<table>
<thead>
<tr>
<th>Навчальна дисципліна</th>
<th>Основи внутрішньої медицини</th>
</tr>
</thead>
<tbody>
<tr>
<td>Модуль №</td>
<td>1</td>
</tr>
<tr>
<td>Змістовний модуль №2</td>
<td>Основи діагностики, лікування та профілактики основних хвороб органів травлення</td>
</tr>
<tr>
<td>Тема заняття</td>
<td>Целіакія та інші ентеропатії</td>
</tr>
<tr>
<td>Курс</td>
<td>4</td>
</tr>
<tr>
<td>Факультет</td>
<td>Медичний</td>
</tr>
</tbody>
</table>

Харків 2013
METHODOLOGICAL RECOMMENDATIONS FOR STUDENTS
"Small intestine diseases: Celiac disease and others enteropathies"

Kharkiv 2013
Module №2 « Fundamentals of diagnostics, treatment and prevention of major diseases of the digestive system»

Topic № 7. «Small intestine diseases: Celiac disease and others enteropathies»

Prevalence of celiac disease ranges from 12 to 203 cases (the average 90-100 cases) per 100000 of population. Both children and adults may get ill. The peak of the disease is between 20-40 years. Among patients 70 % are women. After the age of 50 years disease occurs rarely. The clinical course of disease in old people has a tendency to malignant course.

The educational purposes:

✓ To teach students to distinguish the basic symptoms and syndromes of celiac disease and other enteropathies;
✓ To acquaint students with physical examination of patient with celiac disease and other enteropathies;
✓ To acquaint students with study methods which are applied to diagnostics of celiac disease and other enteropathies; indications and contraindications for every particular method; techniques of their performance; diagnostic value of each of them;
✓ To teach students to interpret the results of studies without assistance;
✓ To teach students to distinguish and diagnose complications of celiac disease and other enteropathies;
✓ To teach students to prescribe treatment of celiac disease and other enteropathies.

What should a student know?

✓ Incidence of celiac disease and other enteropathies;
✓ Etiological factors of celiac disease and other enteropathies;
✓ Pathogenesis of celiac disease and other enteropathies;
✓ The basic clinical syndromes of celiac disease and other enteropathies;
✓ The general and alarm symptoms of celiac disease and other enteropathies;
✓ Physical symptoms of celiac disease and other enteropathies;
✓ Diagnostics of celiac disease and other enteropathies;
✓ Morphological studies of intestines in case of celiac disease and other enteropathies;
✓ Laboratory methods of diagnosing celiac disease and other enteropathies;
✓ Differential diagnosis of celiac disease and other enteropathies;
✓ Classification of celiac disease and other enteropathies;
✓ Complications of celiac disease and other enteropathies;
✓ Treatment of celiac disease and other enteropathies; (change of the lifestyle, a balanced diet, pharmacological therapy).

What should a student be able to do?

• To define the basic clinical and physical syndromes of celiac disease and other enteropathies;
• To perform the interpretation of biochemical and immune-enzyme assay results;
• To perform the interpretation of the data from intestinal biopsies;
• To perform the interpretation of results of instrumental research on the intestines;
• To estimate the compliance of certain patient to the criteria of successful therapy;
• To carry out differential diagnosis;
• To prescribe the scheme of treatment to patient with celiac disease and other enteropathies.
The list of practical skills which should be acquired by student:
- Abdomen examination;
- Superficial palpation of the stomach;
- Deep methodical sliding palpation of organs of the abdominal cavity;
- Inspection of skin and mucous membranes;
- Physical examination of the liver.

Topic contents:
**Celiac disease** (gluten-sensitive enteropathy, celiac-sprue, idiopathic steatorrhea, nontropical sprue) represents diseases of the small intestine which are characterized by atrophy of its mucous membranes caused by genetical intolerance to specific protein of cereals - gluten. Celiac disease is inherited as an autosomal-dominant disease with incomplete penetrating action.

**Pathogenesis** Gluten is a prolamine, its highest concentrations can be found in wheat, barley and rye. Gliadine - is a toxic fraction of gluten. Complete hydrolysis of gliadine separates aminoacids and removes toxic effect. However hydrolysis by pepsin and pancreatic peptidases does not remove the toxic action of gliadine. The recent data show that about 95 % of patients with celiac disease are the carriers of NLA DQ2 heterodimers, which are present at people who have HLADR5, 7 and 17. It is determined that in a molecule of gliadine there is a site responsible for its toxic action. Because of presence of this site in a molecule of gliadine it can be recognized as an immunologically active component by T-lymphocytes, which have genetic peculiarities presented with heterodimer B. Activation of T-lymphocytes is accompanied by an induction of cellular immune reactions with release of cytokines that have cytotoxic properties and cause an atrophy of villi. The number of intraepithelial lymphocytes in patients with celiac disease is increased. They may cause direct or cytokine-mediated cytotoxic effect and favor the development of villi atrophy.

The representation of celiac disease as of rare disease has become outdated. The introduction of screening serological assays in medical practice has shown that its prevalence varies from 1:100 to 1: 300. The increase of celiac disease incidence may be caused by the fact that in the Western Europe and America the use of high gluten wheat had become increased over recent years.

**Clinic**

There are two peaks of disease manifestation:
- The first one - at early children's age;
- The second one - on the third-fourth decade of life.

Three clinical types of celiac disease exist:
- Classic (found at least in half of patients), subclinical (observed more often and typically is slightly expressed by transitional and extraintestinal signs) and latent (usually revealed by screening).

The most typical clinical signs of difficult course of disease are caused by deep disorders of digestion processes in the small intestine: diarrhea, polyfecalia, steatorrhea, meteorism, anorexia, loss of body weight up to cachexia, weakness. Bowel movements are frequent, liquid, and foamy with remains of undigested food.

The basic mechanism of diarrhea in celiac disease is connected with intensification of intestinal secretion due to increase of prostaglandins, which is caused by the following mechanisms: on the one hand there is raised synthesis of prostaglandins by mononuclear, which infiltrate the mucous membrane of the small intestine, and on the other hand - the decrease in their inactivation due to atrophy of mucous membrane. The diarrhea is the reason for loss of electrolytes and dehydration.

Due to malabsorption development a polyhypovitaminosis, hypoosmolality edemas may occur. The exhaustion of calcium and magnesium deposits can cause paraesthesia, muscular spasms and even tetany. Malabsorption is accompanied by osteoporosis and different neurological diseases. Extraintestinal presentations of celiac disease include Dermatits Herpetiformis.
In severe cases, when the pathological process involves entire small intestine, a total syndrome of malabsorption may develop, which can be life-threatening.

**Clinical forms of Celiac disease (after Dierkx R. et al., 1995):**

1. **The active clinical form (typical).** The symptoms vary from the extremely difficult malabsorption syndrome with an exhaustion and edemas to moderately expressed disorders of absorption or atypical symptoms (anorexia, arthralgia, stomatitis, hair loss, retarded growth of children, etc.).

2. **The subclinical form.** Basically there are no typical symptoms of malabsorption, while the morphological changes of a mucous membrane are present. The only symptom of celiac disease can sometimes be micro- or macrocytic anemia.

3. **The latent form.** Happens in the case, when the patient is on a gluten-free diet, and the biopsy of the small intestine is normal. The mucous membrane, however, is flat and gets restored after introduction of gluten-free diet.

4. **The potential form.** The small intestine mucous membrane is normal, however, after loading with gluten the celiac disease may develop.

**Clinical signs of celiac disease**

**Typical clinical syndromes and symptoms:**

- The first signs of disease in the age younger than 10 years (86 %).
- Diarrhea at day and night (91 %), polyfecalia, steatorrhea (80 %)
- Syndrome of impaired absorption I - II – III stage of severity (90 %)
- Anorexia (40 %)
- Meteorism (75 %)
- Pain in abdomen (70 %)
- Iron-deficiency anaemia in children (70 %), B-12-folic acid in elderlies (50 %)

**Clinical symptoms and syndromes which can be observed rarely:**

- The first signs of disease in the age older than 10 years (14 %)
- Alternations of diarrhea and constipation (4 %), diarrhea crises with signs of dehydration against the background of normal bowel movements (5 %)
- Syndrome of exudative enteropathy (10 %)
- Bleeding syndrome (5 %)
- Attacks of dynamic intestinal obstruction due to atonic muscles of the gut wall (5 %)
- Enteropathic osteopathy: osteoporosis, osteomalacia (5 %)
- Deficiency of immunoglobulin A (1 %), leukocytosis (1 %), increase of ESR (1 %)

**Diagnostic Criteria (European Society of Gastroenterology and experts on nutrition, 1969):**

- Stable gluten intolerance;
- Hyperregenerative type of mucosal atrophy of the small intestine develops in the active phase of the disease;
- After introduction of gluten-free diet mucosa of the small intestine is restored;
- Re-introduction of gluten in food leads to mucosal atrophy of the small intestine.

Other criteria:

- The presence of malabsorption syndrome
- Serological studies;
- Morphological study.
**Serological assays of celiac disease**

Several serologic markers have become available that have altered the classic diagnostic pathway. The sensitivity of IgA anti-gliadin antibodies (AGA) is reported to range from 70 to 85 percent, whereas the specificity ranges from 70 to 90 percent. IgA anti-endomysial (EMA) and anti-tissue transglutaminase (tTG) antibodies have sensitivities in excess of 90 percent and specificities of over 95 percent. Significant variability seems to exist in the reported values among the different studies, and these IgA-based tests can be negative in IgA-deficient patients, accounting for about 3 percent of CD cases.

The sensitivity and specificity of the anti-EMA and anti-tTG antibodies, along with the perceived under diagnosis of CD, has led to suggestions of using these tests for population screening. Aside from the recognized influence of CD prevalence on the predictive value of a serologic test result, little consensus exists regarding the value of population screening. Furthermore, specific questions regarding clinically important outcomes resulting from screening remain unclear. In particular, little data is available on adherence to a gluten-free diet in asymptomatic CD patients detected by screening.

**Morphological changes** of a mucous membrane of a small intestine (distal parts of the duodenal gut) in celiac disease:
- Flattening, reduction or total disappearance of mucous membrane villi;
- Enlargement of intestinal crypts
- Deformation of superficial epithelium through destruction of the brush border (microvilli are short and rare). Cylindrical epithelium it is replaced by the cubic one.
- Dense lymphoplasmocytic infiltration and lympho-cellular infiltration of epithelium.

Increased number of intraepithelial lymphocytes and presence of antigliadine antibodies in blood serum are the early morphological signs of celiac disease.

**The differential diagnosis:**
- The general variability of hypogammaglobulinemia (hereditary disease);
- Celiac sprue
- Tropical sprue
- Lymphoma of the small intestine

**Complication**

Without treatment, celiac disease leads to early menopause, infertility, risk of repeated abortions in women. Neurologic complications include cerebral degeneration, polyneuropathy, myopathy, cerebral calcification. The malabsorption syndrome is the reason for development of osteoporosis and pathologic fractures. Dangerous complications include ulcers with bleedings and perforations. Celiac disease raises risk of development of neoplasms, such as lymphoma (malignant histiocytosis of intestines, T-cellular lymphoma) and intestinal adenocarcinoma, especially in people who do not keep a diet.

**Treatment**

The gluten-free diet is the basic therapy of celiac disease.

Main principles of this diet:
- Exclusion of products which contain gluten (wheat, rye, barley and oats), and also products which increase fermentative and decay processes;
- A full value diet with raised contents of fiber and calcium salts;
- Limitation of influence of mechanical and chemical stimuli on intestinal mucous membrane.
Along with a gluten-free diet, a correction of vitamin insufficiency and other derangement caused by malabsorption should be done.
In cases of anemia, preparations of folic acid and iron should be prescribed.
In advanced cases, use 20-30 mg of Prednisolone and gradually decrease the dose.
In case of marked syndrome of malabsorption - infusions, enzyme preparations.

**Disaccharide Deficiency Syndrome (lactoses, maltose, isomaltose, sucrose, triglycerides, etc.)** - decrease in activity or absence of one or several disaccharides is a congenital or acquired disease which leads to disorders of digestion and absorption of disaccharides. This disease is often accompanied by lactose intolerance.
In European race occurs in 5-15%, often against the background of some bowel disease - chronic enteritis, Crohn's disease, ulcerative colitis, receiving antibiotics and oral contraceptives and other medications.
Clinical symptoms: abdominal pain of spastic character, pronounced flatulence, diarrhea (watery diarrhea in 0.5 - 3 hours after eating intolerable disaccharide), polyfecaalia.
Diagnostics. Clinical symptoms appear after consuming milk or other disaccharides.
Coprological study - sour stool - pH less than 6.0.
Lactose tolerance test - slight increase of blood glucose level (less than 3.0 mmol / l) after ingestion of 50 g of lactose, and the presence of flatulence, tenesmus, diarrhea.
Respiratory test - after ingestion of lactose there is an increase of exhaled hydrogen volume.
Morphological examination - the histochemical method confirms the low activity or lack of lactase in a biopsy specimen taken from the small intestine.
Treatment. Dairy-free diet.
If malabsorption syndrome is present - a replacement and corrective therapy should be done.

**Malabsorption syndrome** - a syndrome that includes diarrhea, weight loss, protein deficiency and signs of vitamin deficiencies arising from the disorders of food digestion and absorption in the intestine.
Etiology and pathogenesis.
1. Disorders of digestion due to insufficient food enzymes and bile salt deficiency.
2. Disorders of digesting food due to microbial colonization of the small intestine.
3. Malabsorption of food ingredients caused by decreased absorption surface or by morphological and functional changes of absorptive epithelium of the small intestine.
4. Damage of specific transport mechanisms in case of malfunction of lymphatic system and / or darining bowel blood vessels.
5. Changes in bile acid metabolism caused by microbial colonization of the small intestine or by disorders of intrahepatic circulation of bile acids.
Clinical symptoms and signs

<table>
<thead>
<tr>
<th>Organ, system</th>
<th>Symptom or sign</th>
<th>Pathophysiological basis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive system</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diarrhea</td>
<td>Disorders of absorption or increased secretion of water</td>
</tr>
<tr>
<td></td>
<td>Steatorrhea</td>
<td>Disorders of intraluminal hydrolysis of fats</td>
</tr>
<tr>
<td></td>
<td>Flatulence</td>
<td>Bacterial fermentation of carbohydrates that were not absorbed</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain, anorexia, nausea</td>
<td>Intraluminal accumulation of unabsorbed nutrients, gases. Motility disorders.</td>
</tr>
<tr>
<td>Bile ducts</td>
<td>Gall stones</td>
<td>Disorders of entero-hepatic circulation of bile acids</td>
</tr>
<tr>
<td>Metabolic disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Protein and energy insufficiency</td>
<td>Maldigestion, malabsorption, malassimilation.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Disorders of water and electrolyte metabolism</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intra- and extracellular deficiency of water and electrolytes</td>
</tr>
<tr>
<td></td>
<td>Symptoms of hypovitaminosis.</td>
<td>Reduced of assimilation of fat-soluble vitamins due to disorders of the formation of mixed micelles</td>
</tr>
<tr>
<td>Hemopoetic system</td>
<td>Anemia</td>
<td>Malabsorption of iron, vitamin B12, folic acid</td>
</tr>
<tr>
<td></td>
<td>Hemorrhagic symptoms</td>
<td>Malabsorption of vitamin K and hyporthrombinemia</td>
</tr>
<tr>
<td>Musculoskeletal system</td>
<td>Pain in the bones, paresthesia, osteoporosis</td>
<td>Malabsorption of calcium, magnesium, vitamin D</td>
</tr>
<tr>
<td>Kidneys</td>
<td>Urolithiasis</td>
<td>Oxaluria</td>
</tr>
<tr>
<td>Endocrine organs</td>
<td>Hypothalamic and pituitary insufficiency, hypofunction of thyroid gland, adrenals and sexual glands</td>
<td>Malabsorption of main nutrients</td>
</tr>
</tbody>
</table>

Physical data.

**Malnutrition** - decrease in body weight. According to the degree of weight loss there are 3 stages: I stage (the mild form) - up to 5 kg; II stage (moderate) - to 10 kg; III stage (severe) - progressive weight loss.

On examination of the stomach - bloating, flatulence; on abdominal palpation - local resistance and hypersensitivity to the left and above the navel; on auscultation - rumbling and splashing noise; on percussion - tympanic sound over gases present in bloated loops of small intestine.

Clinical and laboratory criteria for diagnostics of nutrition insufficiency:

<table>
<thead>
<tr>
<th>Index</th>
<th>Standard</th>
<th>Stage of nutrition insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mild</td>
</tr>
<tr>
<td><strong>Albumin, g/L</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;35</td>
<td>35-30</td>
<td>30-25</td>
</tr>
<tr>
<td><strong>Transferrin, g/L</strong></td>
<td>&gt;2,0</td>
<td>2,0-1,8</td>
</tr>
<tr>
<td><strong>Lymphocytes, 10⁹/L</strong></td>
<td>&gt;1800</td>
<td>1800-1500</td>
</tr>
<tr>
<td><strong>Skin reaction to antigens, mm</strong></td>
<td>&lt;15</td>
<td>15-10</td>
</tr>
</tbody>
</table>
Disorders of lipid metabolism – reduce of subcutaneous fat, steatorrhea, change of serum lipid spectrum (hipocholesterolemia, hypophosphatemia, decreased triglycerides).

Disorders of carbohydrate metabolism - the tendency to low blood glucose levels, flat glycemic curve after loading with carbohydrates.

Disorders of water and electrolyte metabolism - dehydration (thirst, dry skin and mucous membranes, oliguria); hyponatremia (anorexia, nausea, muscle weakness, seizures, lethargy, decreased blood pressure, oliguria); hypokalemia (drowsiness, tachyarrhythmia, flattening of the T wave on ECG); hypocalcaemia (paresthesias of hands and feet, muscle pain, positive Chvostek’s and Trousseau’s symptoms).
Principles of treatment

1. Basic diet - high content of animal protein (130-135 g), certain vitamins and minerals, normal content of fat and carbohydrates. The substances containing crude fiber should be excluded.

2. Elemental diet - mixtures with balanced chemical composition and good ability to dissolve, with no ballast substances and eliminated intolerable substances (Nutrizan, Izokal, Filotakt, Ovolakt).

3. Synthetic diets - mixtures consisting of amino acids, unsaturated fatty acids, and glucose polymers of low osmolality (Enpit, Inpytan, Ovolakt)

4. Drug therapy:
   - Infusion therapy, which aims to compensate the protein deficiency (saline, amino acids solutions, albumin, plasma); correction of fluid and electrolyte disorders - saline, glucose, Asparcam, Panangin, calcium gluconate; correction of anemia and vitamin deficiencies - iron supplements, vitamins of B group, ascorbic acid, nicotinic acid, Essentiale);
   - Correction of hemostasis - plasma, Dicynone, Vikasol etc.
   - Immune-stimulating therapy – T-aktyvin, Thymogen, Immunal;
   - Improvement of digestion and absorption processes - enzyme replacement therapy, decrease of pronounced flatulence (espumizan, meteospazmil);
   - Normalization of motility - antispasmodics or prokinetics.
   - In case if malabsorption is caused by intense motility prescribe loperamide (imodium).
   - Good antidiarrheal effect has sandostatin.
   - Enterosorbents - Polifekan, Smecta, Enterosgel, bile acid sequestrants (cholestyramine).
The control of initial level of knowledge

1. What disorder of albumin metabolism can be found in patient with celiac disease?
   A. Decrease of body weight
   B. Reduction of subcutaneous fatty tissue, steatorrhea
   C. Intestinal dyspepsia
   D. Derangements of mineral and vitamin absorption
   E. Hypovitaminosis

2. What is the mechanism of malabsorption development?
   A. Derangements in food absorption
   B. Derangements of food digestion
   C. Anemia, leukopenia
   D. Vomiting and faintness
   E. Hypovitaminosis

3. What medical substances would you recommend in the case of disturbed motor function of the intestines?
   A. Enzymes
   B. Spasmolytics, Prokinetics
   C. M-cholinolytics
   D. Anabolic steroids
   E. Antacids

4. What cells are not present in the small intestine?
   A. Goblet
   B. Endocrine cells
   C. G-cells
   D. Enterocytes
   E. Intraepithelial lymphocytes

5. What artery supplies blood to the small intestine?
   A. Gastro-duodenal
   B. Splenic
   C. Superior mesenteric
   D. Inferior mesenteric
   E. Superior rectal

6. What functions are not performed in the small intestine?
   A. Absorption of vitamins
   B. Production of intestinal hormones
   C. Formation of feces
   D. Protective function
   E. Membrane and endocellular digestion

7. Select an osmotic laxative:
   A. Fibrous food
   B. Chickweed preparations
   C. Buckthorn preparations
   D. Guttalax
   E. Forlax
8. What is the action of Imodium?
   A. Prokinetic effect
   B. Hepatoprotective effect
   C. Anesthetic effect
   D. Sedative effect
   E. All mentioned above

9. In case of marked meteorism, it is better to use:
   A. No-spa
   B. Mezym-forte
   C. Enterosgel
   D. Smecta
   E. Probiotic

10. Which one of the listed below symptoms is not typical for celiac disease?
    A. Orthostatic hypotension
    B. Progression of symptoms
    C. Steatorrhea
    D. Chronic diarrheas
    E. Gluten intolerance

**Celiac disease (initial level of knowledge)**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A</td>
<td>6. C</td>
</tr>
<tr>
<td>2. A</td>
<td>7. E</td>
</tr>
<tr>
<td>3. B</td>
<td>8. A</td>
</tr>
<tr>
<td>5. C</td>
<td>10. D</td>
</tr>
</tbody>
</table>
Control of final level of knowledge

1. Complications of celiac disease do **not** include one of the following:
   A. Diarrhea
   B. Lymphoma of the small intestine
   C. Non-granulomatous enteritis
   D. Celiac Sprue
   E. Neuropathy

2. What is the mechanism of development of celiac disease?
   A. Immunological reaction
   B. Accumulation of toxic substances
   C. Infectious agents
   D. Pregnancy
   E. All of mentioned above

3. Which one of the listed below symptoms contradict the diagnosis of malabsorption?
   A. Abdominal pain that eases after defecation
   B. Lactose intolerance
   C. Excreting faeces with mucous
   D. Dysbacteriosis
   E. Increased body weight

4. What is typical for disaccharide enteropathy?
   A. Vasoconstrictive reaction
   B. Feeling of a lump in the throat
   C. Disorders of urination
   D. Intolerance to milk
   E. Sexual dysfunction

5. The basic treatment of enteropathy includes:
   A. Diet
   B. Preparations of 5-ASK
   C. Antacids
   D. Bismuth preparations
   E. Metronidazol

6. The main laboratory criteria for diagnostics of celiac disease:
   A. Complete blood count
   B. Biochemical tests
   C. Serological blood tests
   D. Bacteriological fecal investigation
   E. Markers of viruses in blood serum.

7. The following metabolic disorders are typical for malabsorption syndrome:
   A. Fluid and electrolyte
   B. Fat
   C. Carbohydrate
   D. Protein
   E. All of the mentioned above
8. What are the gastrogenic reasons for the development of malabsorption syndrome:
A. Gastrectomy
B. Liver cirrhosis
C. Pancreatic resection
D. Crohn's disease
E. Diverticulum

9. What is the pH of feces in case of presence of disaccharidases insufficiency syndrome?
A. More than 6,0
B. Less than 6,0
C. More than 7,0
D. 10,0
E. 10,0-20,0

10. What diet should be kept in case of disaccharidases insufficiency?
A. Dairy-free
B. Protein-free
C. With big quantities of fats
D. Restriction of simple carbohydrates
E. All of the mentioned above

Celiac disease and Enteropathies (final level of knowledge)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. B</td>
<td>6. C</td>
</tr>
<tr>
<td>2. E</td>
<td>7. E</td>
</tr>
<tr>
<td>3. E</td>
<td>8. A</td>
</tr>
<tr>
<td>5. A</td>
<td>10. A</td>
</tr>
</tbody>
</table>
Case-based questions

1. Patient, 55 years old, complains on bloating and rumbling in the abdomen, excessive discharge of gases, liquid stool of foamy character with a sour odor. The symptoms develop after ingestion of dairy products. What is the most likely cause of this condition?
   A. Lactose insufficiency
   B. Gluten insufficiency
   C. Insufficiency of bile acids
   D. Intestinal dyskinesia
   E. Syndrome of malabsorption

2. A man of 48 years old complains of an aching pain in lateral parts of the stomach with alternation of diarrheas and constipation. Pain decreases after defecation and discharge of gases. 2 years ago the patient had a gastrectomy. On palpation – tenderness, alternation of spasmodic and atonic sections, rumbling in the small intestine. What method of examination is the most informative for making diagnosis:
   A. Comprogram
   B. Bimanual rectal examination
   C. Rectomanoscopy
   D. Colonoscopy
   E. Morphological research of the small intestine

3. Patient, 32 years old, took a massive antibiotic therapy. He complains on diffuse abdominal pain, frequent liquid stools (4-6 times a day), general weakness. On palpation the abdomen is soft and tender in the lower regions, the liver and spleen are not palpable. The use of what drug is appropriate in this case?
   A. Imodium
   B. Panzy norm
   C. Essentiale
   D. Linex
   E. Motilium

4. Patient, 48 years old, complains on a periodic pain in the left half of the stomach, faintness after the use of flour products, and frequent liquid excrements. She lost 5 kg of body weight during 2 months. Objective data: patient is underweight, abdomen is soft, rumbling is present. Stool 3-4 times a day, with a touch of neutral fat. What pathology most likely leads to the mentioned above condition?
   A. Gluten enteropathy
   B. Chronic pancreatitis
   C. Chronic hepatitis
   D. Chronic enterocolitis
   E. Autoimmunne gastritis

5. Patient, 62 years old, has been suffering from chronic pancreatitis for 32 years. Complains on recurrent pain in the left upper quadrant, and therefore has to keep a strict diet with restriction of fatty, fried, spicy, smoked foods. During the last 1,5 months the following symptoms occurred: bloating, increased frequency of bowel movements up to 2 - 3 times a day. Stool is profuse, malodorous, with shiny surface, with the remnants of undigested food. Weight loss - 8 kg. What syndrome prevails in clinical presentation of this disease?
A. Astheno-neurological
B. Endocrine insufficiency of a pancreas
C. Syndrome of malabsorption
D. Irritable bowel syndrome
E. Pain syndrome

6. Patient, 41 years old, has celiac disease. She had lost 7kg of her body weight during last 2 months.
   What is the degree of nutrition deficit in this patient?
   A. First
   B. Second
   C. Third
   D. Fourth
   E. Fifth

7. Patient, 14 years old, complains on weakness and weight loss. Her general condition usually worsens after the use of a considerable quantity of flour products. This phenomenon had been observed since her early childhood. The general condition is satisfactory and weight loss is evident. Retardation of physical development. What could be the possible reason for this disease?
   A. Gluten enteropathy
   B. Helminthic invasion
   C. Maldigestion
   D. Dysbacteriosis of intestines
   E. Lactase deficiency

8. Patient, 14 years old, complains on weakness and weight loss. Her general condition usually worsens after the use of a considerable quantity of flour products. This phenomenon had been observed since her early childhood. The general condition is satisfactory and weight loss is evident. Retardation of physical development. What could be the possible reason for this disease?
   What indexes should be determined for making the diagnosis:
   A. IgM
   B. IgA
   C. IgG
   D. IgE, IgM
   E. Ig A- AGA, Ig A- EmA

9. A girl of 17 years old was diagnosed with celiac disease. The patient refuses to follow the gluten-free diet. What complications may develop as the result of this?
   A. Infertility
   B. Osteoporosis
   C. Risk of early abortions
   D. All of mentioned above

10. A patient of 51 years old suffers from celiac disease. Throughout 6 months he has been complaining on pains in bones, paresthesias, cramps. What is the possible cause for such condition?
A. Development of anemia
B. Disorders of fluid and electrolyte balance
C. Development of osteoporosis
D. Disorders of carbohydrate metabolism
E. Disorders of protein metabolism

CORRECT ANSWERS

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>2.</td>
<td>E</td>
<td>6.</td>
<td>B</td>
</tr>
<tr>
<td>3.</td>
<td>D</td>
<td>7.</td>
<td>A</td>
</tr>
<tr>
<td>4.</td>
<td>A</td>
<td>8.</td>
<td>E</td>
</tr>
</tbody>
</table>

Control questions:
1. Give definition for celiac disease and enteropathy
2. The basic clinical syndromes of celiac disease and enteropathy
3. The physical data of celiac disease and enteropathy
4. Clinical symptoms and signs of celiac disease
5. Clinical symptoms and signs of enteropathy
6. Diagnostic methods for celiac disease and enteropathy
7. Complications of celiac disease and enteropathy
8. Treatment principles of celiac disease and enteropathy
9. Lifestyle and diet in case of celiac disease and enteropathy
10. Pharmacological therapy of celiac disease and enteropathy depending on severity and activity of disease
11. Prevention of celiac disease and enteropathy

Practical problems.
1. To examine patients with celiac disease and enteropathy
2. To interpret received results of laboratory research.
3. To interpret received results of instrumental investigations.
4. To perform differential diagnostics between celiac disease and other enteropathies
5. To name complications of celiac disease and enteropathy
6. To write recipes for medications used for treatment of celiac disease and enteropathy
The report of clinical examination of the patient (the uniform form)

Name, Surname

Age _______ Profession

Complaints

Anamnesis morbi

Last exacerbation

Anamnesis morbi

Results of physical examination of the patient:

The preliminary diagnosis:

The examination plan:

Results of additional studies:
Substantiation of the clinical diagnosis:

The clinical diagnosis:
Main disease: ________________________________

Accompanying diseases:

Complication

Treatment:
1. Regimen ______________
2. Diet _________________
3. ______________________
4. ______________________
5. ______________________
The materials for self-preparation: