Theme: Gastric Dyspepsia. Chronic gastritis and duodenitis.

**Gastric (functional) dyspepsia as syndrom**

**Dyspepsia** (indigestion) - a general term or syndrome that combines disorders of intracavitary digestion, resulting from disorders of secretory and enzyme functions, or, in other words, with discrepancies between needs of the body and abilities of the cavitary digestion.

Depending on the lesion of a digestive organ, dyspepsia is distinguished to:

- Stomach-induced or Gastric;
- Intestinal;
- Hepatic (hepatobiliary);
- Pancreatic.

Dyspepsia is developing on the background of organic diseases (secondary dyspepsia of organic origin) or without organic changes of the digestive system (primary or functional dyspepsia).

There is also a term "unexamined dyspepsia" – a previous working syndromic diagnosis at the initial examination of a patient, which is diagnosed only on the basis of complaints, medical history and physical study, before additional laboratory and instrumental methods of research.

Dyspepsia that has no organic base, is called **functional** (primary, non-ulcerative, non-specific, inorganic, idiopathic, irritable stomach syndrome).

Functional dyspepsia appears as a separate disease in the International Statistical Classification of Diseases of last (10th) revision.

**Gastric (functional) dyspepsia as nosological diagnosis**

**Functional (gastric) dyspepsia (FD)** – functional disease of a stomach (of a gastroduodenal area), that signs with typical complaints and is connected with impaired motility, tone and gastric secretion while absence of organic changes in a stomach and in a bulb of duodenum, which could explain these complaints.

Comments on the definition:

- diagnosis of PD is proposed when relevant complaints are at least 3 months in the last six months with a frequency at least 1 time per week;
- main complaints at PD - pain and/or discomfort in epigastric, eructation and nausea;

**Etiological factors**
-nervous regulation disturbance that lead to motor-evacuation disorders in gastroduodenal area

-presence of active Helicobacter that promotes hypersecretion of HCl.

Among provoking factors also are alimentary disturbance, bad habits, taking some kinds of medicaments, stresses.

The main patho-genetical mechanism of FD development is motility, stomach tone and duodenum disturbance.

**Classification of FD**

1. **By clinical peculiarities**

   Roman criteria II (1999) defined ulcerative (pain in epigastric on an empty stomach), dyskinetic (fast satiation, nausea), refluxed (heartburn) and nonspecific (combination of different features) variants of FD.

   Roman criteria III (2005) define two basic variants of the disease:

   **Epigastric pain syndrome**

   **Postprandial distress syndrome**

2. **By the depth of examination**, gastric dyspepsia is divided into:

   -Unexamined dyspepsia (before instrumental and laboratory methods of additional examination).

   -Pre-examined dyspepsia: primary functional dyspepsia or secondary organic (with specific organic pathology).

**Clinical signs of FD** include at least one of the following local complaints:

-Epigastric aching or burning pain on an empty stomach or after nervous stress, decreasing after meal, rest, sedatives, antacids. Night pain is not typical.

-Heaviness, fever, epigastric discomfort after eating, nervous stress, rapid saturation, food eructation, nausea, rarely - vomiting, anorexia.

Local gastric complaints at PD are often combined with signs typical for other digestive organs - esophagus, intestines, etc. (heartburn, bloating, stool disturbance, etc.).

The disease has chronic intermitting nature. Individuals up to 40 years, women, vegeto-labile persons are dominative among patients with PD. There is a clear link between the occurrence of complaints and psycho-emotional factors.

Often, general symptoms of vegetative lability are detected:

asthenia, irritability, fatigue, depressed mood, insomnia, tachycardia, sweating, dry mouth, changing to hypersalivation, etc.

**Diagnostic criteria of FD**
- Typical complaints - now and in medical history, epigastric pain during palpation.
- Sufficiently long period of onset of symptoms - 3 months or more in the last six months.
- Laboratory methods of study: possible detection of Hp, general clinical analysis - normal.
- Instrumental methods of study:
  - fluoroscopy, electrogastrography, scintigraphy - show signs of gastroparesis and other motor-tonus disorders;
  - pH meter - can detect acidity increase, can not find signs of gastroesophageal reflux;
  - esophagogastroduodenoscopy - without organic changes; can not detect organic pathological changes during the ultrasound, ECG, etc..

Consultation with a neurologist, neuropsychiatrist can reveal the presence of a vegetative dysfunction or certain psychoorganic changes.

**Treatment of FD**

**Treatment includes:**

- General measures.
- Drug therapy.
- Neuropsychological and physical rehabilitation.

Stationary treatment is usually not indicated.

**General measures:**

- Removal of nerve stress;
- Adequate time for rest and sleep, fresh air and exercise
- Maintaining of moderate physical activity;
- Eliminating of alcohol, smoking;
- Rational nutrition - 4-5 meals a day, small portions, eat slowly; Avoid strong meat brothes, hot spices, caffeinated and carbonated drinks, fatty meat.

**Drug therapy:**

Drugs of choice - antisecretory drugs (proton pump inhibitors, H2-histamine-blockers, antacids) and motility and tone regulators - prokinetics (metoclopramide, domperidone, itopryd or mosapryd).

Apply at:

- epigastric pain syndrome - a combination of PPI (eg. 20 mg of rabeprazole 2 times a day) or famotidine (20 mg 2 times a day) with sedatives (sedasen, novopassyt, xanax, etc.) or antidepressants (sulpiride, amitriptyline, fluoxetine, and others.)
-postprandial distress syndrome - a combination of prokinetics (10 mg of domperidone 3 times a day) and antidepressants (eg. 50 mg of fluoxetine in the evening);

-active Helicobacter – triple anti-helicobacter therapy during 7 days, after that - monthly course of antacids (eg. 1-2 doses of Maalox 3-4 times a day) or famotidine (20 mg before sleep).

**Neuropsychological and physical rehabilitation:**

- psychotherapy, autogenic training;

- apparatus physiotherapy of soothing effect: electrophoresis with bromine, darsonvalization of head, neck area galvanization etc.

- calming massage of head and back;

- water procedures (therapeutic shower, dousing, soothing baths, swimming).

**Chronic gastritis and duodenitis.**

**Chronic gastritis (CG)** - a chronic inflammatory -dysregenerative process in a stomach mucous membrane, accompanied by epithelial cell regeneration disturbance up to its atrophy and related functional disorders of the body.

**Chronic duodenitis** - a chronic process of similar character in duodenum. A process in duodenum bulb has common mechanisms of development with gastritis and is called **bulbit**.

**Etiological and provoking factors of chronic gastroduodenitis:**

1. Exogenous:
   - violation of eating pattern;
   - smoking, alcohol;
   - remedies (NSAID etc.), other toxic agents;

2. Endogenous:
   - Helicobacter pylori and other infections;
   - genetic predisposition (mostly at CGA);
   - bile reflux;
   - other diseases: diabetes, Crohn's disease, allergies, uremia, thyrotoxicosis, etc.
   - poor condition of masticatory apparatus.
Clinical classification of CG:

By genesis:
1. Autoimmune (type A);
2. Associated with Helicobacter pylori (type B);
3. Chemical CG:
   - Reflux- gastritis (with bile reflux);
   - Drug-induced CG caused by taking of nonsteroid anti-inflammatory drugs (NSAID), other drugs and toxic agents;
4. CG of mixed origin (different combinations of 1 - 3);
5. CG of different genesis (special forms of CG): eosinophilic (allergic), lymphocytic, granulomatous, hypertrophic, hyperplastic (Ménétrièr's disease), associated with other infectious causative agents (bacteria, viruses, fungi, parasites).

By process localization:
1. antral;
2. fundus (fundic);
3. pangastritis (diffuse CG).

By morphological changes:
   a) assessment of presence and intensity of changes in fibrogastroduodenoscopy: state of folds, inflammation character, atrophy, erosions, reflux, etc.

   b) assessment of presence and intensity of changes in biopsy materials (inflammatory changes, atrophy, metaplasia, presence and activity of Hp).

By clinical course:
1. For non-atrophic CG with preserved (increased) secretion:
   a) exacerbation;
   b) remission (unstable remission).

2. For CG with atrophy and secretory insufficiency:
   a) compensated;
   b) subcompensated;
   c) decompensated.

By functional changes (primarily from the secretory function of a stomach):
1. CG with preserved (normal or increased) secretion;
2. CG with secretory insufficiency (moderate or evident).

**Characteristics of main types of CG (by genesis)**

<table>
<thead>
<tr>
<th>Signs</th>
<th>Gastritis B (Hp-associated)</th>
<th>Gastritis A (autoimmune)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevailing localization</td>
<td>Antrum</td>
<td>Fundus, body</td>
</tr>
<tr>
<td>Infectious factor</td>
<td>Yes</td>
<td>Unknown</td>
</tr>
<tr>
<td>Inflammation of mucous coat</td>
<td>Active</td>
<td>Not expressed</td>
</tr>
<tr>
<td>Atrophy</td>
<td>In later period</td>
<td>Early development</td>
</tr>
<tr>
<td>Erosions</td>
<td>Often</td>
<td>Seldom</td>
</tr>
<tr>
<td>Combination with ulcer</td>
<td>Typical</td>
<td>Very seldom</td>
</tr>
<tr>
<td>Secretion level</td>
<td>Depends on prescription of illness, initially-preserved</td>
<td>Expressed hypoacidity</td>
</tr>
<tr>
<td>Hypergastrinemia</td>
<td>No</td>
<td>Typical</td>
</tr>
<tr>
<td>B12 - deficiency anemia</td>
<td>No</td>
<td>Often</td>
</tr>
<tr>
<td>State of pyloric sphincter</td>
<td>Mostly hypertone</td>
<td>Mostly deficiency</td>
</tr>
<tr>
<td>Antibodies to Hp</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Antibodies to parietal cells</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Antibodies to intrinsic factor of Castle</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

**Diagnostic criteria of chronic gastritis, duodenitis:**

- clinical data, medical history (pain, dyspepsia now or/and in history);

- signs of illness in fibrogastroduodenoscopy with multiple biopsy and examination of biopsy material;

- X-ray study of stomach and duodenum - specification of fibrogastroduodenoscopy data and determining the state of motor - evacuation function;

- at suspicion of Hp-associated gastritis, duodenitis - methods of diagnostics of presence and activity of Hp (rapid urease test in biopsy material, histological, microbiological, serologic, respiratory, immune-coprologic tests);

- at suspicion of autoimmune gastritis - detection of antibodies to parietal cells and Intrinsic factor in blood;

Methods for detection of Hp and its activity

1. **Direct (invasive) methods** (biopsy material):

- biochemical (CLO – test, heliko-test) for urease detection;

- histological (stain of biopsy samples and microscopy, including fluorescent microscopy);

- cultivation of Hp on special environments

2. **Indirect (noninvasive) methods**:

- immunological:
a) Serologic (serum antibody test - detection of antibodies to Hp; PCR - detection of Hp DNA in serum);
b) detection of antibodies to Hp in saliva (saliva antibody test);
c) immuno-coprologic (stool antigen test): qualitative and quantitative - breath test (with tagged C13 or C14 urea) - evaluation of urease activity consequences of Hp in samples of exhaled air: qualitative and quantitative.

**Treatment of CG**

**Therapeutic measures at autoimmune gastritis:**

1. **Replacement therapy:**
   1-2 tbsp of gastric juice 3-4 times a day during meal, 1-2 tbsp of pepsydyl 3-4 times a day; 1 tablet of acydyn-pepsin 3-4 times etc.;

2. **Gastric secretion stimulators** (at initial and moderate atrophy):
   1 tbsp of plantain juice 3-4 times a day 30 min before meal, 1 dissolved in water tablet of Limontar 1-3 times a day on an empty stomach; 15-40 drops of common wormwood tincture 3 times a day before meal; 0,5 - 1 g of plantaglucide granule (for ¼ cup water) 2-3 times a day, 20-30 minutes before meal.

3. **Enzymatic drugs** (for prevention of secondary intestinal dyspepsia):
   1-2 pills (tablets, capsules) of Festal, Mezim forte, Creon, Pankurmen or others 2-3 times a day with meal.

4. **Reparants**:
   1-2 tablets of Riboksin 3-4 times a day or 1 ampul a day intravenously with physiologic saline; 1 ampul of Retabolil intramuscularly once per 2 weeks; 0,5 g Methyluracilum 3 times a day during or after meal, etc.;

5. **At concomitant B12-deficiency anemia:**
   300-600 mcg of vitamin B12 intramuscularly;

6. **Apparatus physiotherapy:**

   electrophoresis with 5% calcium chloride on epigastric area №10, alternate-day pelotherapy № 8-10, alternate-day decimeter wave therapy № 8-10, diadynamic therapy № 10 - 15, inductothermy № 8-10, etc. Treatment may continue ambulatory after discharge from the hospital;

7. **Polyvitamins** (ascorbic acid, thiamine, pyridoxine, etc.):
   in tablet form or parenteral introduction as a separate vitamins.
Therapeutic measures at Hp-associated gastritis, duodenitis:

One of Helicobacter pylori eradication treatment regimens, that includes:

a) antibiotic, b) metronidazole or another antibiotic, c) antisecretory agent: proton pump inhibitor (PPI) during 7-10 days

**Scheme №1.**

a) 250 mg of clarithromycin 2 times a day or 1000 mg of amoxicillin 2 times a day, 500 mg of tetracycline or levofloxacin 2 times a day;

b) 500 mg of metronidazole or tinidazol 2 times a day;

c) 20 mg of omeprazole 2 times a day or other proton pump inhibitors (lansoprazole, pantoprazole, rabeprazole, esomeprazole, etc.).

**Scheme № 2.**

a) 250 mg of clarithromycin (klatsyd, klamed) 2 times a day;

b) 1000 mg of amoxicillin (augmentin, amoxiclav) 2 times a day;

c) 20 mg of omeprazole or other PPI 2 times a day.

At the risk of antibiotic resistance, ineffectiveness of previous Hp eradications, antihelicobacter treatment is carried out in a form of quadrupletherapy with additional bismuth drugss: 120 mg of De-nol (gastronorm bis-nol) 4 times a day during 2 weeks.

According to research of American helikobakteriologists, ignoring of Helicobacter pylori presence at a prescription of antisecretory therapy leads to spread of inflammation from antral part of stomach to its mucous membrane.

Chronic inflammatory process in this section after 10-15 years leads to atrophy of major glands, that significantly increases the risk of stomach cancer.

**At chemical gastritis (type C):**

- Elimination of provoking factors (NSAID taking, etc.);

- Periodic or constant intake of antisecretory drugs: proton pump inhibitors - 20 mg of pantoprazole 1-2 time a day; H2 histamine-blockers -20 mg of famotidine (Kvamatel) 1-2 time a day; selective M-cholinergic antagonists - 25-50 mg of Pirenzepine (gastrozepin) in the morning or in the evening, etc.;

- "On demand" (non-sharp pains, heartburn) - short course of antacids: 15 ml (1-2 tablets) of Maalox, Almagel, Alumag 1 - 3 times a day an hour after eating or when having symptoms.

- Prokinetics: 10 mg of metoclopramide (cerucal), domperidone (motilium), itopryd (primer) 2-3 times a day 30 minutes before meal or "on demand"; combined preparation lancidom.