

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

"Approved"
at the meeting of internal
medicine №1 department
Head of 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>	Gastro-esophageal reflux disease. Gastric dyspepsia.
<i>Course</i>	IV
<i>Faculty</i>	of foreign students training

Poltava 2016.

1. Relevance of the topic: **Gastro-esophageal reflux disease (GERD)** is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury. **Functional dyspepsia (FD)** is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial fullness, and early satiation that are unexplained after a routine clinical evaluation.

2. The main goal: To be able to assess the typical clinical picture of GERD and FD, to determine tactics of treatment and prophylaxis.

Specific goals:

- To select the information indicating the presence of GERD and FD in a patient from the data history;
- To create a scheme of diagnostic search;
- To identify the signs of GERD and FD 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 GERD and FD 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 esophageal tissue in GERD	
Radiology	Radiological changes at GERD	Analyze the radiological picture of the chest cavity and abdominal cavity
Propaedeutic therapy	Symptomatology of GERD and FD 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 ₂ -blockers, antacids, prokinetics, antibiotics, antidiarrheal drugs	Prescribe the drugs of these groups
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4. Do the tasks for independent work during preparation for classes.

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

Term	Definition
Gastro-esophageal reflux disease	is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury.
Functional dyspepsia	is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial fullness, and early satiation that are unexplained after a routine clinical evaluation.
Inhibitors of proton pump (IPP)	group of drugs which suppress the production of stomach acid and work by inhibiting the molecule in the stomach glands that is responsible for acid secretion (the gastric acid pump).
H ₂ -blockers	group of which that interfere with acid production by blocking or antagonizing the actions of histamine.
Prokinetics	group of drugs which enhances gastrointestinal motility by increasing the frequency of contractions or making them stronger.

4.2. Theoretical questions for the lesson:

1. Give the definitions of GERD and FD.
2. Specify the risk factors for GERD and FD.
3. The pathophysiological mechanisms of GERD and FD.
4. Diagnostic criteria of GERD and FD.
5. What are the endoscopic characteristics of GERD and its stages?
6. Modern classification of GERD and FD.
7. Specify the principles and features of GERD and FD pharmacotherapy according to modern recommendations.
8. What lifestyle modifications should be recommended for patients with GERD and FD?

4.3. Practical tasks that are performed in class:

1. IPP include:
 - 1) Famotidine
 - 2) Itoprid

3) Pantoprazole

4) Clarithromycine

2. H2-blockers include:

1) Famotidine

2) Itoprid

3) Pantoprazole

4) Clarithromycine

3. Prokinetics include:

1) Famotidine

2) Itopride

3) Pantoprazole

4) Clarithromycine

4. Standart dosage of Omeprazole is:

1) 40 mg

2) 60 mg

3) 20 mg

4) 20-40 mg

5. Standart dosage of Pantoprazole is:

1) 20 mg

2) 60 mg

3) 40 mg

4) 20-40 mg

6. Standart dosage of Rabeprazole is:

1) 40 mg

2) 60 mg

3) 20 mg

4) 20-40 mg

7. Standart dosage of Esomeprazole is:

1) 20 mg

2) 60 mg

3) 40 mg

4) 20-40 mg

8. Standart dosage of Lansoprazole is:

1) 20 mg

2) 60 mg

3) 40 mg

4) 30 mg

9. Atypical symptom of GERD is:

1) chronic cough

2) heartburn

3) chest pain

4) nausea

10. What are the types of FD?

1) typical, atypical

2) postprandial distress syndrome, epigastric pain syndrome

3) pain syndrome, dyspeptic syndrome

4) A, B, C and D types

Topic Content

GASTRO-ESOPHAGEAL REFLUX DISEASE

Definition. **Gastro-esophageal reflux disease (GERD)** is a chronic and relapsing disease that results from the retrograde flow of gastric contents into the esophagus (gastro-esophageal reflux), oropharynx, and/or respiratory tract and causes troublesome symptoms or complications and/or mucosal injury. GERD refers to the abnormal, not physiologic reflux.

Classification. Montreal Classification of GERD (2006): erosive reflux disease (with esophagitis), nonerosive reflux disease (is defined by the presence of troublesome reflux-associated symptoms and absence of mucosal breaks on endoscopy), Barret's esophagus (with long segment or short segment). NB: Erythema is not reliable finding for diagnosis of reflux esophagitis!

LA Classification of Esophagitis by grade (1999):

Grade A	One (or more) mucosal break no longer than 5 mm that does not extend between the tops of two mucosal folds
Grade B	One (or more) mucosal break more than 5 mm long that does not extend between the tops of two mucosal folds
Grade C	One (or more) mucosal break that is continuous between the tops of two or more mucosal folds but which involve less than 75% of the circumference
Grade D	One (or more) mucosal break which involves at least 75% of the esophageal circumference

By symptoms: typical and atypical.

Epidemiology. It is estimated that GERD, defined as at least weekly heartburn or acid regurgitation, has a prevalence ranging from 10 to 20% in the Western world and less than 5% in Asia. The prevalence also tends to be higher in North America than Europe and higher in northern Europe than in southern Europe. The prevalence of heartburn is the highest in USA and Western Europe countries (20%) and the lowest is in China (2,5%).

Risk and etiological factors: obesity, pregnancy, hiatal hernia, tobacco abuse, alcohol consumption, overeating, taking medicines that decrease lower esophageal sphincter contractility (nitrates, calcium channel blockers, beta adrenergic agonists, papaverine, no-spa, anticholinergics, theophylline, morphine, meperidine, diazepam, and barbiturates etc.). A genetic component may also play a role in GERD exacerbation. Character of food can have an influence too, e.g. peppermint, coffee, fatty meal etc.

Pathogenesis. The esophagus is protected from the harmful effects of refluxed gastric contents by the antireflux barrier at the gastroesophageal junction, by esophageal clearance mechanisms, and by epithelial defensive factors. The antireflux barrier consists of the lower esophageal sphincter, crural diaphragm, phrenoesophageal ligament, and the angle of His, which causes an oblique entrance of the esophagus into the stomach. The attachment of the lower esophageal sphincter to the crural diaphragm results in increased pressure during inspiration and when intra-abdominal pressure increases. Disruption of normal defense mechanisms leads to

pathologic amounts of reflux. Reflux of gastric contents from the stomach into the esophagus occurs in healthy individuals, but refluxed gastric contents are normally cleared in a two-step process: volume clearance by peristaltic function and neutralization of small amounts of residual acid by weakly alkaline swallowed saliva. In normal healthy individuals, physiologic reflux occurs primarily when the lower esophageal sphincter transiently relaxes in the absence of a swallow because of a vagally mediated reflex that is stimulated by gastric distention. Physiologic reflux is postprandial, short, asymptomatic and does not cause mucosal injuries of esophagus.

In GERD patients, transient relaxation of the lower esophageal sphincter or a low resting lower esophageal sphincter pressure can result in regurgitation, especially when intra-abdominal pressure is increased. A hiatal hernia, which results in spatial separation between the augmenting effects of the crural diaphragm and the lower esophageal sphincter, predisposes to reflux events by widening the opening of the gastroesophageal junction and decreasing the pressure of the lower esophageal sphincter. The result is an increased exposure of the esophagus to acid, with increased reflux events during transient physiologic relaxation of the lower esophageal sphincter. Hernias also act as a reservoir for gastric contents when normal esophageal clearance mechanisms result in trapping of fluids in the hernia sac; these contents can reflux into the esophagus when the lower esophageal sphincter relaxes during subsequent swallowing.

Obesity results in an increase in intragastric pressure, which increases the gastroesophageal pressure gradient and the frequency of transient lower esophageal sphincter relaxation, thereby predisposing gastric contents to migrate into the esophagus. In addition, obesity enhances the spatial separation of the crural diaphragm and the lower esophageal sphincter, thereby predisposing obese individuals to a hiatal hernia. The normal defense mechanisms based on peristalsis and saliva can also be impaired. Peristaltic dysfunction is associated with an increasing severity of esophagitis, and ineffective peristaltic clearance may occur when the amplitude of esophageal contractions is less than 30 mm Hg. Saliva production may be impaired by a variety of mechanisms, such as smoking and Sjögren's syndrome.

The esophageal mucosa contains several lines of defense. A pre-epithelial barrier is composed of a small unstirred water layer combined with bicarbonate from swallowed saliva and from the secretions of submucosal glands. A second epithelial defense is composed of cell membranes and tight intercellular junctions, cellular and intercellular buffers, and cell membrane ion transporters. The postepithelial line of defense is composed of the blood supply to the esophagus. Acid and acidified pepsin in the refluxate are the key factors that damage the intercellular junctions, increase intracellular permeability, and dilate intercellular spaces. If sufficient quantities of refluxate diffuse into the intercellular spaces, cellular damage may occur. Signs and symptoms of GERD occur when defective epithelium comes into contact with refluxed acid, pepsin, or other noxious gastric contents.

In addition to the direct noxious effects of refluxed acid, pepsin, and bile, refluxed gastric juice stimulates esophageal epithelial cells to secrete chemokines that attract inflammatory cells into the esophagus, thereby damaging the esophageal mucosa.

Clinical features: The classic symptoms of GERD are heartburn and acid regurgitation; atypical symptoms include chest pain, dysphagia, and odynophagia. Extraesophageal manifestations of reflux disease can include cough, laryngitis, asthma, and dental erosions, but these symptoms can be reliably attributed to reflux only if they are accompanied by classic signs and symptoms of reflux disease. Other proposed associations that are not clearly established include pharyngitis, sinusitis, otitis media, and idiopathic pulmonary fibrosis. When excessive gastric contents overwhelm the mucosal protective factors in the esophagus, esophagitis may be

manifest as erosions or ulceration of the esophagus and may also lead to fibrosis with stricturing, columnar metaplasia (Barrett's esophagus) or esophageal adenocarcinoma. However, approximately two thirds of individuals with reflux symptoms have no evidence of esophageal damage by endoscopy.

Typical symptoms:

- 1) Heartburn
 - Retrosternal burning sensation
 - Most commonly post-prandial, nocturnal
 - Fatty foods, spicy foods, acidic foods
 - Relieved with antacids, water, milk
 - Worsened with recumbency
- 2) Acid Regurgitation
 - Perception of gastric content reflux in the mouth or hypopharynx
 - Taste: bitter, acidic

Atypical symptoms:

- 1) Atypical
 - Dysphagia, odynophagia
 - Nausea
 - Chest pain
 - Dyspepsia (non-severe upper abdominal discomfort)
 - Epigastric fullness, bloating
 - Frequent belching
 - Heartburn
- 2) Extraesophageal
 - Chronic cough, bronchospasm, pneumonia, fibrosis
 - Cardial pain, arrhythmia
 - Hoarseness, laryngitis, pharyngitis, globus sensation, vocal cord dysfunction
 - Stomatitis, dental erosions

RED FLAGS:

- Dysphagia (immediately assess for Barrett's Esophagus)
- Odynophagia (Assess for Esophageal Ulcer)
- Nausea/vomiting
- Melena
- Weight loss, anorexia
- Extended duration of symptoms with no response to PPIs
- Family history of peptic ulcer disease
- Symptom onset in the age more than 45 years, especially in males (high risk)
- High body temperature
- CBC changes (anemia, leukocytosis, etc.)

Diagnosis:

- History (patients' complaints, anamnesis)
- Physical examination
- Screening tests (CBC)
- Empiric trial (IPP, Alginates)
- Additional: endoscopy with biopsy, chromoendoscopy, manometry, pH testing, impedance.

- If necessary: bronchography, ultrasound diagnostic, Helicobacter pillory testing, ECG.

When GERD presents with typical signs and symptoms, such as heartburn or acid regurgitation, that are responsive to antisecretory therapy, no diagnostic evaluation is warranted.

Diagnostic endoscopy is warranted in individuals who fail to respond to therapy or have alarm symptoms or signs such as dysphagia, weight loss, anemia, gastrointestinal bleeding, or persistent heartburn. Endoscopy permits the detection of erosive esophagitis and complications such as a peptic stricture and Barrett's esophagus; mucosal biopsy, which is crucial in these settings, also excludes conditions that can mimic GERD, such as eosinophilic esophagitis. However, most patients have no mucosal damage seen on endoscopy, regardless of whether they are on or off antisecretory therapy.

Esophageal manometry is useful to exclude achalasia in patients with suggestive symptoms. Esophageal reflux testing by 24-hour transnasal pH monitoring, by 48-hour devices attached to the esophageal lumen, or by 24-hour combined impedance and pH monitoring, may be performed while patients are not on therapy to detect pathologic acid and nonacid reflux as well to correlate reflux events with atypical symptoms, especially in patients with normal endoscopies.

Barium radiography has no role in the diagnostic evaluation of patients with reflux disease.

Complications: esophageal strictures, Barrett's esophagus, ulcer of esophagus, bleeding, laryngitis, pharyngitis, sinusitis, adenocarcinoma, interstitial fibrosis, dental erosions (dental enamel loss).

Alternative diagnosis in GERD (Differential Diagnosis):

- Coronary artery disease (Ischemic heart disease)
- Gallstones
- Gastric/esophageal cancer
- Peptic ulcer disease
- Esophageal motility disorders
- Pill induced esophagitis
- Eosinophilic esophagitis
- Fungal or viral esophagitis
- Peptic stricture
- Metaplastic disease (Barrett's)
- Dysplastic disease (adenocarcinoma)

Treatment. General Measures:

- Avoidance of foods or beverages that may provoke symptoms, such as alcohol, coffee, spicy foods, fatty food, chocolate etc. and late meals (less than 2-3 hours before bedtime)
- Elevation of the head of the bed to 30 degrees for patients with nocturnal regurgitation or heartburn
- Weight loss should be part of any treatment program for obese patients
- Tobacco cessation.

Medications. Inhibition of gastric acid secretion is the cornerstone of the acute treatment of GERD, and proton pump inhibitors (PPIs) are superior to histamine (H₂)-receptor antagonists for both the healing of esophagitis and the control of symptoms. Once-daily standart dosage of PPIs 30-60 minutes prior to meal for 4-8 weeks (nonerosive form, Grade A, B) or 8-12 weeks (Grade C, D) is adequate. High dose (twice daily) is usually used for severe or refractory symptoms. Given the chronicity of reflux symptoms, long-term

maintenance therapy with PPIs is typically required, with dosing titrated to the lowest dose necessary to control symptoms. Although PPIs are superior to H₂-receptor antagonists for long-term maintenance therapy as well as for short-term relief, H₂-receptor antagonists are useful in patients who are intolerant of PPIs, and can be used at bedtime to supplement PPIs in patients who have persistent symptoms.

PPIs suppress the production of stomach acid and work by inhibiting the molecule in the stomach glands that is responsible for acid secretion (the gastric acid pump). Once symptoms are controlled, people should receive the lowest effective dose of PPIs.

PPIs (standart dosage):

- Esomeprazole 40mg
- Lansoprazole 30mg
- Omeprazole 20mg
- Pantoprazole 40mg
- Rabeprazole 20mg
- Dexlansoprazole (long-acting form) 60mg

H₂-blockers interfere with acid production by blocking or antagonizing the actions of histamine. Histamine encourages acid secretion in the stomach. Famotidine is the most potent H₂ blocker.

Also antacid medications (e.g. Maalox) can be used. Antacids neutralize acids in the stomach, and are the drugs of choice for mild GERD symptoms. They may also stimulate the defensive systems in the stomach by increasing bicarbonate and mucus secretion. They should be prescribed 1-1,5 hours after meal 3-4 times a day for 14 days. The different brands all rely on various combinations of 3 basic ingredients: magnesium, calcium, or aluminum.

Magnesium salts are available in the form of magnesium carbonate, magnesium trisilicate, and most commonly, magnesium hydroxide (Milk of Magnesia). The major side effect of magnesium salts is diarrhea. Magnesium salts offered in combination products with aluminum (Maalox) balance the side effects of diarrhea and constipation.

Calcium carbonate is a potent and rapid-acting antacid. It can cause constipation. There have been rare cases of elevated levels of calcium in the blood (hypercalcemia) in people taking large doses of calcium carbonate for long periods of time. This condition can lead to kidney failure and is very dangerous. None of the other antacids has this potential side effect.

Aluminum salts (Almagel) are also available. The most common side effect of antacids containing aluminum salts is constipation. People who take large amounts of antacids that contain aluminum may also be at risk for calcium loss, which can lead to osteoporosis.

Prokinetic drugs help the stomach empty its contents more quickly and strengthen the esophageal sphincter. These are considered second-line access drugs due to side effects. The most widespread prokinetics are: Domperidone 10mg 3 times a day, Itoprid 50mg 3 times a day.

Antireflux surgery is an option for patients who have documented esophagitis and who are intolerant of PPIs or unresponsive to them. However, surgery has a number of serious complications that may affect quality of life, including dysphagia, vagal nerve injury, gas bloat syndrome, and diarrhea. There are inadequate data to support any of the many proposed endoscopic approaches to GERD at present.

FUNCTIONAL DYSPEPSIA

Definition. Functional dyspepsia (FD) is a medical condition that is characterized by one or more of the following symptoms: epigastric pain, epigastric burning, postprandial

fullness, and early satiation that are unexplained after a routine clinical evaluation. Abdominal bloating and nausea also may be experienced, but they are less specific and are not considered cardinal symptoms of functional dyspepsia. Patients, who were not observed can be made a preliminary diagnosis of *uninvestigated dyspepsia*. Further they will be divided into 2 groups: those with an organic, systemic, or metabolic cause for the symptoms that can be identified by traditional diagnostic procedures where, if the disease improves or is eliminated, symptoms also improve or resolve (eg, peptic ulcer disease, malignancy, pancreaticobiliary disease, endocrine disorders, or medication use) and is described by the term *secondary dyspepsia*; those in whom no identifiable explanation for the symptoms can be identified by traditional diagnostic procedures that are exemplified under the “umbrella” term *functional dyspepsia*.

Classification. In the Rome IV criteria (2016), symptoms have been divided into postprandial distress syndrome and epigastric pain syndrome. The clinical utility of these subgroups is controversial because there is considerable overlap between them.

Epidemiology: FD is a common disorder, with an estimated prevalence of 10%-30% worldwide. Approximately 1 of 2 individuals with functional dyspepsia seeks health care for symptoms at some time in his or her life.

Risk factors: according to recent researches smoking is only marginally associated with dyspepsia and alcohol and coffee are not. Other data are controversial.

Pathogenesis. The pathobiology of functional dyspepsia is complex and multifactorial and not fully understood.

Gastroduodenal motor and sensory dysfunction, as well as impaired mucosal integrity, low-grade immune activation, and dysregulation of the gut-brain axis have all been implicated. Both central and peripheral mechanisms have been proposed. Although enhanced perception of gastric stimuli may be a key central mechanism, the roles of gastric acid, acute and chronic gastric mucosal infections, and gastroduodenal dysmotility remain to be determined. Rome IV proposed next factors, that play a role in FD pathogenesis:

- delayed gastric emptying
- impaired gastric accommodation
- gastric and duodenal hypersensitivity to distention, acid, and other intraluminal stimuli
- *Helicobacter pylori* infection
- psychosocial factors.

The potential relation between these factors and dyspeptic symptoms remains unclear.

Diagnosis. Clinical examination. Dyspepsia can be suspected to be functional based on a clinical history consistent and the absence of alarm features, treatment of overlapping gastroesophageal reflux disease and *H. pylori*. The presence of anxiety, in particular symptom-related anxiety and comorbid IBS, increases the likelihood of functional dyspepsia.

The physical examination is generally normal, although epigastric tenderness may be present. In contrast to gastroparesis, a succussion splash is typically absent. Confirmation of the functional dyspepsia diagnosis requires a normal upper endoscopic examination. Evaluation for *H. pylori* should be performed by stool antigen, urea breath test, or gastric biopsy. If present, the infection should be eradicated, and then symptoms should be reassessed. Additional methods of laboratory and instrumental examination, besides upper endoscopy and HP testing, are: CBC, biochemical blood analysis, fecal occult blood test, ultrasound diagnostic of abdominal cavity, computer tomography.

Rome IV (2016) diagnostic criteria for functional dyspepsia (criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis):

1. One or more of the following:

Bothersome postprandial fullness/early satiation/epigastric pain/epigastric burning

2. No evidence of structural disease

(including at upper endoscopy) that is likely to explain the symptoms.

Rome IV (2016) diagnostic criteria for subgroups of patients with functional dyspepsia:

– *Postprandial Distress Syndrome*

Must include one or both of the following at least 3 days per week:

1. Bothersome postprandial fullness (severe enough to impact on usual activities)

2. Bothersome early satiation (severe enough not to finish a regular-size meal)

AND No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

Supportive remarks: postprandial epigastric pain or burning, epigastric bloating, excessive belching, and nausea can also be present; vomiting warrants consideration of another disorder; heartburn is not a dyspeptic symptom but may often coexist; symptoms that are relieved by evacuation of feces or gas should generally not be considered as part of dyspepsia.

– *Epigastric Pain Syndrome*

Must include one or both of the following at least 1 day per week:

1. Bothersome epigastric pain (severe enough to impact on usual activities)

2. Bothersome epigastric burning (severe enough to impact on usual activities) AND No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

Supportive remarks: pain may be induced by ingestion of a meal, relieved by ingestion of a meal, or may occur while fasting; postprandial epigastric bloating, belching, and nausea can also be present; persistent vomiting likely suggests another disorder; heartburn is not a dyspeptic symptom but may often coexist; the pain does not fulfill biliary pain criteria; symptoms that are relieved by evacuation of feces or gas generally should not be considered as part of dyspepsia.

Nonsteroidal anti-inflammatory medications, dietary supplements, and other prescription or over-the-counter medications can trigger dyspeptic symptoms. The patient's medication list should be reviewed, and nonessential treatments should be discontinued. A psychosocial history may reveal underlying stressors that contribute to symptoms.

Differential Diagnosis. Common organic causes of dyspepsia include peptic ulcer disease and gastroesophageal reflux disease. Delayed gastric emptying is present in a small number of patients with functional dyspepsia but is characteristic and more pronounced in patients with diabetic or idiopathic gastroparesis. Vomiting of undigested food is characteristic of these forms of gastroparesis, but not of dyspepsia. Gastric and esophageal cancers may also present with symptoms of dyspepsia but are much less common. Pancreaticobiliary disorders (including sphincter of Oddi dysfunction, chronic pancreatitis, or pancreatic cancer) also occasionally mimic dyspepsia.

Treatment. Reassurance, education, lifestyle, and dietary recommendations (more frequent, smaller meals and avoiding meals with high fat content) are frequently recommended to FD patients. Avoidance of nonsteroidal anti-inflammatory drugs, coffee, alcohol, and smoking is commonly recommended.

Eradication of *H. pylori* is recommended in patients with chronic dyspepsia and positive HP tests (according to Maastricht IV).

Postprandial distress syndrome should be treated with prokinetics (cisapride and domperidone). Itopride is a novel prokinetic agent that works by antagonizing dopamine D2-receptors and inhibiting acetylcholinesterase, and has been shown to improve postprandial fullness and early satiety with a low rate of adverse reactions. Acotiamide is a novel compound with fundusrelaxing and gastroprokinetic properties, based on a procholinergetic effect that improves dyspeptic symptoms over placebo.

In relieving epigastric pain syndrome proton pump inhibitors (PPIs) and Histamine₂-blockers (H₂-blockers) are recommended to use (PPIs and H₂-blockers were described in GERD chapter).

Psychotropic drugs, especially antidepressants, are often used as second-line drugs in functional gastrointestinal disorders. Often used levosulpiride, which also bears prokinetic properties; and often recruited in psychiatric rather than gastroenterological settings.

Psychological therapies are advocated as rescue therapy for FD symptoms that are severe and not responding to pharmacotherapy. Available controlled trials suggested clinical benefit, but lacked convincing evidence because of small sample sizes and poorly matched treatment groups.

Materials for self-control:

Situation tasks:

1. A 65-year-old patient complains of heartburn, sour eructation, burning, compressing retrosternal pain and pain along the esophagus rising during forward bending of body. The patient hasn't been examined, takes Almagel on his own initiative, claims to feel better after its taking. What is the preliminary diagnosis? What additional tests are necessary?
2. A patient of 37 years old complains of pain in epigastrium, more often during the sleep, or after emotional overload, heartburn. These symptoms amplify after eating. Objectively: a belly is painless on palpation, the liver and spleen are not enlarged. What is the preliminary diagnosis? What additional tests are necessary for the patient? The treatment plan?

Tests:

1. A 38 y.o. man complains of having occasional problems with swallowing of both hard and fluid food for many months. Sometimes he feels intense pain behind his breast bone, especially after hot drinks. There are asphyxia onsets at night. He has not put off weight. Objectively: his general condition is satisfactory, skin is of usual colour. Examination revealed no changes of gastrointestinal tract. X-ray picture of thorax organs presents esophagus dilatation with level of fluid in it. What is the preliminary diagnosis?

- A. Myasthenia
- B. Esophagus achalasia
- C. Cancer of esophagus

- D. Esophagus candidosis
- E. Gastroesophageal reflux

2. A patient suffering from gastroesophageal reflux has taken from time to time a certain drug that "reduces acidity" over 5 years. This drug was recommended by a pharmacist. The following side effects are observed: osteoporosis, muscle asthenia, indisposition. What drug has such following effects?

- A. Inhibitor of proton pump
- B. Aluminium-bearing antacid
- C. H₂-blocker
- D. Metoclopramide
- E. Gastrozepin

3. A 28-year-old male patient complains of regurgitation, cough and heartburn that occurs every day after a meal, when bending forward or lying down. These problems have been observed for 4 years. Objective status and laboratory values are normal. Upper endoscopy revealed esophagitis. What is the leading factor in the development of this disease?

- A. Helicobacter pylori infection
- B. Hypersecretion of hydrochloric acid
- C. Duodeno-gastric reflux
- D. Hypergastrinemia
- E. Failure of the inferior esophageal sphincter

4. A 49-year-old male patient complains of retrosternal pain, heartburn, weight loss of 8kg over the last year, constipation, weakness. The patient has been a smoker for 20 years, and has a 10-year history of Gastroesophageal reflux disease. The patient is asthenic, has dry skin. EGD revealed an ulcer in the lower third of the esophagus and esophageal stricture accompanied by edema, hyperemia and multiple erosions of the mucosa. What study is required for more accurate diagnosis?

- A. Fecal occult blood test
- B. X-ray examination of the esophagus
- C. Respiratory test for Helicobacter pylori
- D. pH-monitoring of the esophagus and the stomach
- E. Biopsy of the esophageal mucosa

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. Functional dyspepsia
- B. Duodenal ulcer
- C. Type A chronic gastritis
- 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. A patient complains of heartburn which gets stronger after overeating. Upper endoscopy shows ulcerative esophagitis. What group of medicines is the first line of therapy?

- A. Inhibitors of proton pump
- B. Prokinetics
- C. Probiotics
- D. Antibiotics
- E. Alginates

Correct answers for the situation tasks:

1. GERD. IPP test, upper endoscopy, pH-monitoring.
2. Functional dyspepsia, epigastric pain syndrome. US examination, upper endoscopy. Treatment e.g. Lansoprazole 30 mg 40 minutes before the breakfast, Famotidine 40 mg 30 minutes before the supper.

The answers for the tests:

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

Recommended literature:

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