Ministry of Healthcare of Ukraine

Poltava State Medical University

Approved
at the meeting of Internal Medicine №1
Department "____"_____
Protocol № ____ from _____
The Head of the Department
Associate Professor Maslova H.S.
_____

Methodical guidelines

for students' self-studying to prepare

for practical (seminar) classes and on the lessons

Academic discipline	Internal medicine
Module №	1
Topic of the lesson	Chronic gastritis. Stomach and duodenal ulcers.
Course	IV
Faculty	of foreign students training

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. Certain aims:

– To analyze symptoms from upper GI tract and make the preliminary diagnosis.

– To explain pathogenesis of gastritis, stomach ulcer and duodenal ulcer generally and in individual patients.

– To propose further management tactics for those with suspected gastritis, stomach ulcer and duodenal ulcer.

– To classify gastritis, stomach ulcer and duodenal ulcer.

– To interpret data of CBC, EGDS, 24-hours pH-monitoring and HP tests.

– To draw schemes, charts of patient's follow-up.

– To analyze data of survey, physical examination, additional methods of investigations to confirm the clinical diagnosis of patients with suspected gastritis, stomach ulcer and duodenal ulcer.

– To make the full diagnosis according to the current classifications and prescribe treatment for patients with gastritis, stomach ulcer and duodenal ulcer.

3. Basic knowledge, abilities, skills required to study the topic (interdisciplinary integration).

Names of previous disciplines	Obtained skills

1. Anatomy 2. Histology 3. Anatomy 4. Physiology 5. Pathology 6. Radiology 7. Propaedeutic internal medicine 8. Pharmacology	To describe the structure of the gastrointestinal tract, blood supply and innervation in health and disease; to establish the preliminary diagnosis, to use additional methods of examination and interpret their data to make final diagnosis; to manage the patient with gastritis, stomach ulcer and duodenal ulcer; to classify gastritis, stomach ulcers and duodenal ulcers, and drugs for their treatment; to identify markers of gastrointestinal tract function and to know their normal values; to draw a scheme of patient's follow-up; to compare gastritis, stomach ulcer and duodenal ulcer with other ulcers and diseases with the same symptoms; to demonstrate practical skills during physical examination of the patient, analyzing the clinical and laboratory results.

4. Tasks for self-studying to prepare for the lesson and on the lesson.

4.1. List of main terms, parameters, characteristics that should be learnt by student during preparation for the classes:

Term	Definition
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 lessons:

1. What is gastritis, stomach ulcer and duodenal ulcer?
2. How gastritis, stomach ulcers and duodenal ulcers should be classified?
3. What are the risk factors for gastritis, stomach ulcers and duodenal ulcers?
4. Explain the pathophysiological mechanisms of gastritis, stomach ulcers and duodenal ulcers.
5. Name the diagnostic criteria of gastritis, stomach ulcers and duodenal ulcers.
6. What are the endoscopic characteristics of gastritis, stomach ulcers and duodenal ulcers?
7. Specify the principles and features of gastritis, stomach ulcers and duodenal ulcers pharmacotherapy according to modern recommendations.
8. What lifestyle modifications should be recommended for patients with gastritis, stomach ulcers and duodenal ulcers?
9. What are the approaches to gastritis, stomach ulcers and duodenal ulcers prevention?

4.3. Practical work (tasks), performed on the lesson:

1. Interpret changes in general blood test in case of gastritis, stomach ulcers and duodenal ulcers.
2. Interpret data of biochemical blood tests in case of gastritis, stomach ulcers and duodenal ulcers.
3. Interpret data of EGDS, 24-ph monitoring, HP tests in case of gastritis, stomach ulcers and duodenal ulcers.
4. Perform survey and physical examination of the patient and make preliminary diagnosis.
5. Manage the patient with suspected gastritis, stomach ulcers and duodenal ulcers, prescribe relevant laboratory and instrumental investigations and further treatment.

Topic content:

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.

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.

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 epigastrial 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 - squizing, heaviness, or sharp puncuating (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)
- H2-blockers (described in GERD)

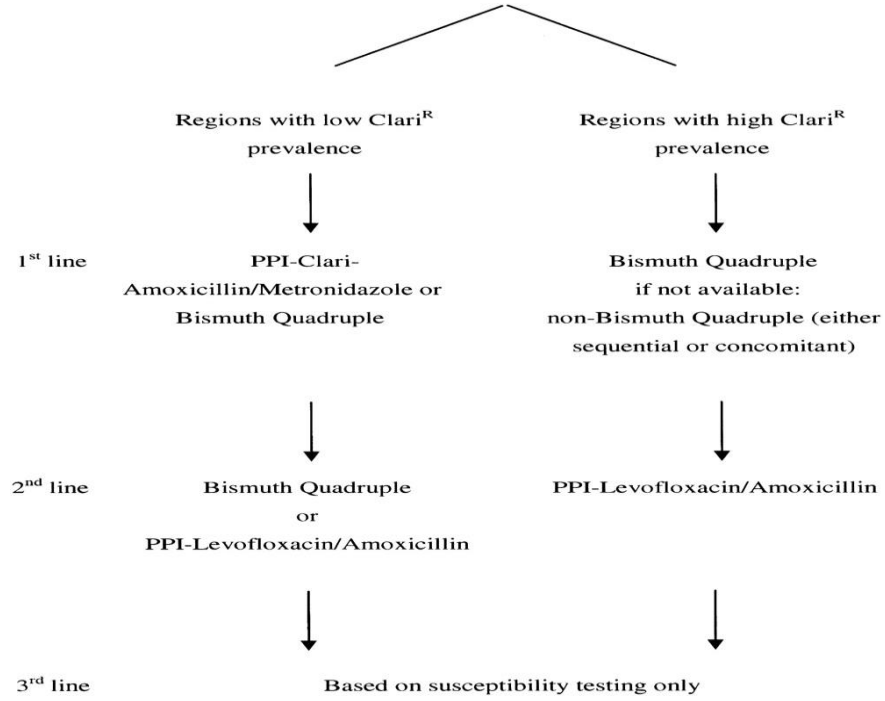
2) anti HP therapy

- 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/rifambutine 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 H2 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 E1 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 B12-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.

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 – epigastrial, 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, gastrine – 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: cyankobalaminum 500mkg/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:

A. Tests and situational tasks for self-control:

1. Etiology of chronic gastritis type A:

A) H. pylori

B) NSAIDs

C) autoimmune

D) chemical damage

2. H2-blockers include:

A) Famotidine

B) Itoprid

C) Pantoprazole

D) Clarithromycine

3. Prokinetics include:

A) Famotidine

B) Itopride

C) Pantoprazole

D) Clarithromycine

4. IPP include:

A) Famotidine

B) Itoprid

C) Pantoprazole

D) Clarithromycine

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

A) long-term NSAIDs intake

B) duodenogastral reflux

C) H. pylori infection

D) stress

6. Triple therapy of H. pylori infection includes:

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

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

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

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

7. Quadruple therapy of H. pylori infection includes:

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

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

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

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

8. What testing of H. pylori may stay positive after HP eradication:

A) histologic examination of gastric mucosa

B) polymerase chain reaction

C) stool antigen test

D) serum antibodies

B. Situational tasks for self-control:

9. 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

10. 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?

Answers: 1-C, 2-A, 3-B, 4-C, 5-C, 6-C, 7-C, 8-D, 9-C, 10-Chronic gastritis. Determination of H. pylori.

Recommended literature

I. Main:

1. Internal Medicine: in 2 books. Book 1. Diseases of the Cardiovascular and Respiratory Systems: textbook / N.M. Seredyuk, I.P. Vakaliuk, R.I. Yatsyshyn et al. Київ, Медицина., 2019. - 664 + 48 кольор. вкл.).
2. Internal medicine: Part 1 (cardiology, rheumatology, haematology): textbook for English-speaking students of higher medical schools / edited by Professor M.A. Stanislavchuk and Professor V.A. Serkova. - Vinnytsia: Nova Knyha, 2019. - 392 p.
3. Медицина за Девідсоном: принципи і практика / Навчальний посібник: пер. 23-го англ. вид.: у3 т. Т.3 С. Ралстона, Я. Пенмана, М. Стрекена, Р. Гобсона; К.: ВСВ «Медицина», 2021. – 642 с.
4. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)/
5. Побічнадіяліків – SideEffectsofMedications: навчальнийпосібнику 2 т. / зазаг.ред. В.М. Бобирьова, М.М. Потяженка. – Вінниця:
6. Cardiovascular diseases. Classification, standards of diagnosis and treatment / Edited by Academician Kovalenko V.M., Prof. Lutaia M.I., Prof. Sirenko Yu.M., Prof. Sychova O.S. – Kyiv. – 2020.
7. Perederii V.H., Tkach S.M. Principles of internal medicine. – Vol.2 / Textbook for students of higher educational institutions. – Vinnytsia: Nova knyha. – 2018.
8. Internal diseases. The textbook based on the principles of evidentiary medicine, 2018.

II. Additional literature:

1. Recommendations of the Association of Cardiologists of Ukraine for the diagnosis and treatment of chronic heart failure / Voronkov L.H. – moderator, working group of the Ukrainian Association of Heart Failure Specialists. – 2017.
2. Respiratory diseases / Ghanei M. - In Tech, 2012. - 242 p.

3. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
4. Principles and practice of interventional pulmonology / Ernst A., Herth F. - Springer, 2012. - 757 p.
5. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
6. Petrov Y. The chief symptoms and syndromes in patients with cardiovascular pathology : The practical handbook fur medical students / Ye. Petrov, Yu. Goldenberg, N. Chekalina; UMSA. - Poltava : TexcepBic, 2010. - 143 .
7. Gastroenterology and Hepatology Board Review: Pearls of Wisdom, Third Edition (Pearls of Wisdom Medicine) by John K. DiBaise (May 11, 2012)
8. Clinical Pulmonology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Oct 30, 2011) - Kindle eBook
9. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Hematology: Clinical Principles and Applications, 4e by Bernadette F. Rodak MS MLS (Feb 18, 2017)
12. Rheumatology, 2-Volume Set: EXPERT CONSULT - ENHANCED ONLINE FEATURES AND PRINT, 5e by Marc C. Hochberg MD MPH, Alan J. Silman MD, Josef S. Smolen MD and Michael E. Weinblatt MD (Oct 19, 2019)
13. Endocrine Pathology: Differential Diagnosis and Molecular Advances by Ricardo V. Lloyd (Nov 5, 2018)
14. Clinical Endocrinology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2017) - Kindle eBook
15. Williams Textbook of Endocrinology: Expert Consult-Online and Print, 12e by Shlomo Melmed, Kenneth S. Polonsky MD, P. Reed MD Larsen and Henry M. Kronenberg MD (May 27, 2016)
16. Electrocardiography, 3e with Student CD (Booth, Electrocardiography for Health Care Personnel) by Kathryn A. Booth (Jan 27, 2017)
17. Echocardiography Review Guide: Companion to the Textbook of Clinical Echocardiography: Expert Consult: Online and Print, 2e (Expert Consult Title: Online + Print) by Catherine M. Otto (Mar 7, 2017).