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

Хронічні панкреатити

Харків 2016

KHARKOV NATIONAL MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL MEDICINE N3

METHODOLOGICAL RECOMMENDATIONS FOR STUDENTS

"Chronic pancreatitis"

Kharkiv 2016

Practical training: "Chronic pancreatitis", 5 hours

Chronic pancreatitis (CP) is persistent inflammation of the pancreas that results in permanent structural damage with fibrosis and ductal strictures, followed by a decline in exocrine and endocrine function. It can occur as the result of chronic alcohol abuse but may be idiopathic. Initial symptoms are recurrent attacks of pain. Later in the disease, some patients develop malabsorption and glucose intolerance. Diagnosis is usually made by imaging studies such as ERCP, endoscopic ultrasonography, or secretin pancreatic function testing. Treatment is supportive, with dietary modification, analgesics, and enzyme supplements. In some cases, surgical treatment is helpful.
The incidence of chronic pancreatitis is 4.8 new cases per 100 000 of population per year. Prevalence is 25 to 30 cases per 100 000 of population. Total number of patients with increased in the world by 2 times for the last 30 years. In Ukraine, the prevalence of diseases of the pancreas increased by 10.3%, and the incidence increased by 5.9%.
True prevalence rate of CP is difficult to establish, because diagnosis is difficult, especially in initial stages. The average time of CP diagnosis ranges from 30 to 60 months depending on the etiology of the disease.

Learning objectives:

- to teach students to recognize the main symptoms and syndromes of CP;
- to teach students to perform physical examination;
- to familiarize students with methods used for the diagnosis of CP, the determination of incretory and excretory pancreatic insufficiency, indications and contraindications for their use, methods of their execution, the diagnostic value of each of them;
- to teach students to interpret the results of conducted study;
- to teach students how to recognize and diagnose complications of CP;
- to teach students how to prescribe treatment for CP.

What should a student know?

- Frequency of CP;
- Etiological factors of CP;
- Pathogenesis of CP;
- Main clinical syndromes of CP, CP classification;
- General and alarm symptoms of CP;
- Physical symptoms of CP;
- Methods of physical examination of patients with CP;
- CP diagnostics, assessment to disorders of endocrine and exocrine pancreatic function;
- Diagnostic capabilities of esophagogastroduodenoscopy, excretory retrograde cholangiopancreatography, abdominal ultrasonography, computed tomography, plain radiography of the abdomen in patients with CP, indications, contraindications;
- Method of duodenal intubation to determine the exocrine pancreatic function, clinical evaluation of its results;
- H.pylori diagnostic methods;
- Methods of functional tests for the diagnostics of exocrine pancreatic insufficiency;
- Complications of CP;
- CP treatment (lifestyle modification, nutrition, drug therapy, surgery).

What students should be able to do?

- To define the main clinical and physical syndromes in CP;
- To interpret the results of clinical, biochemical and immune-enzyme assays;
- To interpret the data of esophagogastroduodenoscopy, excretory retrograde cholangoipancreatography, ultrasonography of the abdomen, CT, plain radiography of the abdomen;

- To interpret the data of duodenal contents analysis in order to determine the exocrine pancreatic function, and to provide clinical evaluation of results;
- To evaluate the functional tests for the diagnostics of exocrine pancreatic insufficiency;
- To be able to identify the types of exocrine pancreatic insufficiency;
- To prescribe treatment for patients with CP.

List of practical skills that students should possess:

- Inspection of skin and mucosa
- determination of presence of malabsorption syndrome;
- examination of the abdomen;
- Inspection of the abdomen;
- Superficial palpation of the abdomen;
- Methodological deep sliding palpation of the abdomen after the method of Obraztsov-Strazhesko;
- Determination of pain points and zones that are typical for CP;

Topics content

CHRONIC PANCREATITIS

Chronic pancreatitis is commonly defined as a continuing, chronic, inflammatory process of the pancreas, characterized by irreversible morphologic changes. This chronic inflammation can lead to chronic abdominal pain and/or impairment of endocrine and exocrine function of the pancreas. By definition, chronic pancreatitis is a completely different process from acute pancreatitis. In acute pancreatitis, the patient presents with acute and severe abdominal pain, nausea, and vomiting. The pancreas is acutely inflamed (neutrophils and edema), and the serum levels of pancreatic enzymes (amylase and lipase) are elevated. Full recovery is observed in most patients with acute pancreatitis, whereas in chronic pancreatitis, the primary process is a chronic, irreversible inflammation (monocyte and lymphocyte) that leads to fibrosis with calcification.

Epidemiology

In population studies, males are affected more commonly than females. Rates in males peak between ages 45 and 54 years and then decline; female rates reach a plateau, which remains stable after age 35 years. Alcohol-induced illness is more prevalent in males, idiopathic and hyperlipidemic-induced pancreatitis is more prevalent in females, and equal sex ratios are observed in chronic pancreatitis associated with hereditary pancreatitis.

Etiology

Causes of chronic pancreatitis (‘TIGAR-O’:)

- **Toxic-metabolic**
  Alcohol
  Tobacco
  Hypercalcaemia
  Chronic renal failure
- **Idiopathic**
  Tropical
  Early/late onset types
- **Genetic**
  Hereditary pancreatitis (cationic trypsinogen mutation)
  SPINK-1 mutation
  Cystic fibrosis

- **Autoimmune**
  Isolated or as part of multi-organ problem
- **Recurrent and severe acute pancreatitis**
  Post-necrotic
  Recurrent acute pancreatitis
- **Obstructive**
  Ductal adenocarcinoma
  Intraductal papillary mucinous neoplasia
  Pancreas divisum
  Sphincter of Oddi stenosis

Chronic pancreatitis occurs most often in patients with alcoholism (45–80% of all cases). The risk of chronic pancreatitis increases with the duration and amount of alcohol consumed, but pancreatitis develops in only 5–10% of heavy drinkers. Tobacco smoking is a risk factor for idiopathic chronic pancreatitis and has been reported to accelerate progression of alcoholic chronic pancreatitis. About 2% of patients with hyperparathyroidism develop pancreatitis. In tropical Africa and Asia, tropical pancreatitis, related in part to malnutrition, is the most common cause of chronic pancreatitis. A stricture, stone, or tumor obstructing the pancreas can lead to obstructive chronic pancreatitis. Autoimmune pancreatitis is associated with hypergammaglobulinemia (IgG4 in particular) and often with autoantibodies and other autoimmune diseases and is responsive to corticosteroids. Between 10% and 30% of cases of chronic pancreatitis are idiopathic, with either early onset (median age 23) or late onset (median age 62). Genetic factors may predispose to chronic pancreatitis in some of these cases and include mutations of the cystic fibrosis transmembrane conductance regulator (*CFTR*) gene, the pancreatic secretory trypsin inhibitory gene (*PSTI*, serine protease inhibitor, *SPINK1*), and possibly the gene for uridine 5-diphosphate glucuronosyltransferase.

Pathophysiology

The cause of chronic pancreatitis usually is metabolic in nature. The proposed pathologic mechanisms of chronic pancreatitis are as follows:

- Intraductal plugging and obstruction - Eg, ethanol (ETOH) abuse, stones, tumors
- Direct toxins and toxic metabolites - These act on the pancreatic acinar cell to stimulate the release of cytokines, which stimulate the stellate cell to produce collagen and to establish fibrosis; cytokines also act to stimulate inflammation by neutrophils, macrophages, and lymphocytes (eg, ETOH, tropical sprue)
- Oxidative stress - Eg, idiopathic pancreatitis
- Necrosis-fibrosis - Recurrent acute pancreatitis that heals with fibrosis
- Ischemia - From obstruction and fibrosis; important in exacerbating or perpetuating disease rather than in initiating disease
- Autoimmune disorders - Chronic pancreatitis has been found in association with other autoimmune diseases, such as Sjögren syndrome, primary biliary cirrhosis, and renal tubular acidosis.
- Secondary forms of autoimmune chronic pancreatitis are associated with primary biliary cirrhosis, primary sclerosing cholangitis, and Sjögren syndrome.
- While alcohol greatly influences the understanding of its pathophysiology because it is the most common etiology (60-70%), approximately 20-30% of cases are idiopathic and 10% of cases are due to rare diseases.

Whatever the etiology of chronic pancreatitis, pancreatic fibrogenesis appears to be a typical response to injury. This involves a complex interplay of growth factors, cytokines, and chemokines, leading to deposition of extracellular matrix and fibroblast proliferation. In pancreatic injury, local expression and release of transforming growth factor beta (TGF-beta) stimulates the growth of cells

of mesenchymal origin and enhances the synthesis of extracellular matrix proteins, such as collagens, fibronectin, and proteoglycans.

Clinical presentation

Clinically, the patient experiences intermittent attacks of severe pain, often in the midabdomen or left upper abdomen and occasionally radiating in a bandlike fashion or localized to the midback. The pain may occur either after meals or independently of meals, but it is not fleeting or transient and tends to last at least several hours. Unfortunately, patients often are symptomatic for years before the diagnosis is established; the average time from the onset of symptoms until a diagnosis of chronic pancreatitis is 5 years. The delay in diagnosis is even longer in people without alcoholism, in whom the average time is 7 years from onset of symptoms to diagnosis.
The natural history of pain in chronic pancreatitis is highly variable. Most patients experience intermittent attacks of pain at unpredictable intervals, while a minority of patients experience chronic pain. In alcohol-induced disease, eventual cessation of alcohol intake may reduce the severity of pain.
Pain is due to a combination of increased pressure within the pancreatic ducts and direct involvement of pancreatic and peripancreatic nerves by the inflammatory process. Pain may be relieved by leaning forward. Weight loss is common and results from a combination of anorexia, avoidance of food because of postprandial pain, malabsorbtion and/or diabetes. Steatorrhea occurs when more than 90% of the exocrine tissue had been destroyed; protein malabsorption only develops in the most advanced cases.
In most instances, the standard **physical examination** does not help to establish a diagnosis of chronic pancreatitis; however, a few points are noteworthy.
During an attack, patients may assume a characteristic position in an attempt to relieve their abdominal pain (eg, lying on the left side, flexing the spine and drawing the knees up toward the chest).
Occasionally, a tender fullness or mass may be palpated in the epigastrium, suggesting the presence of a pseudocyst or an inflammatory mass in the abdomen. Patients with advanced disease (ie, patients with steatorrhea) exhibit decreased subcutaneous fat, temporal wasting, sunken supraclavicular fossa, and other physical signs of malnutrition.

Diagnosis

- **Tests to establish the diagnosis**
  Ultrasound CT (may show atrophy, calcification or ductal dilatation)
  Abdominal X-ray (may show calcification)
  Magnetic Resonance Cholangiopancreatography (MRCP)
  Endoscopic ultrasound
- **Tests of pancreatic function**
  Collection of pure pancreatic juice after secretin injection (gold standard but invasive and seldom used)
  Fecal pancreatic elastase
- **Tests of anatomy prior to surgery**
  MRCP

Blood tests
Serum amylase and lipase levels may be slightly elevated in chronic pancreatitis; high levels are found only during acute attacks of pancreatitis. In the later stages of chronic pancreatitis, atrophy of the pancreatic parenchyma can result in normal serum enzyme levels because of significant fibrosis of the pancreas, resulting in decreased concentrations of these enzymes within the pancreas.
While low concentrations of serum trypsin are relatively specific for advanced chronic pancreatitis, they are not sensitive enough to be helpful in most patients with mild to moderate disease.

Laboratory studies to identify causative factors of chronic pancreatitis include serum calcium and triglyceride levels. When common etiologies are not found, research protocols are available to test for genetic mutations in cationic trypsinogen and *CFTR.*

Fecal tests

Because maldigestion and malabsorption do not occur until more than 90% of the pancreas has been destroyed, steatorrhea is a manifestation of advanced chronic pancreatitis. Neither qualitative nor quantitative fecal fat analysis can detect early disease.

Assays of fecal chymotrypsin and human pancreatic elastase-1 have the same limitations but are useful in confirming advanced chronic pancreatitis with exocrine insufficiency. These tests allow to determine or to rule out pancreatic exocrine insufficiency and its degree. The study of elastase-1 in stool allows to determine pancreatic insufficiency of moderate and severe cases is 95-100%.

Pancreatic Function Tests

Direct tests

These tests are the most sensitive and can be used to detect chronic pancreatitis at its earliest stage; however, they are somewhat invasive, labor intensive, and expensive.

- Determination in duodenal aspirates
- Determination in pancreatic juice

Indirect tests

Noninvasive tests of pancreatic function have been developed for detecting chronic pancreatitis. In principle, these tests work via oral administration of a complex substance that is hydrolyzed by a specific pancreatic enzyme to release a marker substance. The intestine absorbs the marker, which then is measured in the serum or urine. These tests are capable of detecting moderate to severe chronic pancreatitis. The presence of renal, intestinal, and liver disease may interfere with the accuracy of these tests.

Respiratory pancreatic tests:

^{13}C-triglyceride breath test - determines pancreatic lipase activity in the lumen of the intestine and can differentiate pancreatic steatorrhea from enteric steatorrhea.

Protein breath test with ^{13}C-marked egg white - reduced in case of CP - lack of trypsin.

Amylase (^{13}C-corn-starch) breath test – allows to detect deficiency of pancreatic amylase in duodenum (normal – in the end of 4th hour - 10-30%).

Neither currently is freely available.

Imaging

Plain films show calcifications due to pancreaticolithiasis in 30% of affected patients. CT may show calcifications not seen on plain films as well as ductal dilatation and heterogeneity or atrophy of the gland. Occasionally, the findings raise suspicion of pancreatic cancer ("tumefactive chronic pancreatitis"). Endoscopic retrograde cholangiopancreatography (ERCP) is the most sensitive imaging study for chronic pancreatitis and may show dilated ducts, intraductal stones, strictures, or pseudocyst, but the results may be normal in patients with so-called minimal change pancreatitis. MRCP (including secretin-enhanced MRCP) and endoscopic ultrasonography (with pancreatic tissue sampling) are less invasive alternatives to ERCP.

Differential diagnosis

- Ampullary carcinoma
- Cholangitis
- Cholecystitis
- Chronic gastritis
- Crohn disease
- Mesenteric Artery Ischemia
- Myocardial infarction
- Pancreatic cancer
- Peptic ulcer disease

Complications

- Pseudocysts and pancreatic ascites, which occur in both acute and chronic pancreatitis
- Extrahepatic obstructive jaundice due to a benign stricture of the common bile duct as it passes through the diseased pancreas
- Duodenal stenosis
- Portal or splenic vein thrombosis leading to segmental portal hypertension and gastric varices
- Peptic ulcer
- Diabetes mellitus

Treatment

No curative treatment for chronic pancreatitis exists. Medical therapy is determined primarily by symptoms. If no anatomic explanation for abdominal pain can be found, medical therapy can be attempted. This therapy includes pain control with analgesic agents and a trial of noncoated pancreatic enzymes.

The goals of medical treatment are as follows:

- Modify behaviors that may exacerbate the natural history of the disease
- Enable the pancreas to heal itself
- Determine the cause of abdominal pain and alleviate it
- Detect pancreatic exocrine insufficiency and restore digestion and absorption to normal
- Diagnose and treat endocrine insufficiency

Cessation of alcohol consumption and tobacco smoking are important. In early stage alcohol-induced chronic pancreatitis, lasting pain relief can occur after abstinence from alcohol, but in advanced stages, abstinence does not always lead to symptomatic improvement. Patients continuing to abuse alcohol develop either marked physical impairment or have a death rate 3 times higher than do patients who abstain.

A diet low in fat and high in protein and carbohydrates is recommended, especially in patients with steatorrhea. The degree of restriction depends on the severity of fat malabsorption; generally, an intake of 20 g/day or less is sufficient. Patients who continue to suffer from steatorrhea following fat restriction require medical therapy. Malabsorption of the fat soluble vitamins (A, D, E, and K) and vitamin B-12 may also occur. Oral supplementation of these enzymes is recommended.

Opioids should be avoided if possible. Preferred agents for pain are acetaminophen, nonsteroidal anti-inflammatory drugs (naproxen, diclofenac, ketorolac, ibuprofen) and tramadol, along with pain-modifying agents such as tricyclic antidepressants (amitriptyline hydrochloride**)**, selective serotonin uptake inhibitors, and gabapentin or pregabalin. Steatorrhea is treated with pancreatic supplements that are selected on the basis of their high lipase activity. A total dose of 40,000 units of lipase in capsules is given with meals. Higher doses may be required in some cases. The tablets should be taken at the start of, during, and at the end of a meal. Concurrent administration of a H2-receptor antagonist (eg, ranitidine, 150 mg orally twice daily), a proton pump inhibitor (eg, omeprazole, 20–60 mg orally daily), or sodium bicarbonate, 650 mg orally before and after meals, decreases the inactivation of lipase by acid and may thereby further decrease steatorrhea. In selected cases of alcoholic pancreatitis and in cystic fibrosis, enteric-coated microencapsulated preparations may offer an advantage. However, in patients with cystic fibrosis, high-dose pancreatic enzyme therapy has been associated with strictures of the ascending colon. Pain secondary to idiopathic chronic pancreatitis may be alleviated in some cases by the use of pancreatic enzymes (not enteric-coated) or octreotide, 200 mcg subcutaneously three times daily. Micronutrient therapy to correct electrophilic stress on key macromolecules in the pancreas by toxic metabolites has shown promise in early studies. Associated diabetes mellitus should be treated. Autoimmune pancreatitis is treated with prednisone 40 mg/d orally for 1–2 months, followed by a taper of 5 mg every 2–4 weeks. Nonresponse or relapse occurs in 45% of cases (particularly in those with concomitant IgG_4-

associated cholangitis); azathioprine appears to reduce the risk of relapse.

Control of initial knowledge level:

1. Patient complains on nausea, pain in right hypochondrium, diarrhea, and frequent abdominal distension. In anamnesis: systematic alcohol consumption. Objective data: subnutrition, tongue covered with white film, belly is soft, sensitive to palpation in paraumbilical area. Liver and spleen are not enlarged. Feces analysis: steatorrhea, creatorhhea. What diagnosis of the listed below is the most probable one?

A. Chronic hepatitis
B. Helminthiasis
C. Chronic recurrent alcoholic pancreatitis
D. Chronic enterocolitis
E. Chronic cholecystitis

2. Patient complains on the pain in the epigastrium and left subcostal area; repeated vomiting which doesn't bring relief, abdominal distention, diarrhea, weight loss. Objective data: tongue is wet, covered with white film near the root. During profound belly palpation an insufficient painfulness is found in epigastrium and Mayo-Robson's point. What disease should you think of in the first turn?

A. Ulcer
B. Chronic atrophic gastritis
C. Chronic pancreatitis
D. Chronic cholecystitis
E. Chronic enteritis

3. A female patient has been suffering from chronic pancreatitis during previous 5 years. She complains on frequent watery excrements, loss of 12 kg during 2 months. What syndrome does the patient have?

A. Astheno-neurotic
B. Malabsorption
C. Dyspeptic
D. Pain
E. Epigastric

4. A woman, 32 years old, complains on the pain in the left hypochondrium emerging in 2 hours after meal, nausea, abdominal distention, tendency to diarrhea. Objective data: subicteric sclera, painful belly during palpation in Gubergrits-Skulsky's point. The level of which enzymes should be determined?

A. Amylase
B. Lactate dehydrogenase
C. Creatine phosphokinase
D. Gammaglutamattranspeptidase
E. Aspartaaminotransferase

5. A patient complains on the pain in the upper right area of belly emerging in an hours after meal and irradiating to lumbus on the right side. During belly palpation there is painfulness in Chauffard's zone. What part of pancreas is damaged in this patient?

A. Head of pancreas
B. Body of pancreas
C. Tail of pancreas
D. Total pancreas damage
E. Focal damage

6. A patient with chronic pancreatitis has an increased level of blood serum glucose. What pancreatic function disorder does the patient have?
A. Exocrine
B. Endocrine
C. Absorption disorder
D. Acid-forming
E. Pepsinogenous

7. What hormones stimulate pancreatic activity?
A. Cholecystokinin-pancreozymin
B. Insulin
C. Thyrotropic
D. Counterinsular
E. Adrenalin

8. Which type of pancreatic juice secretion is the most typical for chronic pancreatitis?
A. Hyposecretory
B. Ductular
C. Upper obturative
D. Lower obturative
E. Hypersecretory

9. Which changes are typical for endocrine pancreatic insufficiency?
A. Jaundice
B. Nausea, vomiting
C. Hypoglycemic state, pancreatic diabetes development
D. Dyspeptic
E. Vitamin deficiency

10. Which clinic syndrome is associated with exocrine pancreatic disorder?
A. Pain
B. Maldigestion
C. Allergic
D. Epigastric
E. Right reactive vegetative

Correct answers:
1. C
2. C
3. B
4. A
5. A
6. B
7. A

8. A
9. C
10. B

Control of final knowledge level

1. Which of these etiologic factors prevail in formation of primary chronic pancreatitis?
   A. Alimentary factors
   B. Alcoholism
   C. Heredity
   D. Medication
   E. Allergy

2. Which pathogenetic mechanisms are responsible for the development of chronic pancreatitis of alcoholic genesis?
   A. Pancreatic juice composition change with deposition of protein lumps in ducts
   B. Production of antibodies to the gland tissue
   C. Reflux of duodenal content to Wirsung's ducts
   D. Reflux of bile to Wirsung's ducts
   E. Inflammatory changes in gland

3. What causes pain syndrome in case of chronic pancreatitis?
   A. Fibrosis formation in the gland
   B. Necrotic changes
   C. Rise of pressure in ducts
   D. Gland edema
   E. Duct blocking

4. Which food products can cause pain syndrome in case pancreatitis?
   A. Salty food
   B. Alcohol, sweets, cakes
   C. Solid protein food
   D. Acrid and fried food
   E. Milk products

5. What evidences of dyspeptic syndrome are typical for chronic pancreatitis?
   A. Bitter taste, heartburn
   B. Nausea, vomiting without relief, gaseous eructation
   C. One time vomiting with relief
   D. Heartburn, sour ecrutation, nausea
   E. Food eructation, bitter taste

6. Which clinical evidences are typical for exocrine pancreatic function disorder?
   A. Constipation
   B. Diarrhea with blood in feces
   C. Imperative urge to defecate
   D. Abdominal distention, constipation
   E. Meteorism, rumbling, more frequent defecation

7. Which irritators are used for generation of pancreatic juice?
   A. Histamine

B. Insulin
C. Aminophylline
D. Cholecystokinin-pancreozymin
E. Sulfurous magnesia

8. Which pancreatic enzyme is determined in blood of patients with chronic pancreatitis during the first hours?
A. Lipase
B. Tripsin
C. Chymotripsin
D. Nuclease
E. Amylase

9. Which pancreatic ferment stays increased during a long time when chronic pancreatitis exacerbates?
A. Tripsin
B. Lipase
C. Amylase
D. Nuclease
E. Chymotripsin

10. Which treatment method should be used during the first 2-3 days of chronic pancreatitis exacerbation?
A. Diet, spasmolytics, enzymes
B. Diet, M-cholinolytics, antienzymatic agents
C. Hunger, antienzymatic agents, H_2-histamine receptor blockers
D. Diet, alkali, spasmolytics
E. Diet, spasmolytics, enzymes, vitamins

Correct answers (final knowledge level):

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

Case-based questions

1. A female patient, 37 years old, complains on constant dull pain in hypochondrium with irradiation to the back that gets intensified after meals. Other complaints: abdominal distention, frequent defecation with indigested food. Objective data: moderate abdominal distention, painfulness in Chauffard's zone, Desjardin's point, Mayo-Robson's points. What study method is the most informative for proving of exocrine pancreatic insufficiency?
A. Esophagogastroduodenoscopy
B. Peroral cholecystography
C. Retrograde pancreatography
D. Coprogram

E. Ultrasonography of abdominal organs

2. A man, 45 years old, complaints of the aching pain in the left subcostal area, nausea, gaseous eructation, diarrhea after ingestion of fatty food, meteorism, abdominal rumbling, weight loss. He suffers about 25 years when he started noticing the pain in the left subcostal area, gaseous eructation because of improper food, and later, diarrhea, stinking fatty feces. Which preliminary diagnosis is the most probable?

A. Ulcer
B. Chronic gastritis
C. Chronic cholecystitis
D. Gastroesophageal reflux
E. Chronic pancreatitis

3. A man, 46 years old, complains on the severe pain in the upper half of the abdomen, mainly on the left, nausea, vomiting without relief on the 2-nd day after a banquet. Such condition is regularly observed after the diet problems. Objective data: temperature 37°C, pale and wet skin, pulse - 88 beats per 1 min, rhythmic, the belly is moderately distended, acute pain in the pancreas projection. CBC: L - $18*10^9/1$, urine diastase - 256 un. after Wolgemut. What would be the most correct management of this condition by general practitioner?

A. Urgent hospitalization to the surgery department
B. Ambulatory therapy
C. Home ward
D. Planned hospitalization to the therapeutics unit
E. Gastroenterologist consultation

4. A man, 35 years old, complaints on the permanent dull pain in the left hypochondrium after fatty and smoked food, vomiting without relief. Feces look shiny and malodorous. Patient have been suffering from this condition for 8 years, he drinks too much alcohol, smokes a lot. Objective data: reduced nutrition. Pale and dry skin. Foul tongue with white film. The belly is moderately inflated, pain in Chauffard's, Gubergrits-Skulsky's zone, Desjardin's, Mayo-Robson's points. Which disease should you think of first of all?

A. Chronic pancreatitis
B. Chronic cholecystitis
C. Ulcer
D. Chronic gastroduodenitis
E. Chronic enterocolitis

5. A woman, 48 years old, complains on the periodic pain in the right hypochondrium with irradiation to lumbar region, nausea after any kind of food, frequent fluid excrements. She lost 12 kg in 2 months. Objective data: reduced nutrition, soft belly, significant pain in the Desjardin's point. Liver goes beyond the costal margin for 1 cm, unpainful. Defecations 3 - 4 times a day with additives of neutral fat. Gastric analysis: hydrochloric acid - 30 un. Urine diastase - 16 un. Which pathology causes such picture most probably?

A. Gluten enteropathy
B. Chronic pancreatitis
C. Chronic hepatitis
D. Chronic enterocolitis
E. Autoimmune gastritis

6. A woman, 32 years old, complains on the pain in the left hypochondrium which occurs 2 hours after meal, nausea, abdominal distention, frequent liquid stool. Objectively: subicteric scleras, painful belly during palpation in the Gubergrits-Skulsky's point. Liver - on the edge of

costal margin. In blood: amylase - 288 g/h*l, total bilirubin- 20 micromol/1. What is the most possible diagnosis?

A. Chronic gastritis
B. Chronic hepatitis
C. Chronic enterocolitis
D. Chronic cholecystitis
E. Chronic pancreatitis

7. A patient suffers from chronic relapsing pancreatitis with marked exocrine function disorder. After fatty, spicy food, alcohol consumption a defecation with fatty excrements occurs. Deficiency of what factor is the most probable cause of steatorrhea?

A. Amylase
B. Trinsin
C. Gastric acidity
D. Д. Lipase
E. Alkaline phosphatase

8. A patient complains on the permanent pain in the upper half of the belly, more on the left, intensifying after meal. Other complaints: diarrhea, weight loss. Alcohol overusage. He had acute pancreatitis two years ago. Blood amylase - 4 mg (ml per hr.). Coprogram - steatorrhea, creatorrea. Blood sugar - 6,0 mmol/1. Which treatment is indicated for this patient?

A. Contrical
B. Insulin
C. Gastrozepinum
D. Д. Panzynorm forte
E. No-shpa

9. A patient, 62 years old, suffers from periodic episodic pain in the left hypochondrium with irradiation to the back during 32 years, due to that he had to follow a strict diet with the limitation of fatty, fried, spicy, smoked food. During the last 18 months he observes emerging of abdominal distention, more frequent defecations - 2 - 3 times a day. The excrements are thick, stinking, with shiny surface, residues of indigested food. The change of the patient's symptomatology is caused by:

A. Exocrine pancreatic insufficiency
B. Endocrine pancreatic insufficiency
C. Cholestatic syndrome
D. Irritable bowel syndrome
E. Pancreatic insufficiency

CORRECT ANSWERS

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

Control Questions:

1. Give the definition of the chronic pancreatitis.
2. Principal clinical syndromes in case of the chronic pancreatitis.
3. Characteristics of the physical examination of a patient with the chronic pancreatitis.
4. Name the methods of diagnostics of the chronic pancreatitis.
5. Name the evidences of exocrine and endocrine pancreatic insufficiency.
6. Name the types of exocrine pancreatic secretion disorders.
7. Give the classification of the chronic pancreatitis.
8. Name the characteristics of the chronic pancreatitis course.
9. Name the complications of the chronic pancreatitis.
10. Name the causes of the CP development and characteristics of the diseases depending on its etiologic factor.
11. Name the pathogenesis of the CP main forms.
12. Principles of the chronic pancreatitis treatment.
13. Lifestyle changes and diet therapy in case of the chronic pancreatitis.
14. Drug treatment of the chronic pancreatitis at different stages of the disease course.
15. Indications for surgery.
16. CP prevention.

Practical Tasks:

1. Supervise patients with chronic pancreatitis.
2. Interprete the obtained laboratory data.
3. Interprete the obtained data of instrumental study.
4. Write a prescription for the chronic pancreatitis treatment.

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. _____

Further reading:

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. Nair RJ, Lawler L, Miller MR. Chronic pancreatitis. *Am Fam Physician*. 2007;76:1679-1688.
5. Owyang C. Chronic pancreatitis. In: Goldman L, Ausiello D, eds. *Cecil Medicine*. 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 147.
6. Kumar P, Clark M. (eds.) (2005) Kumar & Clark Clinical medicine. 6th ed. Edinburgh: W B Saunders.
7. Keim V, Teich N, Moessner J. Clinical value of a new fecal elastase test for detection of chronic pancreatitis. Clin Lab 2003; 49:209.
8. Schmidt G. Pancreas. Differential diagnosis in ultrasound. A teaching atlas. Georg Thieme Verlag, Stuttgart, Germany 2006. p.146.
9. Bozkurt T, Braun U, Leferink S, et al. Comparison of pancreatic morphology and exocrine functional impairment in patients with chronic pancreatitis. Gut 1994; 35:1132.
10. Axon AT, Classen M, Cotton PB, et al. Pancreatography in chronic pancreatitis: international definitions. Gut 2004; 25:1107.
11. Zuccaro G Jr, Sivak MV Jr. Endoscopic ultrasonography in the diagnosis of chronic pancreatitis. Endoscopy 1992; 24 Suppl 1:347.
12. Wallace MB, Hawes RH, Durkalski V, et al. The reliability of EUS for the diagnosis of chronic pancreatitis: interobserver agreement among experienced endosonographers. Gastrointest Endosc 2001; 53:294.
13. Ooi CY, Gonska T, Durie PR, Freedman SD. Genetic testing in pancreatitis. Gastroenterology 2010; 138:2202.

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. Журавльовою

Методична вказівка переглянута і затверджена на засіданні кафедри: _____

З доповненнями (змінами) _____

Завідувач кафедри Л.В. Журавльова