Lecture complex

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Head Chair, candidate of medical sciences, acting associate professor Asanova G.K.

Lecture complex for the spring (VIII) semester
Lecture №1

1. Topic: Gastroesophageal Reflux Disease (GERD)

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-gastroenterology, to give a general idea about diseases of the digestive system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Gastroesophageal reflux disease (GERD) is common and occurs in 10-20% of adults.

Etiology

The appearance of reflux suggests the failure of the lower esophageal sphincter (NPS), which may be the result of a general decrease in sphincter tone or repeated transient relaxation of the NPS (not associated with swallowing). Transient NPS relaxation is caused by increased pressure in the stomach or subthreshold pharyngeal stimulation.

Factors that ensure the normal functioning of the gastroesophageal transition include the angle of the gastroesophageal transition, diaphragm contraction, and gravity (i.e., vertical position). Factors that can affect the occurrence of reflux include weight gain, fatty foods, caffeine, sodas, alcohol, tobacco smoking, and medications. Medications that lower the tone of NPS include anticholinergics, antihistamines, tricyclic antidepressants, calcium channel blockers, progesterone and nitrates.

Clinical manifestations

The most striking symptom of GERD is heartburn with or without regurgitation of the gastric contents in the oral cavity. Infants develop vomiting, irritability, anorexia, and sometimes signs of chronic aspiration. Adults and infants with chronic aspiration may experience coughing, hoarseness, or stridor.

Esophagitis can cause pain when swallowing and even esophageal bleeding, which is usually hidden, but can sometimes be massive. Peptic strictures cause gradually progressive dysphagia when eating solid foods. Peptic ulcers of the esophagus cause pain, as with a stomach ulcer or duodenal ulcer, but the pain is usually localized in the xiphoid process or in the high sternum. Esophageal peptic ulcers heal slowly, tend to recur and usually lead to strictures when healed.

Diagnostics

- Clinical diagnosis
- Endoscopic examination for the failure of empirical therapy
- 24-hour pH measurement with typical symptoms but no endoscopic changes

A detailed history usually indicates a diagnosis. Patients with typical signs of GERD may be given a trial of acid-suppressive therapy. If treatment is ineffective, the symptoms of the disease persist for a long time, or signs of complications, further examination of the patient is necessary.

Endoscopy with a cytological examination of a scraping with a mucous membrane and / or biopsy of the changed areas is the method of choice. An endoscopic biopsy is the only test that reliably detects the appearance of a cylindrical mucosal epithelium in Barrett's esophagus. Patients with dubious results of endoscopy and persisting symptoms, despite treatment with proton pump inhibitors, need to perform a 24-hour pH measurement. Although fluoroscopy with a sip of barium sulfate indicates esophageal ulcers and peptic stricture, this study is less informative for choosing a treatment method that reduces reflux; in addition, most patients with identified pathology require subsequent endoscopy.

Esophageal manometry is used to assess esophageal motility before surgical treatment.

Complications

GERD can lead to the development of esophagitis, peptic ulcer of the esophagus, strictures of the esophagus, Barrett's esophagus and adenocarcinoma of the esophagus. Factors contributing to the

development of esophagitis include the caustic nature of refluxate, the inability of the esophagus to remove it back, the volume of the gastric contents and the local protective properties of the mucous membrane.

Treatment

• Raising the head end of the bed
• Exclusion of coffee, alcohol, fatty foods, smoking
• Proton pump inhibitors, H2 blockers

It is recommended that patients who are overweight or recently overweight are recommended to reduce body weight.

Drug therapy often includes proton pump inhibitors; all of these drugs are equally effective. These drugs can be used for a long time, but the minimum dose necessary to prevent symptoms should be selected; Periodic or "as needed" reception is also allowed. H2-histamine receptor blockers are less effective, but they can be added to the course of proton pump inhibitors.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What are the main and additional complaints of patients with diseases of the digestive system?
2. What is GERD?
3. What are the complications of GERD?
4. What is palpation of the abdomen?
5. Describe the pain syndrome in diseases of the digestive tract.

Lecture №2

1. Topic: Peptic ulcer of the stomach and duodenum.

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-gastroenterology, to give a general idea about diseases of the digestive system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Peptic ulcer - the cyclic appearance of peptic ulcers in the stomach or duodenum. Peptic ulcer is a limited mucosal defect that extends deeper beyond the muscularis mucosa, with inflammatory infiltration and thrombotic necrosis in adjacent tissues. Peptic ulcers usually appear in the bulb of the duodenum and stomach, less commonly in the lower part of the esophagus or the loop of the duodenum.

Reasons: frequent - infection with Helicobacter pylori, NSAIDs; rare - in particular, treatment in ICU, Zollinger-Ellison syndrome (gastrinoma of the pancreas or duodenum), corticosteroids in combination with NSAIDs, other drugs (potassium chloride, bisphosphonates, mycophenolatemofetil).

H. pylori infection causes more than half the cases of duodenal ulcers and stomach ulcers.

Risk factors for damage to the mucous membrane of NSAIDs: peptic ulcer or ulcer bleeding, H. pylori infection, age> 60 years, the simultaneous use of several NSAIDs or in a large dose, the simultaneous use of GCS (the procerogenic effect of GCS is not proven) or anticoagulants.

Clinical picture and natural course

The main symptom is pain or discomfort in the epigastrium, which appears 1-3 hours after eating, passing after eating or antacids. Often appears at night or early in the morning. Epigastric pain is not very specific for peptic ulcers; in ≈50% of cases, the cause is another disease, most often functional dyspepsia. Nausea and vomiting may occur. Often an asymptomatic course.

Diagnostics

1. Endoscopy: a stomach ulcer is a sharply limited, round defect with a diameter of ≈1 cm or an irregularly shaped depression with infiltrated edges, most often in the region of the corner of the stomach or prepiloric region, usually single; multiple ulcers are often diagnosed after taking NSAIDs. In the duodenum, an ulcer is most often on the anterior wall of the bulb, usually with a diameter of <1 cm. An urgent indication for endoscopy is bleeding from the upper digestive tract.

2. Tests that detect H. pylori infection (before performing the test, in addition to serological test, cancel antibiotics and bismuth for ≥4 weeks, and PPI for 2 weeks). Indications for testing

1) invasive methods (requiring endoscopy):

a) a urease test (most often used), a biopsy specimen of the gastric mucosa is placed on a plate containing urea with the addition of a color indicator, the decomposition of the bacterial urease of urea to ammonia alkalizes the medium and leads to a change in its color (sensitivity and specificity of 95% when analyzing 2 biopsies) ;

b) histological examination of a biopsy of the mucous membrane;

c) the cultivation of bacteria.

2) non-invasive methods:

a) breath tests - the patient receives a portion of urea labeled with 13C or 14C, which is hydrolyzed by bacterial urease to CO2, which is determined in exhaled air;

b) a test that identifies H. pylori antigens in feces - studies are carried out in laboratories by an enzyme immunoassay using monoclonal antibodies (but not kits for quick diagnosis outside the laboratory) are as accurate as a breath test;

c) serological tests - a positive result does not indicate the presence of a current infection, since antibodies are observed for another year or longer after treatment, but they can be used during treatment of PPIs, as well as in patients with other factors that reduce the sensitivity of other tests: recently treated with an antibiotic, with a bleeding stomach ulcer, atrophic gastritis, or a neoplasm of the stomach. The diagnosis is established on the basis of endoscopic examination.

Differential diagnosis

Other causes of dyspepsia, nausea and vomiting, epigastric pain. To differentiate the nature of the gastric ulcer (benign or malignant), a histological assessment of ≥6 samples taken from the periphery and the bottom of the ulceration is necessary. A duodenal biopsy sample is indicated only if there is a suspicion of an etiology other than H. pylori infection.

Treatment

1. Diet: regular meals, with the exception of only dishes that cause or reinforce complaints. Limit the use of coffee and strong alcohol (although there is no evidence to help heal ulcers). Alcohol and nutrition do not affect peptic ulceration.

2. Quitting smoking: smoking makes it difficult to heal an ulcer and increases the risk of relapse.

Treatment for H. pylori infection

Treatment is indicated in any case of confirmed infection.

Treatment of patients with uninfected H. Pylori

Surgical treatment

Complications
1. Bleeding from the upper digestive tract.
2. Perforation
3. Pyloric stenosis
4. Illustrative material: presentation
5. Literature:
1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.
6. Security questions (feedback):
1. The main complaints in PUD and duodenum
2. When does pain occur in PUD and duodenum?
3. What are the reasons leading to the occurrence of PUD and duodenum
4. What diseases should be used for the diagnosis of pulmonary disease and duodenum.
5. What are the complications of PUD and duodenum.

Lecture № 3

1. Theme: Chronic Hepatitis
2. Purpose: To familiarize students with the introduction to the section of clinical medicine-gastroenterology, to give a general idea about diseases of the digestive system.
The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. Theses of the lecture:
The term "Chronic hepatitis" refers to diffuse inflammatory diseases of the liver, in which clinical, laboratory and morphological changes last 6 months or more.
By etiology and pathogenesis, the following forms of chronic hepatitis are distinguished:
• chronic viral hepatitis B,
• chronic viral hepatitis C,
• chronic viral hepatitis D,
• autoimmune hepatitis,
• drug hepatitis,
• cryptogenic chronic hepatitis.
In addition, a number of other liver diseases can have clinical, laboratory and histological signs of chronic hepatitis - this is Wilson-Konovalov's disease, alpha 1-antitrypsin deficiency, the initial stages of primary biliary cirrhosis, primary sclerosing cholangitis.
Chronic viral hepatitis B, C is an inflammatory liver disease caused by infection of the hepatitis B virus (HBV) or C (HCV), which can progress to cirrhosis. Chronic hepatitis B develops in approximately 5% of patients with icteric acute hepatitis. HCV infection accounts for 70% of cases of chronic viral hepatitis, 40% for liver cirrhosis, and 60% for hepatocellular carcinoma in the world. The main routes of transmission of HBV and HCV infections are parenteral (contact with blood or infected medical equipment), sexual, perinatal.

Chronic viral hepatitis B and C is characterized by clinical manifestations consisting of liver damage and / or signs of extrahepatic lesions. The first clinical symptoms often appear years or decades after infection. Asthenic, dyspeptic, icteric syndromes, enlarged liver and spleen are distinguished. Among extrahepatic manifestations, skin purpura, arthritis, myalgia, nephrotic and urinary syndromes and others are determined.

Autoimmune hepatitis is a chronic process in the liver of unknown etiology, the development mechanism of which is associated with the aggression of one's own immune system against components of the liver tissue.

Autoimmune hepatitis is characterized by a peri-portal or more extensive inflammatory process, the presence of hyper-g-globulinemia and tissue autoantibodies, which in most cases respond to immunosuppressive therapy. A genetic predisposition is considered as the main factor in the pathogenesis of autoimmune hepatitis. For the implementation of the process, triggering agents are needed - viruses, drugs and other environmental factors. Autoimmune hepatitis of 3 types is distinguished according to the profiles of detected autoantibodies. Autoimmune hepatitis is characterized by a wide range of clinical manifestations - from asymptomatic to severe, sometimes fulminant hepatitis with the presence or absence of extrahepatic signs. At the initial examination, clinical signs of liver cirrhosis are found in 25% of patients. Extrahepatic manifestations of autoimmune hepatitis - fever, skin vasculitis, arthralgia and arthritis, myalgia, polymyositis, lymphadenopathy, pleurisy, pericarditis, myocarditis, Hashimoto's thyroiditis, glomerulonephritis, ulcerative colitis, diabetes mellitus, and other hemolyte.

A1-antitrypsin deficiency is the first frequency congenital metabolic defect with an autosomal codominant type of inheritance, causing liver damage with cholestasis and cirrhosis in children.

Deficiency of the trypsin inhibitor α1-antitrypsin leads to an increase in the activity of proteases, which causes damage to the tissues of the lungs, pancreas, and kidneys. The mechanism of chronic liver damage with α1-antitrypsin deficiency is not fully understood, it is associated with the accumulation of α1-antitrypsin in the liver tissue.

A1-antitrypsin deficiency is clinically characterized by the following symptoms - hepatomegaly, less often splenomegaly, and jaundice at an early age. In some cases, pathology from the lungs, pancreas, and kidneys is revealed.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is hepatitis?
2. What are the causes of hepatitis?
3. Leading syndromes with hepatitis.
4. How are the borders of the liver determined by Kurlov?
5. What groups of drugs are included in the treatment program for hepatitis?
6. Indications for liver transplantation.

Lecture №4

1. Topic: Cirrhosis of the liver.

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-gastroenterology, to give a general idea about diseases of the digestive system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Liver cirrhosis is a chronic diffuse progressive liver disease. Anatomically characterized by fibrosis, the formation of regenerate nodes that violate lobular architectonics of the organ, and the restructuring of the intrahepatic vascular bed.

The etiological factors of cirrhosis are as follows:

• hepatitis B, C, D viruses;

• genetically determined metabolic disorders - hemochromatosis, Wilson-Konovalov's disease, α1-antitrypsin deficiency, type IV glycogenosis, galactosemia, hereditary tyrosinemia;

• prolonged intra- and extrahepatic cholestasis;

• violation of the venous outflow from the liver - Bad Chiari syndrome, veno-occlusive disease, etc .;

• toxins and drugs;

• bypass surgery on the intestine with the shutdown of a large part of the small intestine.

Liver cirrhosis is determined by two main clinical syndromes - hepatocellular insufficiency and portal hypertension, as well as cholestasis syndrome, the degree of activity of the hepatic process and various extrahepatic manifestations of the disease. Liver cirrhosis is characterized by a change in the size, shape and consistency of the liver - its deformation with a dense pointed edge, an increase and hardening of the spleen is noted, the presence of edematous ascites syndrome is possible. There are "small" hepatic signs - telangiectasias, palmar erythema, subcutaneous venous collaterals, gynecomastia are characteristic. Extrahepatic manifestations of cirrhosis can be very diverse - severe damage to the heart, kidneys, lungs, etc. The timely diagnosis of cirrhosis requires modern material and technical base, the experience of a pediatrician and surgeon.

Primary biliary cirrhosis is a chronic inflammatory cholestatic liver disease of unknown (presumably immune) etiology, leading to progressive irreversible destruction of small intralobular and septal bile ducts. Like other autoimmune diseases, primary biliary cirrhosis is associated with extrahepatic autoimmune syndromes - thyroiditis, collagen diseases, glomerulonephritis, ulcerative colitis. Clinically, primary biliary cirrhosis is manifested by hepatosplenomegaly, asthenic syndrome, pruritus, later jaundice, hyperpigmentation of the skin with xanthelasms and xanthomas.

Primary sclerosing cholangitis is a chronic cholestatic liver disease of a presumably autoimmune nature, characterized by inflammation and fibrosis of the intra- and extrahepatic bile ducts. The defeat of the bile ducts is irreversible and leads to severe cholestasis, the formation of cirrhosis and the development of liver failure. Primary sclerosing cholangitis can be complicated by bacterial cholangitis, strictures of the bile ducts, cholelithiasis, the risk of developing cholangiocarcinoma is high. Clinically, primary sclerosing cholangitis is characterized by astheno-vegetative manifestations, cutaneous pruritus, and jaundice. In more than 75% of patients, primary sclerosing cholangitis is combined with inflammatory bowel diseases: ulcerative colitis or Crohn's disease.

4. Illustrative material: presentation

5. Literature

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...

7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is liver cirrhosis?
2. What are the causes of cirrhosis?
3. How are Kurlov's liver boundaries determined?
4. What groups of drugs are included in the treatment program for hepatitis?
5. Indications for liver transplantation.

Lecture №5

1. Topic: Rheumatoid Arthritis

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-rheumatology, to give a general idea about systemic diseases.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Rheumatoid arthritis is a chronic inflammatory systemic autoimmune disease characterized by polyarthritis, which is based on chronic inflammation of the synovial membrane of the joint, leading to a violation of its function.

Rheumatoid arthritis (RA) affects people of all ages, but most often the disease develops at the age of 30-55 years. Among patients with rheumatoid arthritis there are approximately 2-3 times more women than men. In general, according to scientists, 0.5-2% of the adult population worldwide suffer from this disease. In Russia, rheumatoid arthritis affects 0.6% of the population. The incidence of RA is increasing annually.

Although the causes of rheumatoid arthritis are not fully understood, it is known that factors contributing to its development may be acute respiratory infections, influenza, tonsillitis or exacerbation of chronic infectious diseases; severe emotional stress, as well as hypothermia.

Typically, the disease affects the small joints of the fingers, wrists, feet and ankles; in some cases, later the disease also extends to the hip, shoulder and knee joints; usually the joints are affected symmetrically, and the manifestations of the disease can have a very different intensity. The onset of the disease is gradual, the wave-like, but steadily progressing: all new joints are involved with subsequent severe deformation - "rheumatoid wrist", "rheumatoid foot". Pain in the affected joints is especially aggravated in the second half of the night, in the morning and in the morning. The following symptoms may also be characteristic of RA - "morning stiffness" (sensation of "numb body and joints"), weakness, poor sleep and appetite, moderate fever, chills and weight loss. In addition to everything, various complications in the activity of internal organs add to joint damage over time, which can threaten the patient's life.

Diagnosis of the disease is based on clinical, instrumental and laboratory methods. In most patients with rheumatoid arthritis, autoantibodies are determined in the blood serum (for example, rheumatoid factor and / or antibodies to the cyclic citrulinated peptide). Elevated levels of acute phase indicators (ESR, C-reactive protein, fibrinogen) are detected. Using X-ray examination of the joints, it is possible to identify characteristic changes (erosion in the joints and narrowing of the joint spaces) of the joint surfaces and

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...

5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What are the main complaints of DBST?
2. What should be paid attention to during the general examination of patients with RA?
3. How is palpation of the joints?
4. What information does radiography of the joints provide?
5. What physical methods are used to examine patients with RA?

Lecture №6

1. Topic: Systemic lupus erythematosus

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-rheumatology, to give a general idea about systemic diseases.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Systemic lupus erythematosus (SLE)

A chronic autoimmune disease of unknown etiology is characterized by overproduction of organ-specific autoantibodies to various components of the cell nucleus with the development of immuno-inflammatory damage to tissues and internal organs.

Clinical picture and natural course

Women are sick 6-10 times more often than men. In 90% of cases, the disease affects young women of reproductive age (20–40 years), however, it can develop in both children and the elderly in both sexes. The disease may begin with nonspecific symptoms. Often common symptoms or symptoms prevail within a single system or organ. During periods of exacerbations and remissions, 10–40% of patients experience long-term (> 1 year) remissions or periods without exacerbations, however, in ≈70% of patients, despite the achievement of initial remission or low disease activity, exacerbations develop.

1. General symptoms: weakness and fatigue, subfebrile condition or fever, weight loss.
2. Lesions of the skin and mucous membranes:

1) acute skin form of lupus erythematosus
2) subacute skin form of lupus erythematosus
3) chronic skin form of lupus erythematosus (discoid lupus)
4) other non-specific skin changes
5) vascular changes

3. Damage to the musculoskeletal system
4. Kidney damage (lupus nephropathy
5. Respiratory system lesions
6. Lesions of the cardiovascular system
7. Lesions of the nervous system (neuropsychiatric lupus
8. Hematologic disorders
9. The defeat of the digestive tract

Diagnostics

1. Laboratory research
1) blood test
2) urinalysis
3) immunological studies
2. The study of musculoskeletal biopsy

Differential diagnosis

Mixed and undifferentiated connective tissue disease, Sjogren's syndrome, early RA, systemic vasculitis, APS; drug-induced SLE, (causes); fibromyalgia with the presence of ANA, proliferative diseases of the blood system (especially lymphomas), primary thrombocytopenic purpura, autoimmune anemia, infections. Erythema on the face sometimes should be differentiated from rosacea, seborrheic dermatitis, photodermatosis, dermatomyositis. Symptoms that differentiate systemic connective tissue diseases

Treatment

1. The primary goal is to prolong life, prevent organ damage and improve the quality of life associated with health (CHL), which can be achieved by controlling the activity of the disease and minimizing concomitant diseases and drug toxicity.
2. Distinguish treatment that induces remission, or - if remission is not achievable - the least activity of the disease (see Monitoring), as well as supportive treatment, which is aimed at preventing relapse of the disease.
3. Drugs: the main drugs are GCS. The simultaneous use of other immunomodulating and immunosuppressive drugs can reduce the dose of corticosteroids and increases the effectiveness of treatment. It should strive to use GCS in minimally effective doses or, if possible, completely abolish GCS.

4. Prevention of exacerbations:

1) to avoid being in direct sunlight;
2) to avoid taking drugs that cause drug-induced SLE;
3) the use of antimalarial drugs.

5. Additional activities:

1) prevention of osteoporosis
2) the fight against risk factors for cardiovascular diseases;
3) preventive vaccinations

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is systemic lupus erythematosus?
2. What are the causes of SLE?
3. What are the examination methods for SLE?
4. What diseases carry out diffdiagnosis with SLE?
5. What are the complications of SLE.

Lecture №7

1. Topic: Systemic scleroderma

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-rheumatology, to give a general idea about systemic diseases.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Systemic Scleroderma (SJS)

A systemic disease of connective tissue characterized by progressive fibrosis of the skin and internal organs (leading to their insufficiency), violations of the morphology and function of blood vessels, and disorders of the immune system. The etiology is unknown.

Women get sick 3-4 times more often than men. The peak incidence occurs between the ages of 30 to 50 years.

Clinical options

1. Limited form (OSSD; limited systemic sclerosis - lSSc; formerly called "CREST syndrome"): usually occurs in a chronic form, often without pronounced clinical manifestations; skin changes affect the face and distal parts of the upper and lower extremities; sclerotic skin changes tend to remain at a constant, usually average level of severity for many years; there is no correlation between the degree of sclerosis of the skin and damage to internal organs. Most often, the gastrointestinal tract is affected (especially the esophagus); less often, interstitial lung disease develops, and relatively rarely, heart damage; but more often than with DSSD, severe pulmonary arterial hypertension and primary biliary cirrhosis develop.

With a long-term course of OSDD, the onset and rapid increase in shortness of breath, especially with a sudden onset of right ventricular heart failure, usually indicates the development of pulmonary arterial hypertension and is associated with a poor prognosis.

2. Diffuse form (DSSD; diffuse systemic sclerosis - dSSc): proceeds much harder than the OSSD; characterized by a sudden onset; skin changes are symmetrical, diffuse, affect the face, proximal limbs and trunk (sometimes without involving the fingers); skin sclerosis usually progresses rapidly and peaks within 3–6 years. Almost simultaneously with skin sclerosis, organ changes develop: the lungs are most often affected, then the gastrointestinal tract, heart and kidneys. The rate of occurrence of organ changes and their severity correlate with the degree and prevalence of skin sclerosis. Changes in the internal organs that occurred at an early stage of DSSD (conditionally in the first 3 years of the disease) are decisive for the further course of the disease.

3. Systemic scleroderma without skin changes (systemic sclerosis sinescleroderma): typical symptoms from the systems and internal organs, with accompanying typical organ changes or serological disorders, without skin changes.

4. Cross syndrome is a combination of the clinical signs of systemic scleroderma with the symptoms of another systemic disease of the connective tissue, most often RA, dermatomyositis, SLE or SZST (Sharp's syndrome).

5. High-risk syndrome for the development of systemic scleroderma: Raynaud's syndrome, characteristic for SJS signs with capillaroscopy and specific for SJS ANA (ACA, ASKL-70-A or antibodies to the

nucleoli), but without skin sclerosis and organ changes; 65–80% of people with this syndrome develop SJS over the course of 5 years (mainly SJS).

Organ changes and symptoms

1. Raynaud's syndrome
2. Skin changes
3. Damage to the musculoskeletal system
4. The defeat of the digestive tract
5. Changes in the respiratory system
6. Heart damage
7. Kidney damage

Diagnostics

1. Laboratory research
2. Visualization studies:

WG of the hands
WG gastrointestinal tract study with contrast)
WG and chest CTVR
Doppler echocardiography

3. Endoscopy of the upper gastrointestinal tract
4. Functional studies of the respiratory system
5. Capillaroscopy of the nail rollers
6. Others: exercise tests
7. Skin biopsy

Differential diagnosis

With Raynaud's syndrome of a different etiology, other systemic diseases of the connective tissue, mainly undifferentiated disease of the connective tissue, SZST, cross syndromes, DM, RA.

Treatment

General principles

1. Etiological treatment
2. In order to improve or maintain functional ability (including the prevention of contractures) → physiotherapeutic procedures and kinesitherapy (gymnastics, before which paraffin compresses are often used), occupational therapy.

Early Treatment of DSSD
Treatment of Raynaud's syndrome, ulcers and necrosis of the digital phalanges
Treatment of interstitial lung disease
Pulmonary Hypertension Treatment
Treatment of joint and muscular changes
Treatment of disorders in the digestive tract
Heart disease treatment

Forecast

Depends on the presence and extent of changes in the internal organs. More than half of deaths in patients with SJS are associated with pulmonary fibrosis, pulmonary arterial hypertension, and heart damage. The remaining causes of death are primarily infections, neoplasms and cardiovascular complications that are not directly related to SJS.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...

4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is systemic scleroderma?
2. What are the causes of SSD?
3. What are the survey methods for SJS?
4. What diseases are diagnosed with in case of SJS?
5. What are the complications of SJS?

Lecture №8

1. Topic: Dermatomyositis

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-rheumatology, to give a general idea of systemic diseases.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Dermatomyositis (DM) is a systemic inflammatory disease of the connective tissue, occurring with a predominant lesion of the striated muscles. DM is a form of myositis with concomitant dermatitis. The etiology is unknown. It is believed that autoimmune mechanisms play a major role in the pathogenesis of DM.

Clinical picture

DM is one of the most common idiopathic inflammatory myopathies in adults. Women get sick 2 times more often than men. The disease can occur at any age, the peak incidence occurs at the age of 10-15 years (infant form) and 35-65 years. The onset of the disease can be acute (several days), subacute (weeks), or chronic (months, years). Most untreated patients develop slow muscle atrophy and contracture. 5-year mortality is ≈50%. (increased risk of cancer of the ovary, breast, lung, stomach, intestines, nasal cavity and throat, pancreas, bladder, and non-Hodgkin lymphomas).

1. General symptoms: weakness, fever, weight loss.
2. Symptoms of muscle damage:

1) the predominantly symmetrical weakness of the muscles of the shoulder girdle and / or pelvic girdle, as well as the muscles of the neck and back (in almost all adults), which causes difficulties in movement (getting up from a sitting position, walking up stairs, holding and carrying various objects, even when combing hair, etc.), the muscles often become sensitive and painful;
2) weakness of the respiratory muscles - leads to respiratory failure;
3) weakening of the muscles of the throat, esophagus and larynx - causes dysphagia and dysphonia;
4) damage to the muscles of the eyeball (rarely) - nystagmus, visual impairment.

3. Skin changes: found in DM; their appearance and amplification are not always associated with muscle damage; they can be ahead of myositis or develop on their own (CADM, dermatomyositis sine myositis [dermatomyositis without myositis]). Erythematous changes are often accompanied by itching of the skin and / or increased sensitivity to sunlight.

1) erythema around the eyes in the form of "glasses", with a violet color (the so-called heliotropic), sometimes with swelling of the eyelids - a pathognomonic symptom, occurs in 30-60% of patients; "Erythema decollete" in the form of the letter V; in addition, erythema of the posterior surface of the neck and shoulders (a symptom of a “scarf”), erythema of the lateral surface of the thighs and hips (a symptom of a “holster”);

2) Gottron papules - bluish papules with epidermal hypertrophy located on the extensor surface of the joints of the hands (interphalangeal and metacarpophalangeal); sometimes carpal, elbow, knee and ankle joints; Gottron's symptom is erythematous or bluish spots of the same localization (pathognomonic symptoms, in ≈70% of patients);

3) others - coarsening, peeling and cracking of the skin on the pads of the fingers and the palmar surface of the hands (the so-called hands of a mechanic, rarely); erythema with edema, petechiae and telangiectasias in the area of the nail ridges; trophic ulcers due to vasculitis of the vessels of the skin; generalized erythroderma; inflammation of the subcutaneous tissue (panniculitis); mesh lead; focal alopecia without scarring.

4. Heart damage: in 70% of patients revealed tachycardia or bradycardia, rarely symptoms of heart failure.

5. Damage to the lungs: symptoms of interstitial lung disease (30–40%), mainly dry cough and increasing shortness of breath, over time - chronic respiratory failure; aspiration pneumonia can develop in patients with severe dysphagia.

6. Gastrointestinal tract lesion: symptoms of impaired motility of the esophagus, stomach and intestines, including gastroesophageal reflux; in severe cases, ulcers and bleeding.

7. Damage to the joints: symptoms of non-erosive arthritis or arthralgia, especially peripheral, mainly in the joints of the hands (20-50%).

8. Calcifications - in the subcutaneous tissue, skeletal muscle, fascia and tendons (in> 10% of patients), sometimes massive.

9. Raynaud's syndrome (in 10-15% of patients with PM / DM).

Diagnostics

1. Laboratory research

1) biochemical analyzes - an increase in the activity of enzymes in blood serum - cpk, ast, alt, ldh, aldolases (normal enzyme values do not exclude pm / dm), as well as an increase in the level of myoglobin, esr, crp and gamma globulins;

2) immunological studies

2. Electromyography: reveals signs of primary muscle damage.

3. Histological examination:

1) muscle biopsy

2) lung biopsy examination

4. Imaging studies:mri of muscles, wg and ctec of the chest, wg of bones and joints

Differentialdiagnosis

Dm should be performed with myositis for other systemic diseases of the connective tissue (layering syndromes), as well as for oncological diseases, autoimmune necrotic myopathy (clinically consistent with pm, often associated with systemic autoimmune diseases, viral infection [eg, hiv], statins or oncopathology ;

Treatment

1. Gcs: prednisone n / a 1 mg / kg / day; acute onset or severe course → intravenous methylprednisolone 0.5–1.0 g can be used for 3 days. After improvement (muscle growth, decreased signs of muscle

damage), but not earlier than after 4-8 weeks. After the start of treatment, the daily dose of the oral form of gcs should be gradually reduced to a maintenance dose of 5-10 mg / day. And continue treatment for several years, and sometimes for life.
2. If within 6 weeks. From the beginning of treatment, there is no improvement in the course of the disease or it progresses → one of the drugs should be addedForecast
With proper treatment,> 80% of patients survive 10 years. The prognosis worsens in the presence, including the elderly, of damage to internal organs, especially lungs, a malignant tumor, anti-srp antibodies.
4. Illustrative material: presentation
5. Literature:
1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.
6. Security questions (feedback):
1. What is dermatomyositis?
2. What are the causes of dermatomyositis?
3. What are the examination methods for dermatomyositis?
4. What diseases are used for the differential diagnosis of dermatomyositis?
5. What are the complications of dermatomyositis.

Lecture №9

1. Topic: Anemia
2. Purpose: to familiarize students with the introduction to the section of clinical medicine-hematology, to give a general idea about the diseases of the hematopoietic system.
The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. Theses of the lecture:
Anemia is a decrease in the concentration of hemoglobin (hb), hematocrit (ht) and the number of red blood cells in the blood by> 2 standard deviations from the norm.
Separation by severity:
1) mild - hb 10-12.0 g / dl in women, 13.5 g / dl in men;
2) moderate - hb 8–9.9 g / dl;
3) severe - hb 6.5–7.9 g / dl;
4) life-threatening - hb<6.5 g / dl.
Causes: loss of red blood cells due to bleeding (acute or chronic), hemolysis, or a decrease or violation of erythropoiesis.
The main mechanisms (pathogenetic options): a decrease in the mass of circulating red blood cells (in acute blood loss), iron deficiency (in chronic blood loss), impaired iron utilization by erythroid cells (hereditary and acquired etiological factors), or redistribution of iron into cells of the macrophage system (in case of anemia of chronic diseases) deficiency of vitamin b12 and folic acid, hemolysis (hereditary and acquired etiological factors), violation of erythropoiesis in the bone marrow (aplastic anemia, le ycoses, myelofibrosis, myelodysplastic syndrome).

Objective and subjective symptoms: regardless of the cause and type of anemia - weakness, fatigue, impaired concentration and attention, headache, dizziness, tachycardia and shortness of breath (in severe form), pallor of the skin and mucous membranes, ictericity (with hemolytic anemia).

Iron-deficiency anemia

Anemia caused by impaired heme synthesis due to iron deficiency in the body is characterized by a decrease in the volume of red blood cells with a low hemoglobin content (microcytic hypochromic anemia). The most common (80%) form of anemia.

Causes of iron deficiency:

1) chronic blood loss (the main cause) from the gastrointestinal tract (including due to the use of asa and other nsaids, colon cancer, cancer of the stomach, gastroesophageal reflux disease, gastric and duodenal ulcer, hemorrhoids, angiodysplasia), birth canal, urinary tract (hematuria), respiratory system (diffuse alveolar bleeding), trauma (including surgery), with uterine bleeding (menorrhagia, fibroids, endometriosis), nosebleeds (hereditary hemoragicheskaya telangiectasia), from multiple blood donors;
2) increased need for insufficient intake - puberty, pregnancy (ii and iii trimester) and lactation, increased erythropoiesis in the treatment of b12 hypovitaminosis;
3) malabsorption from the gastrointestinal tract - condition after gastrectomy, bariatric surgery, gastritis caused by h. Pylori, autoimmune gastritis (≈20 years before the development of deficiency vit. B12), celiac disease and condition after intestinal resection, low-protein diet, enriched substances that impair the absorption of iron (phosphates, oxalates, phytins, tannin);
4) nutritional deficiency (vegetarian or vegan diet);
5) iron deficiency anemia resistant to iron therapy (rare, autosomal recessive inheritance).

Clinical picture

1. General symptoms of anemia
2. Symptoms of prolonged iron deficiency (some patients may be absent): taste perversion (clay, chalk, starch), pain, burning sensation and smoothing of the surface of the tongue, dry skin, angulitis (jams in the corners of the mouth), changes in nails (pale, brittle , with longitudinal grooves) and hair (thin, brittle, with forked tips, easily falling out).
3. Symptoms of the underlying disease (eg, colon cancer, etc.).

Diagnostics

1. A general analysis of peripheral blood: a decrease in the level of hb; hypochromic red blood cells of various sizes (anisocytosis), including microcytic, of various shapes (poikilocytosis); leukopenia (in ≈10% of patients; usually with significant iron deficiency); normal or increased platelet count.
2. Iron metabolism, a reduced serum ferritin concentration (<12 ng / ml) is the most informative indicator of iron deficiency in the absence of an active inflammatory process (acute phase protein).
3. Other studies: to determine the cause of iron deficiency

1) endoscopic examination of the upper and lower gastrointestinal tract
2) in case of contraindications to endoscopic studies, the use of imaging research methods is recommended;
3) screening tests for celiac disease (antibodies to tissue transglutaminase or anti-endomysial antibodies) - in all patients;
4) general analysis of urine - in all patients with the goal of eliminating red blood cells;
5) blood hidden in the feces

Differential diagnosis

Other anemia, especially hypochromic and chronic anemia

Treatment

It consists in eliminating the causes of iron deficiency, its replenishment and normalization of the level of hb and ferritin. If necessary (signs of hypoxia of the myocardium, brain), transfuse em.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback)

1. What is anemia?
2. What are the causes of anemia?
3. What types of anemia do you know?
4. List the causes of iron deficiency.
5. What diseases should be used for differential diagnosis of anemia?

Lecture №10

1. Topic: Acute Leukemia (OL)

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-hematology, to give a general idea about the diseases of the hematopoietic system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Acute leukemia is a disease based on the formation of a clone of malignant (blast) cells that have a common progenitor cell. Blasts infiltrate the bone marrow, gradually displacing normal hematopoietic cells, which leads to a sharp inhibition of hematopoiesis. Blast infiltration of internal organs is also characteristic of many types of leukemia.

Acute leukemia is divided into lymphoblastic (ALL) and myeloid (AML). It is believed that the occurrence of acute leukemia can be caused by the following factors:

• unidentified (most often);
• hereditary:
1. Down syndrome
2. bloom syndrome
3. Fanconi anemia
4. ataxia-telangiectasia
5. Klinefelter syndrome
6. imperfectosteogenesis
7. Wiskott's syndrome - Aldrich
8. leukemia in twins
• chemical:
1. benzene
2. alkylating agents (chlorambucil, melphalan)
• radiation exposure
• predisposing hematological disorders (myelodysplasia, aplastic anemia)
• HTLV-I viruses that cause T-cell leukemia and lymphoma in adults.

Five-year survival depends on the type of leukemia and the age of the patients:

• ALL in children - 65 - 75%;

• ALL in adults - 20 - 35%;
• AML in patients younger than 55 years old - 40-60%;
• AML in patients older than 55 years - 20%.

Classification

The differences between ALL and AML are based on the morphological, cytochemical, and immunological features of these types of leukemia. Accurate determination of the type of leukemia is of primary importance for therapy and prognosis. Both ALL and AML, in turn, are divided into several options according to the FAB classification (French-American-British). So, there are three options for ALL - L1, L2, L3 and seven options for AML:

• M0 - undifferentiated AML;
• M1 - myeloid leukemia without cell maturation;
• M2 - myeloid leukemia with incomplete cell maturation;
• M3 - promyelocytic leukemia;
• M4 - myelomonocytic leukemia;
• M5 - monoblastic leukemia;
• M6 - erythroleukemia;
• M7 - megakaryoblastic leukemia.

In accordance with the expressed antigens, ALL is divided into T-cell and B-cell types, which, depending on the degree of maturity, include several subtypes (pre-T-cell, T-cell, early pre-B-cell, pre-B-cell B-cell). There is no clear correlation between morphological and immunophenotypic variants, except that the L3 morphology is characteristic of b-cell leukemia.

Prevalence

ALL most often occurs at the age of 2–10 years (peak at 3–4 years), then the prevalence of the disease decreases, but after 40 years, a second rise is noted. ALL makes up about 85% of leukemia found in children. AML, in contrast, is most common in adults, and its frequency increases with age.

Clinical manifestations

Clinical manifestations of leukemia are caused by blast infiltration of the bone marrow and internal organs. Anemia is manifested by pallor, lethargy, shortness of breath. Neutropenia leads to various infectious complications. The main manifestations of thrombocytopenia are spontaneous hematomas, bleeding from the nose, uterus, injection sites, and gums. Bone pain, lymphadenopathy, hepatosplenomegaly are also characteristic. Possible breathing difficulties due to the presence of mediastinal masses, enlarged testicles, meningeal symptoms. In AML, gum hypertrophy occurs.

Patient Examination

Complete blood count: a decrease in hemoglobin and platelet count is possible; the content of leukocytes is from less than 1.0 • 109 / l to 200 • 109 / l, their differentiation is impaired, blasts are present.

The coagulogram can be changed, especially with promyelocytic leukemia, when in blast cells there are granules containing procoagulants.

A biochemical blood test for high leukocytosis may indicate renal failure.

A chest x-ray reveals the mediastinal masses that are found in 70% of patients with T-cell leukemia.

Bone marrow puncture: hypercellularity with a predominance of blasts.

Immunophenotyping is the determining method in distinguishing between ALL and AML.

Cytogenetic and molecular studies can detect chromosomal abnormalities, such as the Philadelphia chromosome (the product of the translocation of part of the 9th chromosome to the 22nd; determines a poor prognosis for ALL).

Lumbar puncture is used to detect damage to the central nervous system (neuroleukemia).

Treatment

All patients with suspected or established leukemia should be referred to specialized hospitals for examination and treatment as soon as possible.

Supportive therapy involves transfusion of platelets, red blood cells, freshly frozen plasma, antibiotic therapy of infectious complications.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is acute leukemia?
2. What are the causes of OL?
3. What types of acute leukemia do you know?
4. What treatment programs for OL do you know?
5. What diseases should be used for differential diagnosis of OL?

Lecture №11

1. Topic: Chronic leukemia (CL)

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-hematology, to give a general idea about the diseases of the hematopoietic system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Leukemia (leukemia) is a malignant disease of white blood cells. In chronic leukemia, tumor cells resemble normal cells, but differ from them. They live too long and interfere with the formation of certain types of white blood cells.

Lymphocytic and myeloid leukemia got their name in accordance with the cells from which they arose.

Chronic lymphocytic leukemia (lymphocytic leukemia) is the most common type of leukemia in Europe and North America. It accounts for 30% of all leukemia. The annual incidence of CLL in these countries is 3-3.5 per 100 thousand people, and among people over 65 years old - up to 20 per 100 thousand people.

About 70% of patients fall ill between 50 and 70 years of age. The average age at the onset of the disease is 55 years. Only less than 10% fall ill before the age of 40.

Men are sick 2 times more often than women.

Chronic myeloid leukemia is about 20% of all leukemia. In countries of Europe and North America, in terms of the frequency of spread of CML, it takes 3rd place after acute leukemia and CLL. The annual incidence is 1-1.5 per 100 thousand people in all countries and has remained practically stable over the past 50 years. Men get sick more often than women, accounting for 55-60% of patients. Half of the patients fall ill between the ages of 30-50 years, most often between 30-40 years. In children, typical CML is rare, accounting for no more than 1-2% of cases of childhood leukemia.

Causes of chronic leukemia and the possibility of preventing it

Currently, several risk factors associated with the development of chronic leukemia are known. Thus, exposure to high doses of radiation during an atomic bomb explosion or accident at a nuclear reactor increases the risk of chronic myeloid leukemia, but not chronic lymphocytic leukemia.

Prolonged contact with herbicides or pesticides among rural residents may increase the risk of chronic lymphocytic leukemia.
High voltage transmission lines may be a risk factor for leukemia.
Most patients with leukemia have not identified risk factors, so there is no way to prevent this disease. The exception is smoking, which increases the risk of leukemia.

Diagnosis of chronic leukemia

Currently, methods for the early detection of chronic leukemia have not yet been developed.
50% of patients with chronic leukemia do not have any symptoms at the time of detection of the disease. In these patients, the disease is diagnosed according to a blood test performed for another reason.
Common symptoms of chronic leukemia may include fatigue, weakness, weight loss, fever, and bone pain. Most of these symptoms are associated with a decrease in blood cell count.
Anemia (anemia) occurs as a result of a decrease in the number of red blood cells, which leads to shortness of breath, increased fatigue and pallor of the skin.
A decrease in the number of normal white blood cells increases the risk of infectious diseases. In patients with leukemia, the number of leukocytes can be significantly increased, however, these tumor cells do not protect against infection.
The spread of leukemia from the bone marrow to other organs and the central nervous system can lead to headache, weakness, convulsions, vomiting, and visual impairment.
Leukemia can be accompanied by an increase in lymph nodes, liver and spleen.

Diagnostic Methods

Blood analysis.

Blood chemistry

A bone marrow examination makes it possible to establish a diagnosis of leukemia and evaluate the effectiveness of treatment.
Spinal puncture allows you to identify tumor cells in the cerebrospinal fluid and carry out treatment by administering chemotherapy.
In order to clarify the type of leukemia, special research methods are used: cytochemistry, flow cytometry, immunocytochemistry, cytogenetics and molecular genetic research.
X-ray examinations of the chest and bones reveal a lesion of the lymph nodes of the mediastinum, bones and joints.
Computed tomography (CT) makes it possible to detect damage to the lymph nodes in the chest cavity and abdomen.
Magnetic resonance imaging (MRI) is especially indicated in the study of the brain and spinal cord.
Ultrasound examination (ultrasound) allows you to distinguish between tumor and cystic formations, to identify damage to the kidneys, liver and spleen, lymph nodes.

Chronic Leukemia Stages

Depending on the degree of spread of the disease in most malignant tumors, a stage is determined - from 1 to 4.
However, leukemia is a systemic disease in which, at the time of diagnosis, there is a lesion of the bone marrow and other organs, therefore, with leukemia, the stage is not determined.
To assess the prognosis (outcome) of the disease, other characteristics that affect the choice of treatment tactics are taken into account.
Chronic leukemia treatment
Treatment of patients with chronic leukemia depends on the type of disease and prognostic factors.
The drug method is the main one in the treatment of chronic leukemia.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...

2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is CL?
2. What are the causes of CL?
3. What types of CL do you know?
4. What are the instrumental types of diagnosis of CL.
5. What diseases should be used for differential diagnosis of CL?

Lecture № 12

1. Topic: Hypothyroidism

2. Purpose: To familiarize students with the introduction to the section of clinical medicine - endocrinology, to give an idea of diseases of the endocrine system.

The lecture contains data on the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Theses of the lecture:

Hypothyroidism - A clinical syndrome caused by a deficiency of thyroxine (T4) and the subsequent insufficient exposure to triiodothyronine (T3) in the cells of the body, which leads to a total slowdown of metabolic processes and the development of interstitial edema, as a result of the deposition of fibronectin and hydrophilic glycosaminoglycans in the subcutaneous tissue, muscles and other tissues.

The following types of hypothyroidism are distinguished:

1) primary - as a result of damage to the thyroid gland; causes: chronic autoimmune thyroiditis (most often Hashimoto's thyroiditis); other types of thyroiditis; total or subtotal thyroidectomy (the development of an autoimmune process that damages the remaining thyroid parenchyma is possible); congenital hypothyroidism;

2) secondary - due to deficiency or absence of TSH secretion, due to a decrease in pituitary function (neoplasm of the Turkish saddle, inflammatory or infiltrative diseases, damage: vascular origin, traumatic or iatrogenic - radiation, neurosurgical operations);

3) tertiary - due to the absence or deficiency of thyroliberin (TRH [TRH]) caused by damage to the hypothalamus (neoplasm, inflammatory-infiltrative diseases [eg, sarcoidosis]), or as a result of a violation of the integrity of the pituitary leg.

Clinical picture and natural course

With secondary and tertiary hypothyroidism, clinical symptoms are usually less pronounced than with primary hypothyroidism, but signs of insufficiency of other endocrine glands may appear (attention should be paid to symptoms of adrenal insufficiency), symptoms of diabetes insipidus, or other signs directly associated with hypopituitarism.

Subclinical hypothyroidism

Typical symptoms are not detected, but depressed mood, depression may be present, and with additional studies, an increase in the concentration of total cholesterol and the LDL fraction in plasma. If, together with an increase in the concentration of TSH, the concentration of antibodies to thyroid peroxidase (AT-TPO) increases, the risk of developing manifest hypothyroidism increases by 2 times.

Manifest hypothyroidism

1. General symptoms: weight gain, weakness, fatigue and decreased exercise tolerance, drowsiness, general lethargy (psychomotor and speech), a feeling of cold, chilliness.
2. Skin changes: the skin is dry, cold, pale, with a yellowish tint, decreased sweating, hyperkeratosis, for example. on the elbows; edema of the subcutaneous tissue (the so-called mucous edema), which is manifested by gross facial features, characteristic swelling of the eyelids and hands; dry, brittle and thinned hair, sometimes eyebrow loss.
3. Changes in the cardiovascular system: bradycardia, decreased filling of the pulse, decreased sonority of heart sounds; increased shadow of the heart; arterial hypotension, rarely hypertension.
4. Changes in the respiratory system: hoarse, hoarse voice (thickening of the vocal cords, tongue enlargement); decrease in depth and frequency of respiration; signs of inflammation of the upper respiratory tract or, in severe cases, symptoms of respiratory failure.
5. Changes in the digestive tract: chronic constipation, in severe cases, bowel obstruction; ascites (in the late stages of the disease; usually then effusion is also detected in the pericardium and pleural cavity).
6. Changes in the urinary system: decreased water excretion (violation of glomerular filtration is a significant problem due to the risk of developing water intoxication); if there is no apparent puffiness, then these violations are considered minor.
7. Changes in the nervous system: mononeuropathies (eg carpal tunnel syndrome), paresthesia, decreased reflexes, and sometimes hearing loss.
8. Changes in the musculoskeletal system: decreased muscle strength and easy fatigability, bradykinesia, muscle cramps, muscle pain; swelling of the joints, especially the knee (thickening of the synovial membrane and effusion).
9. Changes in the reproductive system: in women, menstrual irregularities (polymenorrhea, heavy menstrual flow), infertility, miscarriages; in men, weakening of libido and sometimes erectile dysfunction.
10. Mental disorders: decreased ability to concentrate, memory impairment, subclinical or manifest depression, emotional lability, sometimes symptoms of bipolar affective disorder or paranoid psychosis; in extreme cases, dementia and coma.

Diagnostics

1. Hormonal studies:

1) serum TSH concentration - increased with primary hypothyroidism (main criterion), decreased with secondary and tertiary;
2) low concentration of free T4 (FT4) in serum;
3) the concentration of free T3 (FT3) in serum - quite often within normal limits, sometimes reduced;
4) the concentration of TSH in serum during the test with TSH (rarely performed): with primary hypothyroidism - excessive secretion of TSH, with a secondary - the absence of a significant increase in TSH, with a tertiary - a moderate and delayed increase.

2. Other laboratory tests:

1) an increased concentration of antithyroid antibodies (mainly AT-TPO) with the development of autoimmune thyroiditis;
2) increased concentrations of total cholesterol and LDL, as well as triglycerides;
3) anemia;
4) sometimeshyponatremia and slight hypercalcemia.

3. Imaging research methods: ultrasound of the thyroid gland,
Ultrasound of the abdominal cavity
Chest WG
echocardiography
thyroidscintigraphy

4. ECG: sinus bradycardia, low voltage of teeth, especially ventricular complexes, flattening or inversion of T waves, lengthening of the PQ interval, rarely complete AV block, prolongation of the QT interval.

Treatment

Long-term replacement therapy

Manifest hypothyroidism is an absolute indication for replacement therapy, usually throughout life. At the initial stage of treatment in case of tachycardia, especially in persons with concomitant heart disease (if there are no contraindications), it is advisable to prescribe β-blockers (eg propranolol).

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Security questions (feedback):

1. What is hypothyroidism?
2. What are the causes of hypothyroidism?
3. What types of goiter do you know?
4. What are the instrumental types of diagnosis of hypothyroidism.
5. What diseases should be used for differential diagnosis of hypothyroidism?

Lecture №13

1. Topic: Chronic rheumatic heart disease.

2. Purpose: To introduce students to the Department of Clinical Medicine - Endocrinology, to give an idea about diseases of the cardiovascular system.

The lecture provides information about the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. Abstracts of lectures:

Rheumatism is a systemic inflammatory connective tissue disease caused by group A hemolytic streptococcus that occurs in genetically predisposed people and includes the heart and joints in the pathological process.

Classification

Classification of the Russian Association of Rheumatologists (2003):

1. Clinical variants: acute rheumatic fever, secondary rheumatic fever.
2. Clinical manifestations.
3. Major: carditis, arthritis, chorea, erythema annulare, subcutaneous rheumatic nodules.
4. Additional: fever, arthralgia, abdominal syndrome, serositis.
5. Completion: healing; chronic rheumatic heart disease (without heart failure, heart failure).
6. Stage of insufficiency.

Risk factors: humid climate, unfavorable socio-economic situation, genetic predisposition.

Diagnostic criteria

Presence of two major or one major and 2 minor criteria for B-hemolytic streptococcal infection (positive culture of A-streptococcus from the body or positive test for A-streptococcal antigen, high or increasing anti-streptococcal ASL-O, anti-DNase B). antibody titer indicates a high probability of the disease.

The onset of the disease is associated with a previous streptococcal infection of the nasopharynx, accompanied by symptoms of poisoning, arthralgia with limitation of active and passive movements, large joints, volatility and symmetry of the lesion are more often affected. In acute rheumatic fever: fever manifests itself in 90% of patients, subcutaneous nodules, annular erythema, chorea, rheumatic heart disease with cardialgia, shortness of breath, orthopnea, arrhythmia, with auscultation - a characteristic noisy appearance for a known heart disease.

Complaints and anamnesis: pain and discomfort in the region of the heart, shortness of breath, palpitations, tachycardia.

Physical examination:

1. The main criteria are carditis, polyarthritis, chorea, erythema annulare, subcutaneous rheumatic nodules.
2. Minor criteria - clinical: arthralgia, fever.

Instrumental research:

1. ECG: slow conduction, decrease in T amplitude and S-T interval in the precordial regions, arrhythmias.
2. Signs of mitral and aortic insufficiency with Doppler echocardiography.
3. Radiography of the heart: an increase in the volume of the heart, a decrease in contractility.

Indications for consultation with a specialist: according to indications.

List of basic and additional diagnostic measures

List of main diagnostic measures:

1. Complete blood count.
2. General analysis of urine.
3. Determination of Le-cells - according to indications.
4. Coagulogram according to indications.
5. Determination of total proteins.
6. Determination of protein fractions.
7. Determination of C-reactive protein.
8. Electrocardiography.
9. Determination of streptokinase.
10. X-ray of the heart in 3 projections with contrast enhancement of the esophagus.
11. ECHOCG (DOPPLER ECHOCG).

List of additional diagnostic measures: bacteriological examination of a smear from the skin.

Laboratory research:

1. CKD: increased ESR, leukocytosis, shift of the leukocyte formula to the left.
2. CBA: increased levels of α2- and γ-globulins, seromucoids, haptoglobin, fiboin.
3. II blood: decrease in the number of T-lymphocytes, decrease in the function of T-suppressors, increase in the level of immunoglobulins and the titer of antistreptococcal antibodies.

Treatment tactics

Treatment of rheumatism and rheumatism is carried out in a hospital. In the outpatient period, secondary prevention of acute rheumatic fever (ARF) is carried out - benzathine - benzylpenicillin:

1. For patients with CKD without carditis (polyarthritis, chorea) - 5 years.
2. In CKD patients with carditis, prophylaxis should be carried out for more than 5 years (at least until the patient reaches 25 years of age).
3. Patients with valvular heart disease and patients with surgical correction of heart disease - for life.

Purpose of treatment:

1. Prevention and (dispensary control) relapses of acute rheumatic fever and rheumatic heart failure.
2. Return of bacteremia that occurred during treatment.

Treatment without drugs: treatment table number 10.

Medical treatment:

1. Antibacterial therapy.
2. Antibiotics are effective in the treatment of acute rheumatic fever after acute tonsillitis (Grade A).
3. When treating the oral cavity, esophagus and respiratory tract.
4. Amoxicillin* 2 g orally 1 hour before treatment. If it is impossible to ingest ampicillin 2 g intravenously or intramuscularly 30 minutes before treatment.
5. If you are allergic to penicillin - azithromycin * 500 mg or clarithromycin * 500 mg or clindamycin 600 mg.
6. Clindamycin* 600 mg IV or cefazolin* if allergic to penicillin and ingestion is not possible.
7. In the treatment of the gastrointestinal and genitourinary tracts.
8. Amoxicillin* 2 g orally 1 hour before treatment. If oral administration is not possible, ampicillin* 2 g IV or IM 30 minutes before treatment.
9. In case of allergy to penicillin vancomycin* 1 g intravenously over 1-2 hours, complete administration 30 minutes before treatment.
10. Non-steroidal anti-inflammatory drugs (NSAIDs): diclofenac sodium*.
11. The introduction of glucocorticoids in severe carditis, especially with symptoms of heart failure: prednisolone * (0.5-2 mg / kg of body weight) every 6-12 hours, then after 2 days the daily dose is increased to 120-160 mg. After that, at a normal erythrocyte sedimentation rate, it is reduced by 5 mg every 2 days for a week. A therapeutic dose of aspirin is prescribed before the abolition of prednisolone to prevent complications and 2 weeks after the abolition.
12. Sanitation of the source of infection.
13. Symptomatic therapy.
14. Treatment of arrhythmia: in case of atrial fibrillation: digoxin, calcium or amiodarone antagonists*, according to indications - cardioselective B-blockers: under the control of heart rate, blood pressure, ECG*.
15. Heart failure: diuretics: furosemide - the dose is selected depending on the degree of cardiac decompensation, with edematous syndrome - veroshpiron * 100-300 mg / day.
16. Potassium preparations: asparkam*, panangin*.
17. Treatment of patients with an artificial mitral or aortic prosthesis: indirect anticoagulants - phenylin * - the dose is selected depending on the IPT (75% less), warfarin * 2.5-5 mg / day - initial dose, control of the level of INR (2 ,8 -4 ,4) - 1 time per month.

List of essential and complementary medicines

List of essential medicines:

1. *Benzathine-benzylpenicillin powder for injection in a vial of 1,200,000 units, 2,400,000 units.
2. * Amoxicillin + clavulanic acid tablets, its shell is 500 mg/125 mg, 875 mg/125 mg.
3. * Diclofenac potassium 12.5 mg, tab.
4. Diclofenac sodium 25 mg, tablets for external use 1% gel.

List of additional drugs:

1. *Prednisolone 5 mg, tab.
2. * Digoxin 0.025 mg, tab.
3. *Amiodarone 200 mg, tab.
4. * Furosemide 40 mg, tab.
5. * Asparkam 0.5 g, tab.
6. *Warfarin 2.5 mg, tab.

Treatment effectiveness indicators:

1. Prevention of recurrence of ENT and exacerbation of rheumatic heart disease.
2. Prevention of bacteremia caused by drug treatment.

* - drugs included in the list of essential (essential) medicines.

Indications for hospitalization: complications of acute rheumatic fever. With complications of acute rheumatic fever, all patients are hospitalized, 2-3 tbsp. activity, 1 tbsp. within 2 weeks. ineffectiveness of treatment during activity, the occurrence of complications.

Primary Prevention:

- a complex of individual and social measures aimed at preventing primary morbidity (physical education, raising the standard of living, improving housing conditions);
- early and effective treatment of other acute streptococcal diseases of the upper respiratory tract and pressure in order to prevent a primary attack of rheumatism.

Preventive action:

If a patient has rheumatic heart disease, secondary prevention of bacterial endocarditis is carried out several times.

Prevention: benzathine benzylpenicillin * 2.4 ml intramuscularly 1 time in 3 weeks.

Secondary prevention: benzathine-benzylpenicillin D - 2.4 million units 1 time in 3 weeks - during the year. Principles of further management, clinical examination: prevention of a recurrent ENT attack and exacerbation of rheumatic heart disease.

4. Illustrative material: presentation

5. Literature:

1. Harrison's Principles of Internal Medicine. Joseph Loscalzo. ...
2. Pocket Medicine. ...
3. Davidson's Principles and Practice of Medicine. ...
4. Macleod's Clinical Examination. ...
5. Bates' Guide To Physical Examination and History Taking. ...
6. Step-Up to Medicine. ...
7. CURRENT Medical Diagnosis and Treatment. ...
8. Goldman-Cecil Medicine.

6. Control questions (feedback):

1. What is chronic rheumatic heart disease?
2. What are the causes of chronic rheumatic heart disease?
3. What is the CRHD clinic?
4. What are the instrumental types of CRHD?
5. How is the differential diagnosis of CRHD performed?