Міністерство охорони здоров'я України
Харківський національний медичний університет

Кафедра Внутрішньої медицини №3
Факультет VI по підготовці іноземних студентів

ЗАТВЕРДЖЕНО
на засіданні кафедри внутрішньої медицини №3
29 серпня 2016 р. протокол № 13
Зав. кафедри _____д.мед.н., професор Л.В. Журавльова

МЕТОДИЧНІ ВКАЗІВКИ
для студентів

з дисципліни «Внутрішня медицина (в тому числі з ендокринологією)
студенти 4 курсу I, II, III медичних факультетів, V та VI факультетів по підготовці іноземних студентів

Целіакія та інші ентеропатії

Харків 2016

KHARKOV NATIONAL MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL MEDICINE N3

METHODOLOGICAL RECOMMENDATIONS FOR STUDENTS

"Celiac disease and others enteropathies"

Kharkiv 2016

Practical class. «Small intestine diseases: Celiac disease and others enteropathies», 4 hours

Celiac disease is a common cause of malabsorption of one or more nutrients. Although celiac disease was originally considered largely a disease of white individuals, especially persons of European descent, recent observations have established that it is a common disease with protean manifestations, a worldwide distribu- tion, and an estimated incidence in the United States that is as high as 1 in 113 people. Its incidence has increased over the past 50 years. Celiac disease has had several other names, including nontropical sprue, celiac sprue, adult celiac disease, and gluten-sensitive enteropa- thy. The etiology of celiac disease is not known, but environmental, immunologic, and genetic factors are important. Celiac disease is con- sidered an "iceberg" disease. A small number of individuals have clas- sical symptoms and manifestations related to nutrient malabsorption along with a varied natural history; the onset of symptoms can occur at all points from the first year of life through the eighth decade. A much larger number of individuals have "atypical celiac disease", with manifestations that are not obviously related to intestinal malabsorp- tion (e.g., anemia, osteopenia, infertility, and neurologic symptoms). Finally, an even larger number of persons have "silent celiac disease"; they are essentially asymptomatic despite abnormal small-intestinal histopathology and serologies.

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, 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.

Topics content:

Celiac disease, also known as celiac sprue or gluten-sensitive enteropathy, is a chronic disorder of the digestive tract that results in an inability to tolerate gliadin, the alcohol-soluble fraction of gluten. Gluten is a protein commonly found in wheat, rye, and barley.
When patients with celiac disease ingest gliadin, an immunologically mediated inflammatory response occurs that damages the mucosa of their intestines, resulting in maldigestion and malabsorption of food nutrients.

Etiology

Celiac disease results from a combination of immunological responses to an environmental factor (gliadin) and genetic factors.

One ***environmental*** factor is the clear association of the disease with gliadin, a component of gluten that is present in wheat, barley, and rye. In addition to the role of gluten restriction in treatment, the instillation of gluten into both the normal-appearing rectum and the distal ileum of patients with celiac disease results in morphologic changes within hours.

Immune mechanisms

The interaction of alcohol-soluble gliadin in wheat, barley, and rye products with the mucosa of the small intestine is crucial to the pathogenesis of celiac disease. Endogenous tissue transglutaminase deamidates glutamine in gliadin, converting it from a neutral to a negatively charged protein. Negatively charged gliadin has been shown to induce interleukin 15 in enteric epithelial cells, stimulating the proliferation of natural killer cells and intraepithelial lymphocytes to express NK-G2D, a marker for natural killer T lymphocytes. Gliadin can produce symptoms and histological changes in the small intestine when administered to patients with asymptomatic celiac disease. Antigliadin antibodies can frequently be identified in untreated patients.

Genetic factors

Genetics play an important role in celiac disease. The incidence of celiac disease in relatives of patients with celiac disease is significantly higher than in the general population. The prevalence in

first-degree relatives of patients with celiac disease is approximately 10%. Concordance for the disease in monozygotic twins approaches 75% and is approximately 30% for first-degree relatives. However, serologic studies provide clear evidence that celiac disease is present worldwide. Furthermore, all patients with celiac disease express the HLA-DQ2 or HLA-DQ8 allele, although only a minority of people expressing DQ2/DQ8 have celiac disease. Absence of DQ2/DQ8 excludes the diagnosis of celiac disease.

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. 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. Helper T cells mediate the inflammatory response. Endogenous tissue transglutaminase deamidates gliadin into a negatively charged protein, increasing its immunogenicity. Absence of intestinal villi and lengthening of intestinal crypts characterize the mucosal lesions in untreated celiac disease. More lymphocytes infiltrate the epithelium (intraepithelial lymphocytes). Destruction of the absorptive surface of the intestine leads to a maldigestive and malabsorption syndrome.

Clinical presentation

Signs and symptoms

Gastrointestinal symptoms

- Diarrhea is the most common symptom in untreated celiac disease and is present in 45-85% of all patients. Diarrhea caused by celiac disease is due to maldigestion and malabsorption of nutrients. The stools might be watery or semiformed, light tan or gray, and oily or frothy. The stools have a characteristic foul odor. In infants and young children, extensive diarrhea can lead to severe dehydration, electrolyte depletion, and metabolic acidosis.
- Malabsorption of ingested fat (steatorrhea) results in the delivery of excessive dietary fat to the large bowel. This results in the production of hydroxy fatty acids by bacteria, which causes secretion of fluids into the intestine.
- Flatulence (28% of patients) and borborygmus (35-72% of patients) results from the release of intestinal gas by the bacterial florae feasting on undigested and unabsorbed food materials and often becomes excessive or even explosive.
- Weight loss (present in 45% of all patients) is variable because some patients might compensate for the malabsorption by increasing dietary intake. In infants and young children with untreated celiac disease, failure to thrive and growth retardation are common.
- Weakness and fatigue (prevalence 78-80%) are usually related to general poor nutrition. In some patients, severe anemia can contribute to fatigue. Occasionally, severe hypokalemia due to the loss of potassium in the stool can cause muscle weakness.
- Severe abdominal pain (prevalence 34-64%) is unusual in patients with uncomplicated

celiac disease. However, abdominal bloating or cramps with excessive malodorous flatus is a common complaint.

Extraintestinal symptoms

- Anemia (10-15% of patients) is usually due to impaired absorption of iron or folate from the proximal small intestine. In severe celiac disease with ileal involvement, absorption of vitamin B-12 might be impaired.
- A bleeding diathesis is usually caused by prothrombin deficiency due to impaired absorption of fat-soluble vitamin K.
- Osteopenia and osteoporosis (prevalence 1-34%) might cause bone pain for several reasons, including defective calcium transport by the diseased small intestine, vitamin D deficiency, and binding of luminal calcium and magnesium to unabsorbed dietary fatty acids.
- Neurologic symptoms (frequency 8-14%) that result from hypocalcemia include motor weakness, paresthesias with sensory loss, and ataxia. Seizures might develop because of cerebral calcifications.
- Skin disorders, including dermatitis herpetiformis (a pruritic papulovesicular skin lesion involving the extensor surfaces of the extremities, trunk, buttocks, scalp, and neck), is associated in 10-20% of patients with celiac disease.
- Hormonal disorders, such as amenorrhea, delayed menarche, and infertility in women and impotence and infertility in men, have been described.

Physical examination

A physical exam may reveal the following:

- Abdominal examination shows a protuberant and tympanic abdomen due to distention of intestinal loops with fluids and gas. Ascites occasionally can be detected in patients with severe hypoproteinemia.
- Evidence of weight loss, including muscle wasting or loose skin folds
- Orthostatic hypotension
- Peripheral edema
- Ecchymoses
- Hyperkeratosis or dermatitis herpetiformis
- Cheilosis and glossitis
- Evidence of peripheral neuropathy
- Chvostek sign or Trousseau sign

Diagnosis

The diagnosis of celiac disease requires the detection of characteristic histologic changes on small-intestinal biopsy together with a prompt clinical and histologic response after the institution of a gluten-free diet. If IgA antiendomysial or anti-tissue transglutaminase antibodies have been detected in serologic studies, they too should disappear after a gluten-free diet is started. With the increase in the number of patients diagnosed with celiac disease (mostly by serologic studies), the spectrum of histologic changes seen on duodenal biopsy has increased and includes findings that are not as severe as the classic changes shown in. The classic changes seen on duodenal/jejunal biopsy are restricted to the mucosa and include (1) an increase in the number of intraepithelial lymphocytes; (2) absence or a reduced height of villi, which causes a flat appearance with increased crypt cell proliferation resulting in crypt hyperplasia and loss of villous structure, with consequent villous, but not mucosal, atrophy; (3) a cuboidal appearance and nuclei that are no

longer oriented basally in surface epithelial cells; and (4) increased numbers of lymphocytes and plasma cells in the lamina propria. Although these features are characteristic of celiac disease, they are *not* diagnostic because a similar appearance can develop in tropical sprue, eosinophilic enteritis, and milk-protein intolerance in children and occasionally in lymphoma, bacterial overgrowth, Crohn's disease, and gastrinoma with acid hypersecretion. However, a characteristic histologic appearance that reverts toward normal after the initiation of a gluten-free diet establishes the diagnosis of celiac disease. Readministration of gluten, with or without an additional small-intestinal biopsy, is not necessary.

A number of patients exhibit *gluten sensitivity*; i.e., they have gastrointestinal symptoms that respond to gluten restriction but do not have celiac disease. The basis for such gluten sensitivity is not known.

Laboratory tests

The American College of Gastroenterology recommends that antibody testing, especially immunoglobulin A anti-tissue transglutaminase antibody (IgA TTG), is the best first test for suspected celiac disease, although biopsies are needed for confirmation; in children younger than 2 years, the IgA TTG test should be combined with testing for IgG-deamidated gliadin peptides.
Other laboratory tests include the following:

- Electrolytes and chemistries - Electrolyte imbalances; evidence of malnutrition
- Hematologic tests - Anemia, low serum iron level, prolonged prothrombin time (PT)
- Stool examination - Fat malabsorption
- Oral tolerance tests - Lactose intolerance
- Serology - Immunoglobulin A (IgA) antibodies

Patients diagnosed with celiac disease should be examined for deficiencies, including low bone density. Patients already on a gluten-free diet without prior testing need to be evaluated to assess the likelihood that celiac disease is present; genetic testing and a gluten challenge are most helpful.

Imaging studies

Radiographic evaluation of the small bowel after barium ingestion is helpful in making a diagnosis of untreated celiac disease. Abnormal radiographic findings can include dilatation of the small intestine, a coarsening or obliteration of the normally delicate mucosal pattern, and fragmentation or flocculation of the barium in the gut lumen.

Endoscopy and biopsy

Upper endoscopy with at least 6 duodenal biopsies is considered the criterion standard to help establish a diagnosis of celiac disease. Histologically, duodenal biopsies can be graded into the following 5 stages:

- Stage 0 - Normal
- Stage 1 - Increased percentage of intraepithelial lymphocytes (>30%)
- Stage 2 - Increased presence of inflammatory cells and crypt cell proliferation with preserved villous architecture
- Stage 3 - Mild (A), moderate (B), and subtotal to total (C) villous atrophy
- Stage 4 - Total mucosal hypoplasia

<u>Differential diagnosis</u>

- Bacterial gastroenteritis

- Bacterial overgrowth syndrome
- Chron disease
- Enteropathy-Type T-Cell Lymphoma
- Eosiniphilic gastroenteritis
- Inflammatory bowel disease
- Iron deficiency anemia
- Irritable bowel syndrome
- Jejunoileitis
- Malabsorption

Complications

The risk for malignant disease is increased in patients with celiac disease. These malignancies include adenocarcinoma of the oropharynx, esophagus, pancreas, small and large bowel, and hepatobiliary tract. Other malignancies with an increased incidence in patients with celiac disease are enteropathy-associated T-cell lymphoma with a poor prognosis and T- and B-cell non-Hodgkin lymphoma.
Refractory celiac disease occurs in approximately 5% of patients despite strict adherence to a gliadin-free diet. Refractory celiac disease is characterized by symptoms of malabsorption, weight loss, diarrhea, abdominal distention, and anemia. Refractory celiac disease is subdivided into two types: Type 1 is characterized by a normal intraepithelial lymphocyte phenotype, and type 2 is characterized with an increased number of intraepithelial lymphocytes, possibly due to an increase in epithelial interleukin 15 expression.

Treatment

Diet
The primary management of celiac disease is dietary. Complete elimination of gluten-containing grain products (including wheat, rye, and barley) is essential to treatment. However, complete avoidance of gluten-containing grain products is relatively difficult for patients to achieve and maintain because certain products, such as wheat flour, are virtually ubiquitous in the international diet.
Main principles of diet:
- exclusion of products which contain gluten (wheat, rye, barley and oats), and also products which increase fermentative and decay processes;
- 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, a preparations of folic acid and iron should be prescribed.

The goals of ***pharmacotherapy*** are to reduce morbidity and to prevent complications.
A small percentage of patients with celiac disease fail to respond to a gluten-free diet. In some patients who are refractory, **corticosteroids** might be helpful. Use 20-30 mg of Prednisone and gradually decrease the dose In patients who fail to respond to corticosteroids, other comorbid conditions, such as lymphomas of the small intestine, have to be ruled out.

Disaccharide Deficiency Syndrome (lactoses, maltose, isomaltose, sucrose, trigvlcerides, 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), polyfecalia.
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.
6. Disorders of motor function of the gastrointestinal tract (stagnation of the contents, change of sphincters activity, accelerated passage).

Clinical symptoms and signs

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

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:

Index	Standard	Stage of nutrition insufficiency		
		Mild	Moderate	Severe
Albumin, g/L	>35	35-30	30-25	<25
Transferrin, g/L	>2,0	2,0-1,8	1,8-1,6	<1,6
Lymphocytes, 10^9/L	>1800	1800-1500	1500-900	<900
Skin reaction to antigens, mm	<15	15-10	10-5	<5

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 aminoacids, 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, aminoacids 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

Correct answers:

1. A	**6. C**
2. A	**7. E**
3. B	**8. A**
4. B	**9. E**
5. C	**10. D**

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. Bacterological 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. A11 of the mentioned above

Correct answers:

l . B	**6. C**
2. E	**7. E**
3. E	**8. A**
4. D	**9. B**
5 . A	**10. A**

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. Insufficency 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. Panzynorm
   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. Helminthicinvasion
C. Maldigestion
D. Dvsbacteriosisof 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 markers 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

1. A	**5. C**	**9. E**
2. E	**6. B**	**10. C**
3. D	**7. A**	
4. A	**8. E**	

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:

1. Davidson's "Principles and Practice of Medicine" 21st edition, Alimentary tract and pancreatic disease, p. 835-919.
2. Current Medical Diagnosis and Treatment, Gastrointestinal disorders, 2014, p. 564-662
3. Harrison's, Principles of Internal Medicine, 19th edition, Gastoenterology and Hepatology, p.257-398
4. V.G.Peredery, S.M.Tkach. Clinical lectures of internal diseases in 2 volumes. Kiev, the Manuscript, 1998.
5. Hvorostinka V. N. Jabluchansky Н.И, Panchuk С.Н, Pasieshvili Л.М. Therapeutic gastroenterology. Kharkov, the Basis, 1999, 368.
6. Degtyaryov I.I.Diseases of digestive organs. - K: People, 2000, - 412
7. S.M.Gandzha, V.M.Kovalenko, N.M.Shuba, etc. Internal diseases. K: I Am health ", 2002. - 992.
8. N.I.Shvets, A.V.Pida, T.M.Bentsa, etc. Standards of practical skills in therapy. Kiev, Glavmeddruk, 2005, 540.
9. N.I.Shvets, A.V.Pidaev, T.M.Bentsa, etc. Urgent conditions in clinic of internal medicine. Kiev, 2006. - 752 p.
10. Ostapenko V. G, Zhmudikov F.M. Acute diseases of abdomen. - m: Belarus, 1993. - 432 with.
11. Конден Р, Najhus R.Klinicheskaja Surgery - M. Practice, 1998. - 716 c.
12. Malaja L.T., Hvorostinka V. N. Therapy: Manual for students and doctors-interns. - X: the Fact, 2001.1032.
13. Hvorostinka V. M, Moisejennko T.A., Zhuravlova L.V. Faculty therapy: Manual-X.:Fact, 2000,-888.
14. Diseases of digestive organs: the Manual in schemes and tables / under edition of. V.M.Hvorostinka. - X: the fact, 2001.-239.
15. Malaja L.T., Hvorostinka V. N. Therapy. Diseases of digestive organs. Diseases of kidneys. Immunology. Allergic diseases. Systemic diseases of a connecting tissue. Diseases of blood system: the Management for doctors-interns and students. 2nd edition.-Kharkov: Folio, 2005.-879.

Інформаційні ресурси

сайт кафедри внутрішньої медицини № 3 ХНМУ http://www. vnmed3.kharkiv.ua/, встановлене інформаційно-освітнє середовище Moodle на піддомен сайта http://distance-training. vnmed3.kharkiv.ua

Методична вказівка складена: асистентом А.К. Журавльова

Методична вказівка переглянута і затверджена на засіданні кафедри: _____

З доповненнями (змінами) _____

Завідувач кафедри Л.В. Журавльова