air, bitter and like rotten eggs), heartburn, metallic taste in mouth, bleeding gums, reduced appetite, weight loss.

Physical examination

General inspection: skin is pale, dry, sometimes darkening due to hypocorticism development, sores at the corners of a mouth (angular cheilitis), furred tongue.

Laboratory and instrumental examination methods:

1. **Clinical blood test:** a decrease in the number of red blood cells and in hemoglobin concentration, an increase in color index, leukopenia, thrombocytopenia.
2. **Biochemical blood test:** hyperbilirubinemia, increased χ -globulins.
3. **Immunological blood test:** a decrease in T-lymphocyte suppressors, an increase in T-lymphocyte helper cells and immunoglobulins, presence of immune complexes and antibodies to: parietal cells, gastromucoprotein, vitamin B₁₂, KH⁺, K--ATP-ase, and gastrin-binding proteins

4. **Assessment of the gastric secretory function:** considerable decrease in production of the gastric acid and pepsinogen that can result in achlorhydria.

5. **X-ray examination of the stomach:** folds of the gastric mucosa are less numerous

6. Upper Endoscopy (EGD): mucosal folds are lower or may be absent at all, the mucous membrane is pale, thin, atrophic, presence of excess mucus, dumping of stomach contents into the duodenum, sluggish peristalsis, mucus on the stomach walls, erosions are rare

Peptic Ulcer

Peptic ulcer (PU) is a pathological process based on inflammation of the mucous membrane of the gastroduodenal area with the formation of local erosive damage of infectious or non-infectious origin as a response to the endogenous disbalance between local "aggressive" and "protective" factors

It occurs in 6–10 % of the adult population, more often in men than in women (male to female ratio is 4: 1), with a predominant lesion of the urban population.

Etiological factors:

– *Helicobacter pylori* infection (in almost 100 % of cases it is a leading factor in the occurrence of duodenal ulcers, in 80–90 % of cases – stomach ulcers);

– acute and chronic stress;

– alimentary factor (it role is insignificant, however, you should avoid eating foods that cause excessive gastric secretion: very sharp, rough, too hot or too cold);

– alcohol abuse is not the root cause of the development of peptic ulcer, but contribute to the stimulation of the secretion of hydrochloric acid and development of ischemia of the gastric mucosa;

– smoking (in smokers this disease is twice as common as non-smokers, since nicotine causes narrowing of blood vessels and ischemia of the gastric mucosa, stimulates the secretion of hydrochloric acid, accelerates the evacuation of food from the stomach, and creates the conditions for the formation of the gastroduodenal reflux);

– effect of drugs (acetylsalicylic acid; non-steroidal hormones that have a necrotizing effect on the gastric mucosa and lead to its inhibition of the formation of protective prostaglandins; reserpine, which stimulates the secretion of hydrochloric acid; glucocorticoids that disrupt microcirculation, also stimulate the secretion of hydrochloric acid and reduce the ability to regenerate and reduce gastric mucosa);

– endogenous factors (chronic diseases of the lungs and cardiovascular system, which lead to the development of ischemia of the gastric mucosa, cirrhosis of the liver, pancreatic diseases).

Clinical features uncomplicated peptic ulcers depend on the location of the ulcer, the age of the patient, the presence of concomitant diseases and complications, but the leading syndromes are pain and dyspeptic.

● *The pain* is localized in the center of the epigastrium or to the left from the median line (gastric ulcers); in the epigastrium, to the right of the median line (duodenal and prepyloric ulcers); behind the sternum or to the left from it (in

the precordial region or at the apex of the heart) – gastric cardia ulcers; in the back or in the epigastrium, to the right from the median line (postbulbar ulcers)

- *Heartburn* is caused by gastroesophageal reflux, irritation of the mucous membrane of the esophagus with gastric contents and is not always associated with food intake.

- *Belching* due to cardia insufficiency and antiperistaltic contractions of the stomach. It can be air, sour, rotten.

- *Nausea* is usually associated with concomitant gastritis, is characteristic of the mediogastric, often for postbulbar ulcers.

- *Vomiting* is caused by increased vagus tonus, increased gastric motility, and gastric hypersecretion. It occurs at the "height" of pain, contributes to a significant reduction or even disappearance of pain. Vomit contains acidic gastric contents.

- *Appetite* is often good, sometimes increased, less often reduced. With severe pain, cytophobia occurs - a fear of pain after eating.

- *Constipation* – disorders of the motor function of the large intestine resulting from spastic contractions of the colon, physical inactivity, antacids, and low fiber in the diet.

Physical examination.

General inspection: most often asthenic or normosthenic constitutional type, cold and sweaty palms

Palpation in the exacerbation phase: pain in the epigastric, duodenal area, moderate muscles resistance of the anterior abdominal wall

Percussion: local tenderness over the same area

Laboratory and instrumental examination methods:

1. ***General blood test:*** in complicated forms of peptic ulcer – leukocytosis and accelerated ESR, anemia (due to bleeding).

2. ***Stool test:*** melena, Gregersen test.

3. Diagnosis of *H. pylori* infection.

4. Intra-gastric pH-metry to assess acid-forming function (rapid method) and many hours or daily pH monitoring for the individual selection of a particular drug.

5. Fibrogastroduodenoscopy allows you to distinguish an active ulcer, scarring, post-ulcer scars.

Exacerbation phase. The following symptoms are characteristic: the form of the ulcer is round, oval, slit-like; edges have clear boundaries, hyperemic, swollen; the bottom is covered with fibrinous overlays of grayish-white or yellow color; in a biopsy from the ulcer bottom - detritus, accumulation of mucus, white blood cells, red blood cells, desquamated epithelial cells, edema, infiltration by lymphocytes, plasma cells.

Healing stage. The red scar phase: a decrease in hyperemia and swelling of the mucous membrane around the ulcer, a decrease in the depth of the ulcer, cleansing of the bottom of fibrin, the appearance of granulation tissue, mild edema and infiltration by plasma cells and lymphocytes in the biopsy specimen.

Scar stage. The “white” scar phase: the absence of active inflammation, replacement of connective granulation tissue, severe deformation of the wall of the stomach or duodenum.

6. **Morphological study of biopsy** (targeted gastroscopic biopsy) to detect ulcerative forms of gastric cancer, epithelial metaplasia.

7. **X-Ray examination signs:** “niche” (crater) symptom surrounded by an inflammatory border; convergence of mucosal folds towards “niche”, “Pointing finger” symptom – local indrawing of the greater curvature opposite an ulcer on the lesser curvature pointing fingerlike to a crater, accelerated passage of the barium suspension in the ulceration area.

Complications of peptic ulcers:

- perforation
- gastrointestinal bleeding
- stenosis due to ulceration scarring
- penetration
- malignancy

TESTS

1. **"Virchow's gland" is typical for which of the following disease?**
A. *Hepatitis.* C. *Peritonitis.* E. *Colitis.*
B. *Pancreatitis.* D. *Cancer of the stomach.*
2. The lag of the anterior abdominal wall during respiratory movements is observed at which of the the following disease?
A. *Cholecystitis.* B. *Ascites.* C. *Obesity.* D. *Peritonitis.* E. *Gastritis.*
3. The greater curvature of the stomach is palpated in which of the following region?
A. *To the left and above the navel.* D. *To the left and bottom of the navel.*
B. *To the right and above the navel.* E. *To the right and bottom of the navel.*
C. *Above the navel.*
4. The pyloric stomach is palpated in which of the following region?
A. *Left and above the navel.* D. *Left and bottom of the navel.*
B. *To the right and above the navel.* E. *To the right and bottom of the navel.*
C. *Above the navel.*
5. A positive symptom of Shchetkin-Blumberg is observed at which of the following complication of peptic ulcer?
A. *Perivisceritis.* B. *Penetration.* C. *Malignization.* D. *Perforation.* E. *Bleeding.*
6. “Hunger pain” is typical for which of the following disease?
A. *Cholecystitis.* C. *Pancreatitis.* E. *Hepatitis.*
B. *Cholangitis.* D. *Duodenum ulcer disease.*
7. Melena is observed with which of the following?
A. *Intestinal bleeding* D. *Hemolytic jaundice*
B. *Intraabdominal bleeding* E. *Parenchymatous jaundice*
C. *Gastric bleeding*

Correct answers: 1D, 2D, 3A, 4B, 5D, 6D, 7C.

Навчальне видання

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