

**MINISTRY OF HEALTHCARE OF UKRAINE  
HSEEU "Ukrainian Medical Stomatological Academy"**

**"Approved"**  
**at the meeting of internal**  
**medicine №1 department**  
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**Protocol № 2 from 15.09.2016**

**GUIDELINES  
FOR STUDENTS  
INDEPENDENT WORK  
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Basics of Internal Medicine
<i>Content module</i>	Fundamentals of diagnostics, treatment and prevention of gastroenterological diseases
<i>Study subject</i>	<b>Peptic ulcers and other gastric and duodenal ulcers. Chronic gastritis.</b>
<i>Course</i>	IV
<i>Faculty</i>	of foreign students training

Poltava 2016.

**1. Relevance of the topic:** A **peptic ulcer** is a mucosal defect that penetrates the muscularis mucosae. Gastric and duodenal ulcers usually occur in an area of inflamed mucosa. This inflammation, termed gastritis, duodenitis, or bulbitis, can sometimes be recognized during endoscopy by signs of edema, reddening, and swelling of the mucosa, but microscopic evaluation of endoscopic biopsy specimens is required for a definitive diagnosis of mucosal inflammation. **Chronic gastritis** is an inflammatory, dystrophic chronic disease of the lining of the stomach, that is characterized by cell infiltration, abnormal regeneration and can lead to atrophy of glandular epithelium, metaplasia and/or dysfunction of secretory, motoric or incretory activities of stomach. Gastritis is mostly a histological term that also needs biopsy to be confirmed.

**2. The main goal:** To be able to assess the typical clinical picture of peptic ulcer and chronic gastritis, to determine tactics of treatment and prophylaxis.

Specific goals:

- To select the information indicating the presence of peptic ulcer and chronic gastritis in a patient from the data history;
- To create a scheme of diagnostic search;
- To identify the signs of peptic ulcer and chronic gastritis in an objective study of the patient (general examination, palpation, percussion, auscultation)
- To analyze and interpret the changes in the results of the laboratory and instrumental methods of investigation, depending on the course of the disease;
- To formulate and justify a preliminary diagnosis of peptic ulcer and chronic gastritis according to classification;
- To conduct differential diagnostics of diseases with the similar clinical picture;
- To develop a strategy of treatment depending on the disease and the existing complications;
- To provide medical care;
- To assess the patient's prognosis and to propose a plan of preventive actions;
- To apply deontological communication skills with patients.

### 3. Basic knowledge, abilities, skills (interdisciplinary integration)

Discipline	To know	To be able to
Anatomy	The structure of the gastrointestinal tract, blood supply, innervation	
Histology	The structure of the wall of the esophagus, stomach, intestines in health and disease	To interpret results of upper endoscopy with biopsy
Regional anatomy	Interposition of the gastrointestinal organs	
Physiology	Indicators of gastrointestinal tract function, its value	To determine the function of gastrointestinal organs
Morbid anatomy	Changes in the structure of the wall of stomach	
Radiology	Radiological changes	Analyze the radiological picture of the abdominal cavity

Propaedeutic therapy	Symptomatology of peptic ulcer disease and chronic gastritis and its complications	Conduct an objective examination of the patient, analyze the clinical and laboratory results
Pharmacology	The mechanism of action, indications and contraindications for the IPP, H <sub>2</sub> -blockers, antacids, prokinetics, antibiotics, antidiarrheal drugs	Prescribe the drugs of these groups

#### 4. Do the tasks for independent work during preparation for classes.

##### 4.1. The list of key terms, parameters, characteristics:

Term	Definition
Peptic ulcer	is a mucosal defect that penetrates the muscularis mucosae.
Gastritis	inflammation associated with epithelial cell damage and regeneration.
Gastropathy	mucosal injury (in which there is cell damage and regeneration) without inflammation.
Atrophy	loss of normal mucosal glands.
Metaplasia	change in epithelial cell types.
Chromoendoscopy	is an endoscopic technique that uses stains during endoscopy to highlight differences in mucosa, as well as dysplastic and malignant changes that are not apparent in white light.

##### 4.2. Theoretical questions for the lesson:

1. Give the definitions of ulcer and gastritis.
2. Specify the risk factors of stomach ulcer and chronic gastritis.
3. Name the pathophysiological mechanisms of stomach ulcer and chronic gastritis.
4. Name the diagnostic criteria of peptic ulcer disease and chronic gastritis.
5. What are the endoscopic characteristics of peptic ulcer disease and chronic gastritis?
6. Modern classification of peptic ulcer disease and chronic gastritis.
7. Specify the principles and features of peptic ulcer disease and chronic gastritis pharmacotherapy according to modern recommendations.
8. What lifestyle modifications should be recommended for patients with peptic ulcer disease and chronic gastritis?

##### 4.3. Practical tasks that are performed in class:

1. Etiology of chronic gastritis type A:
  - 1) H. pylori
  - 2) NSAIDs
  - 3) autoimmune
  - 4) chemical damage
2. H<sub>2</sub>-blockers include:

**1) Famotidine**

2) Itoprid

3) Pantoprazole

4) Clarithromycine

3. Prokinetics include:

1) Famotidine

**2) Itopride**

3) Pantoprazole

4) Clarithromycine

4. IPP include:

1) Famotidine

2) Itoprid

**3) Pantoprazole**

4) Clarithromycine

5. The most common etiological factor of peptic ulcer disease:

1) long-term NSAIDs intake

2) duodenogastral reflux

**3) H. pylori infection**

4) stress

6. Triple therapy of H. pylori infection includes:

1) IPPs standard dose bid, clarithromycin 250 mg bid, amoxicillin 500 mg bid/metronidazole 500 mg bid

2) IPP standard dose bid, bismuth subcitrate 120 mg qid, metronidazole 500mg tid, tetracycline 500 mg qid

**3) IPPs standard dose bid, clarithromycin 500 mg bid, amoxicillin 1000 mg bid/metronidazole 500 mg bid**

4) IPPs standard dose bid, levofloxacin 500mg qd, amoxicillin 1000mg bid/rifambutine 300mg qd

7. Quadruple therapy of H. pylori infection includes:

1) IPPs standard dose bid, levofloxacin 500mg qd, amoxicillin 1000mg bid/rifambutine 300mg qd

2) IPPs standard dose bid, clarithromycin 500 mg bid, amoxicillin 1000 mg bid/metronidazole 500 mg bid

**3) IPP standard dose bid, bismuth subcitrate 120 mg qid, metronidazole 500mg tid, tetracycline 500 mg qid**

4) IPPs standard dose bid, clarithromycin 250 mg bid, amoxicillin 500 mg bid/metronidazole 500 mg bid

8. What testing of H. pylori can be positive after HP eradication:

1) histologic examination of gastric mucosa

2) polymerase chain reaction

3) stool antigen test

**4) serum antibodies**

9. "Big ulcer" of stomach has size of:

**1) 2-4 cm**

2) 4-6 mm

3) 0,5-1 cm

4) 4-6 cm

10. "Small ulcer" of duodenum has size of:

1) 0,3-0,5 cm

**2) less than 0,3 cm**

3) 1-1,5 cm

4) 0,4-0,6 cm

## **Topic Content**

### **PEPTIC ULCER DISEASE**

**Definition.** A **peptic ulcer** is a mucosal defect that penetrates the muscularis mucosae. Gastric and duodenal ulcers usually occur in an area of inflamed mucosa. This inflammation, termed gastritis, duodenitis, or bulbitis, can sometimes be recognized during endoscopy by signs of edema, reddening, and swelling of the mucosa, but microscopic evaluation of endoscopic biopsy specimens is required for a definitive diagnosis of mucosal inflammation.

**Epidemiology.** The worldwide prevalence of gastritis reflects the prevalence of *H. pylori*. Colonization with this bacterium is virtually always associated with chronic active gastritis, which persists as long as an individual remains colonized and only slowly disappears 6 to 24 months after the eradication of *H. pylori*. Although peptic ulcer disease is strongly related to *H. pylori* gastritis and duodenitis, the epidemiology of ulcer disease has shown secular variations even when *H. pylori* was ubiquitous.

**Etiology.** *Helicobacter pylori* (HP) infection, non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, cytostatics, Zollinger-Ellison syndrome, systemic inflammatory diseases, stress, alcohol, genetic.

**Pathogenesis.** Most peptic ulcers are associated with colonization with *H. pylori*, which has urease activity. Urease activity creates a "cloud" of ammonia around the bacterium, thus neutralizing the lethal effects of gastric acid. Motility allows the bacterium to penetrate the mucus layer and promotes specific association of the bacteria with epithelial cells, further allowing evasion of gastric acidity.

On the basis of their activities, NSAIDs are divided into cyclooxygenase 1 (COX1) and COX2 inhibitors. The COX1 enzyme is involved in the production of prostaglandins, which play a role in normal cell regulation. The COX2 enzyme, which is also involved in the production of prostaglandins, is induced by inflammatory responses. Most NSAIDs have a nonselective COX inhibitory effect; selective COX2 inhibitors are associated with fewer gastroduodenal ulcers, but their use is limited by their adverse coronary effects. Because of the strong association between NSAIDs and ulcer disease and the risk for recurrence of ulcers with their continued use, patients with ulcers must be thoroughly assessed for the use of NSAIDs.

Gastroduodenal ulcers can result from underlying malignant disease. In the stomach, these tumors are related to gastric adenocarcinoma and, rarely, to mucosa-associated lymphoid tissue (MALT) lymphomas. Malignant ulcers in the duodenum may result from primary duodenal carcinomas or from penetrating pancreatic cancers.

Peptic ulcers can result from chronic gastric hyperacidity related to hypergastrinemia. The most important hypergastrinemic disorder is Zollinger-Ellison syndrome, a condition of marked hyperacidity leading to severe peptic ulcer disease caused by a gastrin-producing endocrine tumor.

But the most common element of ulcer pathogenesis is imbalance between factors of aggression (pepsin, hydrochloric acid, hypertonus of n.vagus) and defense of mucous membrane and physiological regeneration. It leads to chronic inflammation and results in ulceration.

**Classification:**

According to location:

- gastric ulcers are subdivided into proximal ulcers, located in the body of the stomach, and distal ulcers, located in the antrum and angulus of the stomach; located along the curvature
- duodenal ulcers usually are located on the anterior or posterior wall of the duodenal bulb, or occasionally at both sites (“kissing” ulcers); lesions distal to the duodenal bulb are termed postbulbar ulcers; located in bulb – bulb ulcer.

According to size:

- small (less than 1cm in stomach; less than 0,3 in duodenum)
- average (1-2cm in stomach; 0,3-0,5cm in duodenum)
- big (2-4cm in stomach; 0,6-1,0cm in duodenum)
- huge (giant)

According to HP association:

- HP associated
- HP nonassociated

According to periods:

- exacerbations
- remission

According to grades:

- I – without complications, detected for the first time;
- II – without complication, with yearly exacerbations;
- III – with complications,
- IV – recurrence after surgery.

According to complications:

- stenosis
- penetration
- perforation
- bleeding
- malignisation.

*Example of diagnosis:* Peptic ulcer disease, I grade, HP-positive, acute small (0,1x0,2cm) ulcer of duodenal bulb, period of exacerbation.

**Clinical symptoms:**

- dyspepsia (heartburn, blenching, nausea, vomiting giving relief, constipation)
- pain syndrome (always associated with meal, in epigastrium or pyloroduodenal area, intensive, may radiate to the back, thorax, other parts of abdomen, may be nocturnal (specific for duodenal ulcer), “painful hunger” relieved by food (specific for duodenal ulcer), may be postprandial and relieved by fasting (specific for duodenal ulcer))
- general weakness

**NB!** Remember about “red flags” symptoms!

**Physical examination.** Tongue is coated with white fur. Pain in epigastric or pyloroduodenal area at palpation.

The patient may present with pallor and may be hypovolemic. It is always useful to inquire about the characteristics of the stool, because ulcer-related bleeding may manifest not

only obviously in the form of hematemesis but also insidiously as melena (black feces). In the case of massive ulcer bleeding with the rapid bowel passage of blood, patients may also present with red rectal blood loss. When a patient has acute perforation, severe epigastric and abdominal pain develops, and the patient appears distressed. Characteristically, intense contracture of the abdominal muscles is apparent on palpation, together with rebound tenderness and other signs of peritoneal irritation. With large amounts of intraabdominal air, percussion may reveal hypertympany over the liver.

**Laboratory and instrumental methods:**

- CBC
- biochemical blood test
- serum gastrin elevation
- gastrin provocative tests (intravenous secretin, meal)
- gastric analysis
- feces occult blood test
- upper endoscopy with biopsy (is the primary investigative tool in patients suspected of having acid peptic disease)
- ultrasound diagnostic of abdominal cavity
- gastroduodenoscopy barium contrast (inferior alternative)
- endoscopic ultrasound (selected cases only)
- ECG
- computed tomography (useful in selected cases)

**HP testing:**

- histologic examination of gastric mucosa
- bacteriologic examination of gastric mucosa
- fast urease test (in biopsy specimens)
- stool antigen test (more accurate)
- carbon-13 urea breath test (noninvasive and relatively simple test, but it is more expensive than stool or blood testing)
- serum antibodies (is not helpful to verify whether *H. pylori* has been eradicated with antibiotics because it may take many months or even years for *H. pylori* antibodies to fall to undetectable levels)
- polymerase chain reaction (PCR)

**Differential diagnosis** includes many disorders of the upper abdominal organs, including malignant diseases of the stomach, duodenum, pancreas, or bile ducts. The differential diagnosis of upper abdominal symptoms also includes liver and gallstone disease, pancreatitis, and motility disorders. In many patients with upper abdominal dyspeptic complaints, no underlying cause can be identified. In this “nonulcer” or functional dyspepsia group, complaints characteristic of gastroesophageal reflux, ulcer symptoms, or dysmotility symptoms may be prominent. A few of these patients (generally 5%) benefit from eradication of *H. pylori*.

Differential diagnosis of gastric and duodenal ulcer. Gastric ulcer: peak 50-60 y., pain often diffuse, variable - squeezing, heaviness, or sharp punctuating (may absent), poorly localized, may radiate to back, 1-3 h after food, aggravated by meals, severe gastric pain well radiating indicate penetration or perforation, seasonal occurrence (autumn, spring). Duodenal ulcer: male patients, peak 30-40 y., pain well localized epigastric, chronic, intermittent, relieved by alkalic food, often late onset 6-8 h after meal or independent (night), familiar occurrence, smokers, blood O type, complication - penetration onto pancreas.

**Treatment.** The goal of therapy for peptic ulcer disease is to relieve symptoms, heal craters, prevent recurrences, and prevent complications.

1) acid suppression

- Proton pump inhibitor (described in GERD)
- H<sub>2</sub>-blockers (described in GERD)

2) anti HP therapy

- First-line HP eradication therapy. Triple therapy (IPPs standard dose bid, clarithromycin 500mg bid, amoxicillin 1000mg bid/metronidazole 500 mg bid 10(7)-14 days)
- Sequential therapy (Standard-dose IPP bid 10 days, clarithromycin 500mg bid 5 days and after it amoxicillin 1000mg+metronidazole 500mg bid 5 days)
- Second-line. Quadruple therapy (IPP standard dose bid, bismuth subcitrate 120 mg qid, metronidazole 500mg tid, tetracycline 500 mg qid 10-14 days)
- “Rescue therapy” (IPPs standard dose bid, levofloxacin 500mg qd, amoxicillin 1000mg bid/rifampicine 300mg qd 10-14 days)

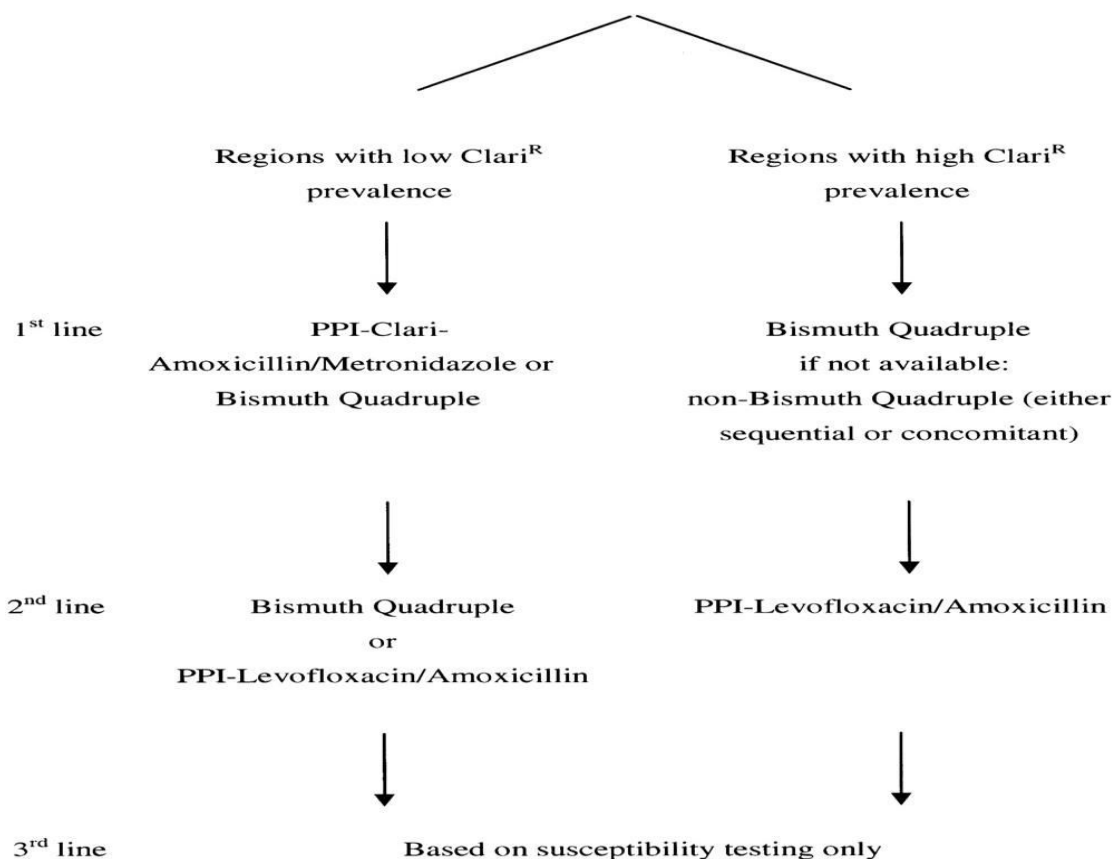
3) Sucralfate is the aluminum salt of a sulfated disaccharide. The drug forms a barrier or coating over the ulcer crater, stimulates prostaglandin synthesis, and binds to noxious agents such as bile salts. Although the exact mechanism of action is unclear, it appears sucralfates stimulate prostaglandins, which promote improved mucosal integrity and enhance epithelial regeneration. Because it requires multiple doses per day, patients are less likely to follow a sucralfate regimen even though it has been shown to be as effective as an H<sub>2</sub> blocker in healing both duodenal and gastric ulcers. Sucralfate is not absorbed systemically, and its only remarkable side effect is constipation. Misoprostol is a prostaglandin E<sub>1</sub> analog that increases mucosal resistance and inhibits acid secretion to a minor degree.

4) Misoprostol has been advocated for prophylaxis of NSAID-induced mucosal injury. The drug has significant side effects, primarily mild to moderate diarrhea, and is too costly to be used by most patients on long-term NSAIDs.

5) symptomatic treatment (mebeverine 200mg bid, itoprid 50 mg tid, UDCA 250 mg before sleep)

6) surgical treatment.





## CHRONIC GASTRITIS

**Definition.** Chronic gastritis is an inflammatory, dystrophic chronic disease of the lining of the stomach, that is characterized by cell infiltration, abnormal regeneration and can lead to atrophy of glandular epithelium, metaplasia and/or dysfunction of secretory, motoric or incretory activities of stomach.

Gastritis is mostly a histological term that needs biopsy to be confirmed.

- *Gastritis*: inflammation associated with epithelial cell damage and regeneration.
- *Gastropathy*: mucosal injury (in which there is cell damage and regeneration) without inflammation.
- *Atrophy*: loss of normal mucosal glands.
- *Metaplasia*: change in epithelial cell types.

Gastritis is categorized by endoscopic and histologic criteria, with granulocytes predominating in active gastritis and mononuclear cells in chronic gastritis.

**Etiology.** *Helicobacter pylori* affected in about half of populations in the world, is the major cause of gastritis. Other sources include chemical agents (nonsteroidal anti-inflammatory drugs, bile reflux into stomach, etc.) and autoimmunity. *H. pylori* are considered as a grade 1 carcinogen of gastric cancer. Dietary factors, alcohol, smoking and other diseases (diabetes mellitus, Crohn's disease, etc).

**NB!** According to some authors by irritants such as drugs (eg, nonsteroidal anti-inflammatory agents and alcohol), bile reflux, *gastropathy* is usually caused.

**Pathogenesis.** Chronic inflammation of gastric mucous membrane leads to regeneration breaks, that causes atrophy and possible mucous dysplasia of stomach.

**Classification.** According to endoscopic and histological divisions, combining topographical, morphological and etiological information to generate reproducible and clinically useful diagnoses Sidney classification was worked out (1996):

- Atrophic, autoimmune – Type A (diffuse, body and fundus of the stomach, associated with B<sub>12</sub>-anemia)
- Nonatrophic – Type B (HP associated, antral)
- Multifocal (HP, diet factors, antrum+corpus)
- Chemical – Type C (chemical factors, alcohol, reflux-gastritis (bile), nonsteroidal anti-inflammatory drugs-associated)
- Radiation
- Lymphocytic (idiopathic, celiac disease-associated)
- Noninfectious granulomatosis (Crohn-disease, granulomatosis, sarcoidosis etc.)
- Eosinophilic (allergic)
- Other – bacterial, viral, fungal (specific gastritis).

Gastritis is also classified by the segment of involved stomach: antral-predominant gastritis, corpus-predominant gastritis, or pangastritis.

To evaluate the severity of atrophic changes new classification was proposed: Operative Link for Gastritis Assessment (OLGA, 2008). This system ranks the gastric cancer risk according to both the topography and the severity of gastric atrophy according to routine biopsy sampling.

		CORPUS			
		No atrophy (grade 0)	Mild atrophy (grade 1)	Moderate atrophy (grade 2)	Severe atrophy (grade 3)
ANTRUM	No atrophy (grade 0)	STAGE 0	STAGE I	STAGE II	STAGE II
	Mild atrophy (grade 1)	STAGE I	STAGE I	STAGE II	STAGE III
	Moderate atrophy (grade 2)	STAGE II	STAGE II	STAGE III	STAGE IV
	Severe atrophy (grade 3)	STAGE III	STAGE III	STAGE IV	STAGE IV

**Complaints.** Gastritis can be asymptomatic. But the most common symptoms are:

- dyspepsia (indigestion) – upper abdominal postprandial fullness, heartburn, nausea, belching, early satiation, bloating
- pain syndrome – epigastric, especially after consumption of spicy, roasted food, usually dull, not intensive, after the meal, but it does not have regular and certain association with it
- general weakness
- symptoms of vitamins deficiency (type A gastritis)

**Physical examination.** Tongue is coated with white fur. Tenderness or pain in epigastrium at palpation. Signs of vitamins deficiency (pallor).

**Clinical, laboratory and instrumental examination.** Comprehensive assessment of clinical examination, serologic test (e.g., antibodies for infection or autoimmunity), endoscopy and histologic examination could be diagnostic tools for patients with gastritis.

Upper endoscopy with biopsy (2 from antrum, 2 from gastric body, 1 from incisura: site most likely to show atrophic gastritis and premalignant dysplasia). Typical histologic findings of

gastritis are: chronic inflammatory infiltrates in lamina propria (lymphocytes, plasma cells and histiocytes), active inflammatory infiltrates in lamina propria and gastric glands (neutrophils and eosinophils) and loss of glandular units with replacement into fibrosis and smooth muscle proliferation, called as atrophy. Chromoendoscopy allows to visualize areas of intestine metaplasia.

- HP tests (described in peptic ulcer disease section)
- pH-monitoring
- antibodies to parietal cells (type A)
- antibodies to internal Castle factor (type A)
- gastropanel (IgG to HP, pepsinogen 1 and 2, gastrin – 17)
- CBC (inflammatory signs, anemia)

**Differential diagnosis.** Should be made with functional dyspepsia, peptic ulcer disease, GEDR, according to leading syndrome.

**Treatment.**

- Type A – treatment of anemia: cyanokobalaminum 500mcg/ml (1-2ml) intramuscular 6 days, then it should be used once a week, after – once at 2 months (to treat anemia).
- Type B – HP eradication (described in peptic ulcer disease section).
- Type C – antacids, alginates, ursodeoxycholic acid (if bile reflux occurs) capsules 250 mg. PPIs, H2-blockers and prokinetics, ferments can be used if there is a need.

**Materials for self-control:**

**Situation tasks:**

1. Patient P., 35 years old, complains of pressing epigastric pain in 1 hour after eating, heartburn, sour belch. He is considered to be ill during last 2 years. A pain in pyloroduodenal area presents upon the abdominal palpation. Upper endoscopy found an antral gastritis. What is the preliminary diagnosis? What additional tests are necessary?
2. A man, 67 years old, complains of appetite loss, feeling of heaviness and bloating in epigastrium after meal, air belch with smell of spoiled meal, nausea. Analysis of gastric secretion detected achilia. EGDS visualized thinned stomach mucosa without vascular picture under it. What is the probable diagnosis? What additional tests are necessary for the patient?

**Tests:**

1. A 27 y. o. man complains of pain in epigastrium which is reduced by meal. EGDS visualizes antral erosive gastritis, in biopsy material of antral mucous *Helicobacter Pylori* was detected. What is the most probable diagnose?

- A. Gastritis of type A
- B. Gastritis of type B
- C. Reflux - gastritis
- D. Menetrier's disease
- E. Rigid antral gastritis

2. 69 y. o. man complains of appetite loss, sensation of heaviness and bloating in epigastrium after meal, belching with air and smell of spoilt food, nausea. Achilia was established according to analysis of stomach secretion. Thinning of mucosa with vivid blood vessels was visualized on FGDS. What is the most probable diagnosis?

- A. Rigid gastritis
- B. Stomach cancer
- C. Atrophic gastritis
- D. Chronic colitis
- E. Non-ulcerative dysphagia

3. A 33 y. o. male patient was got to hospital. The patient is pale, after an attempt to stand up he complains of strong dizziness. There was coffee-like vomiting approximately an hour ago. BP- 90/60 mm Hg., pulse- 120 b/min. From anamnesis: the patient has been suffering from ulcer of stomach, which didn't disturb him for 4 years. An ulcer was visualized at upper endoscopy. Your diagnosis:

- A. Ulcer of stomach, complicated with bleeding
- B. Ulcer of duodenum, complicated with bleeding
- C. Erosive gastritis
- D. Acute pleurisy
- E. Acute myocardial infarction, abdominal form

4. A man, 21 years old, complains of periodic epigastric pain. According to performed examination, chronic gastritis with hyperacidity was found out. Prescribed treatment has positive results. What medicine is expedient to use for primary ulcer prophylaxis?

- A. Famotidine
- B. Cerucal
- C. Vicalin
- D. Maalox
- E. Gastrofarm

5. The 48 years old patient complains of periodic pain in epigastrium, without irradiation, heartburn, which amplify after meals, migraine and sleeplessness. After reception of 20 mg of rabeprazole during first two days these symptoms disappeared. For what disease this clinical picture is typical?

- A. Type A chronic gastritis
- B. Duodenal ulcer
- C. Functional dyspepsia
- D. Chronic pancreatitis
- E. Chronic hepatitis

6. A 31-year-old male patient complains of periodic heartburning. Objectively: HR- 70/min, AP- 125/75 mm Hg. Upper endoscopy confirms esophageal ulcer. Which of the given drugs will be a compulsory element of the treatment?

- A. Omeprazole
- B. Famotidine
- C. Pirenzepine

- D. Atropine
- E. Maalox

7. 42 years old man was found an absence of free hydrochloric acid in all phases of gastric juice. Upper endoscopy visualized pale, thinned gastric mucosa, folds were smoothed. What is the most credible diagnosis?

- A. Cancer of stomach
- B. Chronic gastritis, type B
- C. Chronic gastritis, type C
- D. Menethrie`s disease
- E. Chronic gastritis, type A

8. Patient S., 23 years old, complains of a dull pain, heavy and bloating feeling in epigastrium after meal, belch with rotten food taste in mouth, nausea, diarrhea. Objectively: skin is pale, body weight is reduced a little. Upon the palpation abdomen is soft, pain is marked in epigastrium. Liver does not come from the edge of a costal arc. In blood test: Hb - 110 g/l, E -  $3,4 \times 10^{12}/l$ , leukocyte formula - without changes. Blood sedimentation - 16 mm/h. Name necessary research, which will help to put the diagnosis:

- A. Esophagogastroduodenoscopy
- B. Scintigraphy of digestive organs
- C. Analysis of gastric juice
- D. Ph-monitoring
- E. Duodenal tubage

Correct answers for the situation tasks:

1. Chronic gastritis. Determination of H. pylori.
2. Atrophic gastritis. Detection of autoantibodies to parietal cells in serum.

The answers for the tests:

- 1-B, 2-C, 3-A, 4-A, 5-C, 6-A, 7-E, 8-A.

#### **Recommended literature:**

1. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 24th ed. USA 2012, Elsevier p. 2569
2. Harrison's Principles of Internal Medicine / D.Kasper, A.Fauci, S.Hauser, D. Longo.-19 ed. – N.Y.: McGraw-Hill Professional, 2015. - Vol. 1, Vol.2. - 3000 p.
3. Maastricht V/Florence Consensus Report on Managing Helicobacter pylori Infection <http://www.jwatch.org/na42519/2016/10/20/maastricht-v-florence-consensus-report-managing>

