Lecture complex

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Lecture № 1

1. Topic: Acute and chronic bronchitis

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

Acute bronchitis - acute diffuse inflammation of the mucous membrane (endobronchitis) or the entire wall of the bronchi (panbronchitis).

The etiology of acute bronchitis is a series of pathogenic factors affecting the bronchi:

1) physical: hypothermia, inhalation of dust

2) chemical: inhalation of acid and alkali vapors

3) infectious: viruses - 90% of all acute bronchitis (rhinoviruses, adenoviruses, respiratory syncytial viruses, influenza), bacteria - 10% of all acute bronchitis (Mycoplasma pneumoniae, Chlamydia pneumoniae, Bordetella pertusis, Streptococcus pneumoniae) and their associations.

The main etiological factor is infectious, the rest play the role of a trigger. There are also predisposing factors: smoking, alcohol abuse, heart disease with congestion in the pulmonary circulation, the presence of foci of chronic inflammation in the nasopharynx, oral cavity, tonsils, genetic inferiority of the bronchial mucociliary apparatus.

The pathogenesis of acute bronchitis:

Adhesion of pathogens on the epithelial cells lining the trachea and bronchi + decrease in the effectiveness of local protective factors (the ability of the upper respiratory tract to filter inhaled air and release it from rough mechanical particles, change air temperature and humidity, cough and sneeze reflexes, mucociliary transport) Þ pathogen invasion Þ hyperemia and edema of the bronchial mucosa, desquamation of the cylindrical epithelium, the appearance of mucous or mucopurulent exudate Þ further violation of mucociliary clearance Þ edema with lining of the bronchi, hypersecretion of the bronchial glands Þ development of the obstructive component.

Classification of acute bronchitis:

1) primary and secondary acute bronchitis

2) according to the level of damage:

a) tracheobronchitis (usually against the background of acute respiratory diseases)

b) bronchitis with a primary lesion of the bronchi of medium caliber

c) bronchiolitis

3) according to clinical symptoms: mild, moderate and severe

4) as bronchial patency: obstructive and non-obstructive

Clinic and diagnosis of acute bronchitis.

If bronchitis develops against the background of acute respiratory viral infections, first there is hoarseness of the voice, sore throat when swallowing, a feeling of rawness behind the sternum, an irritating dry cough (manifestations of tracheitis). The cough is aggravated, may be accompanied by pain in the lower chest and behind the sternum. As the inflammation in the bronchi subsides, the cough becomes less painful, profuse mucopurulent sputum begins to separate. Symptoms of intoxication (fever, headaches, general weakness) vary greatly and are determined more often by the causative agent of the disease (with adenovirus infection - conjunctivitis, with parainfluenza virus - hoarseness of the voice , with the influenza virus - high fever, headache and scarce catarrhal phenomena, etc.).

Laboratory data is not specific. Inflammatory changes in the blood may be absent. When cytological examination of sputum, all fields of view cover white blood cells and macrophages.

Treatment of acute bronchitis.

1. Home mode, plentiful drink
2. Mucolytic and expectorant drugs
3. In the presence of bronchial obstructive syndrome: short-acting beta-agonists
4. In uncomplicated acute bronchitis, antimicrobial therapy is not indicated;
5. Symptomatic treatment (NSAIDs, etc.).

Chronic bronchitis (CB) is a chronic inflammatory disease of the bronchi accompanied by persistent cough with sputum separation of at least 3 months a year for 2 or more years, while these symptoms are not associated with any other diseases of the bronchopulmonary system, upper respiratory tract or other organs and systems.

Allocate HB:

a) primary - an independent disease that is not associated with damage to other organs and systems, often has a diffuse character

b) secondary - etiologically associated with chronic inflammatory diseases of the nose and paranasal sinuses, lung diseases, etc., is more often local.

Etiology of chronic bronchitis:

1) smoking:
2) inhalation of polluted air
3) the impact of occupational hazards
4) damp and cold climate - contributes to the development and exacerbation of CB.
5) infection
6) acute bronchitis
7) genetic factors and hereditary predisposition

The pathogenesis of chronic bronchitis.

1. Violation of the function of the local bronchopulmonary defense system and the immune system
2. Structural adjustment of the bronchial mucosa
3. The development of the classical pathogenetic triad

The clinical picture of chronic bronchitis.

Subjectively:

1) cough
2) sputum removal
3) shortness of breath

Objectively:

1) upon examination, significant changes are not detected; during the period of exacerbation of the disease, sweating, an increase in body temperature to subfebrile numbers can be observed.
2) percussion clear pulmonary sound, with the development of emphysema - box sound.
3) auscultation of exhalation, hard breathing (“roughness”, “unevenness” of vesicular breathing), dry rales (due to the presence of viscous sputum in the lumen of the bronchi, in large bronchi - bass low tone, in middle bronchi - humming, in small bronchi - wheezing) . If there is liquid sputum in the bronchi, moist rales (in large bronchi, coarse-bubbly, in medium bronchi, medium-bubbly, in small bronchi, fine-bubbly). Dry and wet rales are unstable, can disappear after vigorous coughing and sputum discharge.

Diagnosis of chronic bronchitis.

1. Laboratory data:

a) UAC
b) sputum analysis
c) TANK

2. Instrumental research:

a) bronchoscopy
b) bronchography
c) radiography of the lungs
d) study of the function of external respiration (spirography, peak flowmetry) Complications of chronic obesity.
1) directly caused by infection: a) pneumonia b) bronchiectasis c) bronchial obstruction syndrome d) bronchial asthma
2) due to the evolution of bronchitis: a) hemoptysis b) pulmonary emphysema c) diffuse pneumosclerosis d) respiratory failure e) pulmonary heart.
Treatment of CB is different in the period of remission and in the period of exacerbation.
1. During remission
2. During the period of exacerbation:
a) etiotropic treatment
b) pathogenetic treatment
- mucolytic and expectorant drugs
- therapeutic bronchoscopy with sanitation of the bronchi
- bronchodilators
- drugs that increase the body's resistance
c) symptomatic treatment
The outcome of chronic bronchitis: in obstructive form or chronic kidney disease with damage to the distal lung, the disease quickly leads to the development of pulmonary failure and the formation of a pulmonary heart.
4. Illustrative material: presentation
5. Literature: indicated in syllabus
6. Security questions (feedback):
1. What is bronchitis?
2. What are the complaints of acute bronchitis.
3. What are the causes of bronchitis.
4. What are the syndromes of bronchitis.
5. What are the signs of attention during auscultation of the patient?

Lecture № 2:
1. Topic: Pneumonia.
2. Purpose: To familiarize students with the introduction to the section of clinical medicine-pulmonology, to give a general idea about respiratory 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:
Pneumonia is a form of acute respiratory infection affecting the lungs. The lungs consist of small sacs called alveoli, which when breathing in a healthy person are filled with air. With pneumonia, the alveoli are filled with pus and fluid, which makes breathing painful and limits the flow of oxygen.
Pneumonia is the single most important infectious cause of child deaths worldwide. In 2015, 920 136 children under 5 years old died from pneumonia, which is 15% of all deaths of children under 5 years old worldwide. Pneumonia is ubiquitous, but children and families suffer the most from the disease in South Asia and sub-Saharan Africa. Pneumonia can be prevented with simple measures; it can be treated with simple, inexpensive drugs with proper care.
Causes

Pneumonia is caused by a number of pathogens, including viruses, bacteria and fungi. The most common are:

• streptococcuspneumoniae is the most common cause of bacterial pneumonia in children;
• haemophilusinfluenzae type b (Hib) - the second most common cause of bacterial pneumonia;
• respiratory syncytial virus is a common cause of viral pneumonia;
• In HIV-infected children, one of the most common causes of pneumonia is pneumocystis jiroveci. These microorganisms result in at least one quarter of all deaths of HIV-infected children from pneumonia.

Transmission of infection

There are several ways pneumonia can spread. Viruses and bacteria, which are usually present in the baby's nose or throat, can infect the lungs when they are inhaled. They can also spread by dropping by coughing or sneezing. In addition, pneumonia can be transmitted through the blood, especially during childbirth or immediately after it. Additional studies are needed to study the various pathogenic microorganisms that cause pneumonia and their transmission pathways, as this is crucial for treatment and prevention.

Symptoms

Symptoms of viral and bacterial pneumonia are similar. However, the symptoms of viral pneumonia may be more diverse than the symptoms of bacterial pneumonia.

In children under the age of 5 years with symptoms of coughing and / or shortness of breath, accompanied or not accompanied by high fever, pneumonia is diagnosed with rapid breathing or retraction of the lower chest, if the chest is retracted or retracted when inhaling (in a healthy person when you inhale the chest expands). Wheezing is more common with viral infections.

Risk factors

Although most healthy children are able to cope with the infection through the defenses of their own body, children with impaired immune systems are at greater risk of developing pneumonia. A child's immune system can be weakened by malnutrition or malnutrition. This applies especially to infants who are not exclusively breastfed.

Treatment

Bacterial pneumonia can be treated with antibiotics. A preferred antibiotic is amoxicillin in dispersible tablets. They are usually prescribed at a medical center or hospital, but in the vast majority of cases, pneumonia in children can be effectively treated at home with inexpensive oral antibiotics. Hospitalization is recommended only in very severe cases.

Prevention

Prevention of pneumonia in children is one of the main components of a strategy to reduce child mortality. Immunization against Hib, pneumococcus, measles and whooping cough is the most effective way to prevent pneumonia.

Combating environmental factors such as indoor air pollution (for example, by using affordable, environmentally friendly stoves) and creating hygiene rules in crowded homes also reduces the number of children with pneumonia.

4. Illustrative material: presentation
5. Literature: indicated in syllabus
6. Security questions (feedback):

1. What are the main and additional complaints of patients with respiratory diseases?
2. What is pain and cough?
3. What are the types of chest.
4. Describe chest percussion.
5. What is pneumonia?
3. What are the causes of pneumonia?

4. What are the syndromes of pneumonia.

Lecture № 3:

1. Topic: Chronic obstructive pulmonary disease (COPD).

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

Chronic obstructive pulmonary disease (COPD) is a progressive life-threatening lung disease that causes shortness of breath (initially with physical exertion) predisposing to exacerbation and serious illness.

Chronic obstructive pulmonary disease is a lung disease characterized by chronic restriction of air flow in the airways. Symptoms of COPD worsen over time. Dyspnea during exercise gradually turns into dyspnea at rest. This disease is often not diagnosed and can be life threatening. Previously, COPD was often called the terms "chronic bronchitis" and "emphysema."

Risk factors

The main cause of COPD is tobacco smoke (including inhalation of second-hand tobacco smoke, or second-hand smoke). Other risk factors include:

• indoor air pollution (for example, from the use of solid fuels for cooking and heating);
• air pollution;
• the presence of dust and chemicals in the workplace (fumes, irritants and fumes);
• frequent infections of the lower respiratory tract in childhood.

Who is at risk?

Men have been more likely to suffer from COPD in the past, however, because women in high-income countries smoke as much as men and women in low-income countries are more affected by pollution indoor air (for example, as a result of using solid fuel for cooking and heating), today the disease affects men and women in almost equal proportions.

Over 90% of COPD deaths occur in low- and middle-income countries, where effective strategies to prevent and control the disease are not always or not always available.

Symptoms

Chronic obstructive pulmonary disease develops slowly and, as a rule, manifests itself in people older than 40-50 years. The most common symptoms of COPD are shortness of breath ("lack of air"), chronic cough, and sputum discharge. As the state of health worsens, the patient may have difficulties even with the implementation of ordinary daily activities, such as climbing a small flight of stairs or carrying a suitcase. In addition, patients often experience exacerbations, i.e. serious episodes of severe shortness of breath, coughing, and sputum lasting from several days to several weeks. These episodes can lead to a marked reduction in disability and the need for emergency medical care (including hospitalization), and sometimes death.

Diagnosis and treatment

Typically, a suspicion of chronic obstructive pulmonary disease occurs in people with the symptoms described above. The diagnosis can be confirmed by a breath test called spirometry, which allows you to measure how much air a person can exhale at a time, with maximum effort, and how quickly.

Chronic obstructive pulmonary disease is incurable. However, available medications and physiotherapy can alleviate symptoms, increase exercise capacity and improve quality of life, and reduce the risk of death. The most effective and cost-effective treatment for COPD in smokers is smoking cessation. This will slow down the course of the disease and reduce mortality from causes

associated with COPD. In some (but not all) patients with COPD, the administration of inhaled corticosteroids gives a positive effect.
4. Illustrative material: presentation
5. Literature: indicated in syllabus
6. Security questions (feedback):
1. What are the main respiratory sounds.
2. What is COPD?
3. What are the reasons leading to the development of COPD.
4. What research methods are needed to diagnose COPD?
5. What are the complications of COPD.

Lecture number 4:
1. Topic: Bronchial asthma.
2. Purpose: To familiarize students with the introduction to the section of clinical medicine-pulmonology, to give a general idea about respiratory 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:
Bronchial asthma (AD) is a heterogeneous disease, usually characterized by chronic inflammation of the respiratory tract and the presence of symptoms such as wheezing, shortness of breath, constriction in the chest and cough, with a variable frequency and intensity associated with varying degrees of expiratory flow disturbance air through the airways. The restriction of air flow is caused by: spasm of smooth muscles and swelling of the mucous membrane of the bronchi, the formation of mucous plugs, and over time, also with the restructuring of the bronchial wall. In terms of etiology, asthma is divided into allergic (most often it begins in childhood, often accompanied by other atopic diseases, usually eosinophilia of induced sputum and a good response to inhaled GCS) and non-allergic (usually in adults, often the worst response to inhaled GCS). In addition, BA phenotypes are distinguished:
1) with a late onset;
2) with constant bronchial obstruction;
3) accompanied by obesity.
In allergic asthma, the binding of an allergen to specific IgE antibodies on the surface of mast cells releases mediators (including histamine, proteolytic enzymes, cysteine leukotrienes) that cause bronchial obstruction. In some cases, 6–8 hours after the early phase of an allergic reaction, a late phase occurs in which mast cells, basophils and other cells release cytokines and chemokines, greatly enhancing the influx of inflammatory cells, in particular, eosinophils, into the bronchi. The pathogenesis of non-allergic asthma is not fully understood, but the histopathological picture is similar to allergic asthma. Damage to the bronchial epithelium stimulates repair processes, resulting in a restructuring of the bronchial wall, which leads to the fact that in especially severe cases, the obstruction becomes irreversible.
Factors causing asthma attacks and exacerbations or provoking their persistence: allergens, respiratory tract infections (mainly viral), air pollution (including tobacco smoke, aerosols used in the household, paint fumes, etc.), physical activity , strong emotions, weather changes, drugs (β-blockers, NSAIDs), food and nutritional supplements.
Factors that increase the risk of exacerbations of AD: uncontrolled symptoms of AD (including excessive consumption of short-acting β2-agonists (monthly> 1 pack containing 200 doses), improper use of inhaled GCS (non-compliance with the patient's prescribed drug, incorrect inhalation technique) low FEV1 (especially <60% of the due), serious psychological or socio-economic problems, exposure to tobacco smoke or allergens (in sensitized people), concomitant diseases (obesity, inflammation

mucous membranes of the nose and paranasal sinuses, food allergy), eosinophilia, sputum or blood, pregnancy, ≥1 severe exacerbation of asthma during the past 12 months, transferred intubation or treatment in the ICU on the BA.

Risk factors for fixing bronchial obstruction: failure to use inhaled corticosteroids, exposure to tobacco smoke or other harmful substances (including in the workplace), low initial FEV1, chronic excessive secretion in the respiratory tract, eosinophilia of sputum or blood.

Clinical picture and natural course

1. Subjective symptoms: paroxysmal shortness of breath, mainly expiratory (sometimes felt as constriction in the chest), which occurs spontaneously or under the influence of treatment; wheezing dry, paroxysmal cough (accompanying shortness of breath or as the only symptom [the so-called cough variant of asthma]; isolated coughing in adults is rarely a symptom of AD). In patients with allergic AD, symptoms of other allergic diseases, most often allergic rhinitis, are accompanied. Subjective and objective symptoms are variable and may not occur with the exception of episodes of seizures and exacerbations of AD.
2. Objective symptoms: diffuse, bilateral wheezing (mainly expiratory) and dry wheezing, prolonged expiration (sometimes symptoms that are heard only with forced expiration); during exacerbations, the work of auxiliary muscles and tachycardia.
3. Natural course: AD can occur at any age. If it begins in adulthood, it is more often non-allergic and has a more severe course. During AD, exacerbations occur that develop suddenly (within a few minutes or hours) or gradually (within a few hours or days) and without treatment can lead to death. Long-term uncontrolled asthma leads to progressive, irreversible airway obstruction.

Diagnostics

Additionalresearchmethods

1. Spirometry: in most patients, the result of basic spirometry is within normal limits. AD is characterized by obstruction, especially with variable intensity (significant fluctuations between subsequent studies, or under the influence of treatment); in bronchodilation test - a significant improvement in FEV1 and / or FVC (≥200 ml and ≥12% of the due) and often even the elimination of obstruction (in severe BA or in BA with bronchial remodeling, obstruction may be irreversible), as well as bronchial hyperreactivity in a provocative test with methacholine or histamine. In special cases, the diagnosis can be confirmed using specific provocative tests with an allergen, acetylsalicylic acid, factors present at the workplace, and physical activity.
2. Peak expiratory flow rate (PSV): characteristic is the average (within 2 weeks of measurement) daily variability of PSV ([PSVmax - PSVmin] / PSVredn)> 10%; measurements are used to confirm the diagnosis, monitor the disease (it should be considered useful in patients with severe bronchial asthma or with a weak perception of symptoms) and to determine the factors that provoke symptoms (e.g. occupational factors).
3. Chest RG: usually normal, with exacerbation, there may be signs of hyperpneumatization of the lungs (air traps) and complications of exacerbation (eg pneumothorax).
4. Pulse oximetry and arterial blood gasometry: use to assess the severity and monitor the course of exacerbations → see below.
5. Studies that determine the IgE-mediated allergy: skin tests, the concentration of total and specific IgE - can determine the sensitizing allergen in a patient with allergic asthma.
6. Investigation of induced sputum for eosinophilia: in experienced centers, it can be used to change treatment in patients with moderate or severe AD.
7. Study of the concentration of nitric oxide in exhaled air (FENO): as an additional study in the differential diagnosis of COPD → table. 3.8-1. In previously untreated patients, an increased rate (> 50 ppb) correlates with a good response to inhaled corticosteroids.

The diagnosis of AD (according to GINA) requires the presence of symptoms of the disease, as well as a change in the severity of bronchial obstruction in functional studies → Table 3.9-1. It is necessary to assess the severity of the disease.

The goals of treatment:

1) achieving and maintaining control over the symptoms and normal life activity (including the ability to tolerate physical activity);

2) minimizing the risk of exacerbations, prolonged bronchial obstruction and undesirable effects of therapy.

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What is BA?

3. What are the reasons for the development of AD.

4. What are the rules for examining a patient with AD?

5. What are the signs of attention when examining the chest of a patient with AD?

3. Chest RG: usually normal, with exacerbation, there may be signs of hyperpneumatization of the lungs (air traps) and complications of exacerbation (eg pneumothorax).

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4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What is BA?

3. What are the reasons for the development of AD.

4. What are the rules for examining a patient with AD?

5. What are the signs of attention when examining the chest of a patient with AD?

Lecture № 5:

1. Theme: Pleurisy.

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

Pleurisy is an inflammation of the pleura, the membrane covering the lungs and the wall of the chest cavity. With pleural irritation, a disease such as pleurisy develops. It leads to inflammation of the membranes that surround the lungs and line the chest cavity. Hippocrates also described the clinical signs of this disease in 400 BC. e. In the pleural cavity, fluid may be accumulated, which is called a pleural effusion, or exudate. However, such a liquid may not be, then we are talking about dry pleurisy. The most common symptom of pleurisy is a sudden onset of chest pain. During breathing, this pain feels like vague discomfort and can become sharp, intense and acute. Breathing is usually rapid and shallow, as deep breathing intensifies pain. Other symptoms: shortness of breath, cough, fever or weight loss - depending on the underlying cause of the disease. The most common of these is a viral infection. Other causes include pneumonia, pulmonary embolism, autoimmune disorders, lung cancer, complications after heart surgery, tuberculosis, pancreatitis, chest trauma (rib fracture), asbestosis, and allergic reactions caused by drugs. Sometimes the reason remains unknown. Other conditions that can cause these symptoms include pericarditis, heart attack, cholecystitis, and pneumothorax.

Diagnosis and treatment

With a stethoscope, the doctor listens to a characteristic scratching sound called pleural friction. Diagnostics may also include a chest x-ray, electrocardiogram (ECG), and blood tests.

The treatment of pleurisy depends on its specific cause. For example, antibiotics are prescribed for bacterial infections, and specific treatment is not necessary for viral infections. Analgesics, such as paracetamol or ibuprofen, can help reduce chest pain, regardless of the cause of pleurisy.

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What are the main and additional complaints of patients with respiratory diseases?
2. What is a cough?
3. What are the types of chest.
4. What is pleurisy?
5. Describe the combat syndrome in pleurisy.

Lecture № 6:

1. Topic: Acute and chronic glomerulonephritis

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

Glomerulonephritis (GN) is a heterogeneous group of diseases that is characterized by inflammation exclusively or mainly of the glomerular apparatus of the kidneys. Changes associated with other structures of the kidneys (tubules, interstitial substance, vessels) are secondary and are the result of disorders (mainly proteinuria) caused by glomerular anomalies. The sources of inflammation are pathological immunological processes, in many cases the causes and pathogenesis are unknown. During GBV, exacerbations, relapses, and remissions may occur.

Primary GN: the disease affects only the glomeruli, and clinical symptoms and laboratory abnormalities are the result of structurally functional disorders in the glomeruli. In some cases of primary GN, the cause is known (e.g. post-infectious, post-vaccination GN); in the majority - are idiopathic.

Secondary GN: glomerular damage is the result of another pathological process, often multi-organ or multisystem.

In most cases, the nature and severity of morphological changes in the glomeruli, the degree of their damage cannot be determined on the basis of the clinical picture of the disease, since with very similar histopathological changes, various clinical forms can develop. In addition, at different stages of a particular type of GN, its clinical picture may change (e.g. initially nephrotic syndrome, then chronic GN, or first asymptomatic microhematuria, and then rapidly progressive GN → see below), which is the result of various disease activity and sometimes transformation or the intersection of one GB with another. For these reasons, GN is classified based on the histopathological picture, since it indicates the pathogenesis of GN and the nature of damage to kidney structures, which are crucial for choosing a treatment and evaluating the prognosis. Most types of GN can be primary or secondary glomerulopathy, and diseases occur under various clinical forms.

Clinical picture and natural course

In most cases, GN is present or one of the following clinical forms predominates:

1. Nephrotic syndrome

2. Nephritic syndrome: characterized by the occurrence of arterial hypertension, decreased urine volume and edema, usually moderate. Urinalysis shows proteinuria ≤3.5 g / day. In the urine sediment, leached and altered red blood cells, red blood cells and granular cylinders.

3. Asymptomatic microhematuria with or without proteinuria: constant or periodic microhematuria, also macrohematuria during exacerbation periods with varying degrees of proteinuria not exceeding the degree of nephrotic. Initially, there are no other clinical signs of GBV. CKD symptoms may develop over time.

4. Acute GN

5. Chronic GN: progressive CKD is caused by prolonged GN with a latent course. In many cases, after several years there are no signs of active inflammation in the glomerulus, further progression of CKD is the result of damage to a significant number of glomeruli and progressive secondary interstitial fibrosis with renal tubular atrophy. The clinical picture is typical for CKD and depends on its stage. Urinalysis usually shows proteinuria not exceeding the degree of nephrotic, in some cases with a small microhematuria.

6. Rapidly progressive GN: nephritic syndrome is accompanied by very rapidly progressing renal failure.

Additional research methods

1. Urinalysis: varying degrees of proteinuria and / or microhematuria; granular, erythrocyte, waxy cylinders.

2**. Blood tests:** an increase in serum creatinine, when the result of GN is a decrease in GFR; in certain GN variants, immunological markers are present.

3. Kidney biopsy: the only accurate way to diagnose GB and its type. It is necessary in case of suspicion of primary GN (with the exception of nephrotic syndrome in children; in ≈80% of cases it is caused by GN with minimal changes) in a patient with nephrotic syndrome, rapidly progressing renal failure or microhematuria (after excluding its causes in the urinary tract), both the choice of treatment, including immunosuppressive therapy, and the prognosis depend on the histopathological type of GN, the degree of damage to the renal glomeruli, and the severity of secondary tubulointerstitial changes.

An accurate histopathological diagnosis is also necessary in many cases of suspected secondary glomerulonephritis, e.g. in order to confirm the diagnosis of a systemic disease or type of glomerulonephritis with a previously diagnosed systemic disease (systemic lupus erythematosus), or when there is doubt about the nature and extent of damage to the renal glomeruli.

Treatment

1. Primary GN: immunosuppressive therapy, reducing the rate of progression of chronic renal failure and combating factors that accelerate this progress (arterial hypertension, proteinuria, hyperlipidemia, smoking, obstruction of the outflow of urine).

2. Secondary GN: tactics include treatment of the underlying disease (which can lead to the complete disappearance of nephropathy), as well as slowing the progression of CKD.

3. Nephrotic syndrome: general principles of treatment

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What are the causes of GBV?
2. Describe the pain syndrome in GB.
3. Describe the nephrotic syndrome?
4. What is nephritic syndrome?
5. What are the complications of GBV?

Lecture № 7:

1. Topic: Acute renal failure

2. Purpose: To familiarize students with the introduction to the section of clinical medicine-nephrology, to give a general idea about the diseases of the urinary 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 renal failure is a sudden, potentially reversible violation of homeostatic renal function.

Currently, the incidence of acute renal failure reaches 200 per 1 million people, with 50% of patients requiring hemodialysis. Since the 1990s, there has been a steady trend, according to which acute renal failure is increasingly becoming not a monoorgan pathology, but a component of multiple organ failure syndrome. This trend continues in the 21st century.

Classification. Acute renal failure is divided into prerenal, renal and postrenal.

Prerenal acute renal failure is caused by hemodynamic impairment and a decrease in the total volume of circulating blood, which is accompanied by renal vasoconstriction and a decrease in renal blood circulation. As a result, renal hypoperfusion occurs, the blood is not sufficiently cleansed of nitrogenous metabolites, azotemia occurs. Prerenal anuria accounts for 40 to 60% of all cases of acute renal failure.

Renal acute renal failure is more often caused by ischemic and toxic damage to the renal parenchyma, less often by acute inflammation of the kidneys and vascular pathology. In 75% of patients with renal acute renal failure, the disease proceeds against the background of acute tubular necrosis.

Postrenal acute renal failure is most often accompanied by anuria and occurs as a result of obstruction at any level of the extrarenal urinary tract.

Etiology. The main causes of prerenal acute renal failure are cardiogenic shock, cardiac tamponade, arrhythmia, heart failure, pulmonary embolism, i.e. conditions accompanied by a decrease in cardiac output. Another reason may be severe vasodilation caused by anaphylactic or bacteriotoxic shock. Prerenal acute renal failure is often caused by a decrease in extracellular fluid volume, which can be caused by conditions such as burns, blood loss, dehydration, diarrhea, cirrhosis and ascites caused by it.

Renal acute renal failure is caused by exposure to the kidney of toxic substances: salts of mercury, uranium, cadmium, copper. Poisonous fungi and some medicinal substances have a pronounced nephrotoxic effect, primarily aminoglycosides, the use of which in 5-20% of cases is complicated by

moderate acute renal failure and in 1-2% severe. In 6-8% of all cases of acute renal failure, it develops with the use of non-steroidal anti-inflammatory drugs. Radiopaque substances have nephrotoxic properties, which requires careful use in patients with impaired renal function. Hemoglobin and myoglobin, circulating in large quantities in the blood, can also cause the development of renal acute renal failure. The reason for this is massive hemolysis caused by transfusion of incompatible blood, and hemoglobinuria. The causes of rhabdomyolysis and myoglobinuria can be traumatic, for example, crash syndrome, and non-traumatic, associated with muscle damage in prolonged alcoholic or narcotic coma.

Postrenal acute renal failure accounts for approximately 5% of all cases of impaired renal function. The reason for it is a mechanical violation of the outflow of urine from the kidneys, most often due to obstruction by calculi of the upper urinary tract on both sides. Other causes of impaired urine outflow are ureteritis and periureteritis, tumors of the ureters, bladder, prostate, genitalia, narrowing and tuberculous lesions of the urinary tract, metastases of breast or uterine cancer in the retroperitoneal tissue, bilateral sclerotic periureteritis of unclear retroperitoneal processes, dystrophic.

Pathogenesis. In acute renal failure due to prerenal factors, the cause of the pathological mechanism is ischemia of the renal parenchyma. Even a short-term decrease in blood pressure below 80 mm Hg. Art.leads to a sharp decrease in blood flow in the kidney parenchyma due to the activation of shunts in the juxtamedullary zone. A similar condition can occur with a shock of any etiology, as well as a result of bleeding, including during surgical intervention. In response to ischemia, necrosis and epithelial rejection of the proximal tubules begin, and the process often reaches acute tubular necrosis. Reabsorption of sodium is sharply disrupted, which leads to its increased intake in the macula densa region and stimulates the production of renin, which supports spasm of the arterioles and ischemization of the parenchyma.

With toxic damage, the epithelium of the proximal tubules also most often suffers, while in the case of toxic effects of the pigments myoglobin and hemoglobin, the situation is exacerbated by obstruction of the tubules by these proteins.

In postrenal acute renal failure, a violation of the outflow of urine from the kidneys causes overstretching of the ureters, pelvis, collective tubules, and the distal and proximal nephrons. The consequence of this is massive interstitial edema. If the outflow of urine is restored quickly enough, the changes in the kidneys are reversible, however, with prolonged obstruction, severe disturbances in the blood circulation of the kidneys can occur, which can result in tubular necrosis.

Symptoms The course of acute renal failure can be divided into the initial, oligoanuric, diuretic and phase of complete recovery.

The initial phase can last from several hours to several days. During this period, the severity of the patient's condition is determined by the cause of the development of the pathological mechanism of acute renal failure. It was at this time that all the previously described pathological changes develop, and the entire subsequent course of the disease is their consequence. A common clinical symptom of this phase is circulatory collapse, which is often so short that it goes unnoticed.

The oligoanuric phase develops in the first 3 days after an episode of blood loss or exposure to a toxic agent.

The diuretic phase lasts 9-11 days. Gradually, the amount of urine excreted begins and after 4-5 days it reaches 2-4 liters per day or more. Many patients experience a loss of a large amount of potassium in the urine - hyperkalemia gives way to hypokalemia, which can lead to hypotension and, even, paresis of skeletal muscles, and heart rhythm disturbances. Urine has a low density, it has a reduced content of creatinine and urea, but after 1 week. diuretic phase with a favorable course of the disease, hyperazotemia disappears and the electrolyte balance is restored.

In the phase of complete recovery, further restoration of renal function occurs. The duration of this period reaches 6-12 months, after which kidney function is fully restored.

Diagnostics. Diagnosis of acute renal failure, as a rule, is not difficult. Its main marker is a continuous increase in the level of nitrogen metabolites and potassium in the blood along with a decrease in the amount of urine excreted. In a patient with clinical manifestations of acute renal failure, the determination of its cause is mandatory. The differential diagnosis of prerenal acute renal failure from renal is extremely important, since the first form can quickly go into the second, which will aggravate the course of the disease and worsen the prognosis. First of all, it is necessary to conduct a differential diagnosis of postrenal acute renal failure from its other types, for which an ultrasound of the kidneys is performed, which allows to determine or exclude the fact of bilateral obstruction of the upper urinary tract by the presence or absence of dilatation of the pyelocaliceal system.

Treatment. In the initial phase of acute renal failure, treatment should first of all be aimed at eliminating the cause of the development of the pathological mechanism. In shock, which is the cause of 90% of acute renal failure, the main thing is therapy aimed at normalizing blood pressure and replenishing the volume of circulating blood. The introduction of protein solutions and large molecular weight dextrans, which should be administered under the control of an indicator of central venous pressure, is effective, so as not to cause hyperhydration.

In case of poisoning with nephrotoxic poisons, it is necessary to remove them by washing the stomach and intestines. Unitiol is a universal antidote for poisoning with salts of heavy metals. Hemosorption, undertaken even before the development of acute renal failure, can be particularly effective.

In the case of postrenal acute renal failure, therapy should be aimed at early recovery of urine outflow.

In the oliguric phase in acute renal failure of any etiology, the introduction of osmotic diuretics in combination with furosemide, the dose of which can reach 200 mg, is necessary. The introduction of dopamine in "renal" doses is shown, which will reduce renal vasoconstriction. The volume of injected fluid should make up for its loss with stool, vomiting, urine and an additional 400 ml consumed by Breathing, sweating. The diet of patients should be protein-free and provide up to 2000 kcal / day. To reduce hyperkalemia, it is necessary to limit its intake with food, as well as to conduct surgical treatment of wounds with the removal of necrotic sites, drainage of cavities. In this case, antibiotic therapy should be carried out taking into account the severity of kidney damage.

An indication for hemodialysis is an increase in potassium content of more than 7 mmol / L, urea up to 24 mmol / L, the appearance of symptoms of uremia: nausea, vomiting, lethargy, as well as hyperhydration and acidosis. Currently, they are increasingly resorting to early or even preventive hemodialysis, which prevents the development of severe metabolic complications. This procedure is carried out every day or every other day, gradually increasing the protein quota to 40 g / day.

Complications Forecast. Mortality in acute renal failure depends on the severity of the course, the age of the patient, and most importantly, the severity of the underlying disease, which caused the development of acute renal failure. In patients who survived after acute renal failure, complete restoration of renal function is observed in 35-40% of cases, partial in 10-15%, and from 1 to

Lecture № 8:

1. Topic: Chronic kidney disease

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

Chronic kidney disease (CKD) is a long-term progressive decrease in renal function. Symptoms develop slowly and in advanced stages include anorexia, nausea, vomiting, stomatitis, dysgeusia, nocturia, apathy, chronic fatigue, itching, decreased mental clarity, muscle convulsions and cramps, fluid retention, hypotrophy, peripheral neuropathies and epileptic seizures. Diagnosis is based on

laboratory tests of renal function, which are sometimes supplemented by a kidney biopsy. Treatment is primarily aimed at the underlying disease, but also includes the normalization of water and electrolyte balance, blood pressure control, treatment of anemia, various types of dialysis and kidney transplantation.

Etiology

CKD can be caused by any cause of significant renal impairment.

The most common causes in the United States in order of incidence include the following:

• Diabetic nephropathy

• hypertensivenephrosclerosis

• Various primary and secondary glomerulopathies

Metabolic syndrome, which is characterized by arterial hypertension and type 2 diabetes, is a common cause of kidney damage with an ever-increasing prevalence.

Pathophysiology

CKD in the early stages is described as a decrease in renal reserve or renal failure, which can progress (end-stage renal failure). Initially, the loss of renal tissue function has almost no obvious pathological manifestations, because the remaining tissue works hard (functional adaptation of the kidneys).

Decreased renal function correlates with the ability of the kidneys to maintain water and electrolyte homeostasis. In the early stages, the ability of the kidneys to concentrate urine is impaired, and then a decrease in the ability to excrete an excess of phosphates, acid and potassium is added. With severe renal failure (GFR ≤15 ml / min / 1.73 m2), the ability to effectively dilute or concentrate urine is lost. Thus, the osmolality of urine is usually approximately 300-320 mosmol / kg, approaching the plasma osmolality (275-295 mosmol / kg), and the volume of urine does not immediately respond to changes in the volume of fluid drunk.

Clinical manifestations

With a moderate decrease in renal reserve, the course is usually asymptomatic. Even patients with mild or moderate renal failure may not have symptoms of elevated levels of AMA and creatinine. Nocturia is often observed, especially due to the inability to concentrate urine. Apathy, fatigue, lack of appetite and decreased clarity of thinking are often the earliest manifestations of uremia. In more severe kidney disease (for example, the estimated glomerular filtration rate [rSKF] <15 ml / min / 1.73 m2) neuromuscular symptoms may appear, including severe muscle twitching, peripheral sensory and motor neuropathies, muscle cramps, hyperreflexia, syndrome restless legs and cramps (usually as a result of hypertensive or metabolic encephalopathy). Anorexia, nausea, vomiting, weight loss, stomatitis and an unpleasant aftertaste in the mouth are very common. Skin tint may become tan. Sometimes urea crystals then stand out on the surface of the skin, forming a uremic frost. Itching can cause serious inconvenience. Nutritional deficiency leading to generalized tissue loss is a hallmark of chronic uremia.

Diagnostics

• Determination of electrolyte, AMA, creatinine, phosphate, calcium levels, general blood count

• Urinalysis (including urinary sediment microscopy)

• Quantification of protein in urine (24-hour urinary protein excretion or protein / creatinine ratio in a single serving of urine)

• Ultrasound procedure

• Sometimes a kidney biopsy

As a rule, the presence of CKD is first suspected with an increase in serum creatinine. The first step is to determine whether renal failure is acute, chronic or acute, which has become chronic (for example, an acute disease that further impairs the function of the kidneys in a patient with CKD - Differential diagnosis of acute and chronic renal failure). The cause of renal failure is also identified. Sometimes

determining the duration of renal failure helps determine the cause; sometimes it's easier to determine the cause than duration, and finding out the reason helps determine the duration.

Treatment

• Treatment of the underlying cause of the disease
• A feasible restriction in the diet of protein, phosphates and potassium
• Vitamin D Supplements
• Anemia treatment
• Treatment of concomitant diseases (eg, heart failure, diabetes mellitus, nephrolithiasis, prostatic hypertrophy)
• Correction of doses of all drugs as necessary
• Hemodialysis with a significant reduction in GFR, if symptoms and signs are not sufficiently amenable to medical intervention
• Maintaining sodium bicarbonate at 23 mmol / L

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What are the main complaints about diseases of the genitourinary system?
2. What should you pay attention to during a general examination of patients with kidney disease?
3. How is palpation of the kidneys?
4. What information does palpation of the kidneys provide?
5. What are the reasons for the development of CKD?
6. What are the indications for kidney transplantation?

Lecture № 9:

1. Topic: Arterial hypertension.

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

In the United States, about 75 million people suffer from hypertension. About 81% of these people are aware that they have AH, only 75% receive treatment and only 51% adequately control blood pressure. Among adults, hypertension is more common among African Americans (41%) than among Europeans (28%) or Americans of Mexican descent (28%), as well as among African Americans, morbidity and mortality are higher.

Blood pressure increases with age. About two-thirds of people> 65 years of age suffer from hypertension, and people with normal blood pressure at the age of 55 have a 90% risk of developing hypertension. Arterial hypertension may develop during pregnancy

Arterial hypertension may be:

• Primary (from 85% of cases)
• Secondary

Clinical manifestations

Arterial hypertension is usually asymptomatic until complications in the target organs develop. Uncomplicated arterial hypertension can cause dizziness, facial flushing, headache, fatigue, nosebleeds, and irritability. Severe hypertension (hypertensive crisis) can cause serious cardiovascular, neurological, renal symptoms and retinal damage (for example, symptomatic coronary arteriosclerosis, heart failure, hypertensive encephalopathy, renal failure).

The presence of a 4th cardiac sound is one of the earliest signs of hypertensive cardiomyopathy.

Diagnostics

• Multiple blood pressure measurements to confirm
• Urinalysis and albumin / creatinine ratio in urine; if there are deviations from the norm, then consider the need for an ultrasound of the kidneys
• Blood tests: fasting, creatinine, potassium lipid levels
• Kidney ultrasound with elevated creatinine
• It is necessary to evaluate the presence of aldosteronism if potassium levels are reduced
• ECG: in the presence of left ventricular hypertrophy, echocardiography is necessary
• Sometimes the definition of thyroid stimulating hormone
• Assess the presence of pheochromocytoma or sleep disturbances if blood pressure is labile and rises suddenly or severe arterial hypertension occurs

The more severe arterial hypertension and the younger the patient, the more extensive the examination should be. As a rule, when arterial hypertension is recently diagnosed, a routine examination is performed to:

• Identification of damage to target organs
• Definitions of cardiovascular risk factors

Surveys include:

• Urinalysis and urinary albumin to creatinine ratio
• Blood tests (creatinine, potassium, sodium, fasting glucose, lipid profile and often thyroid-stimulating hormone)
• ECG

Forecast

The higher the blood pressure and the more serious changes in the retina, and also if there are other signs of damage to the target organs, the worse the prognosis. Systolic blood pressure is a predictor of fatal and non-fatal cardiovascular events to a greater extent than diastolic blood pressure. Without treatment, 1-year survival is 10% in patients with retinal sclerosis, cotton exudates, narrowing of the arterioles and hemorrhage (grade 3 retinopathy) and 5% in patients with the same changes in combination with optic nerve edema (grade 4 retinopathy) . IHD is the most common cause of death among hypertensive patients receiving treatment. Ischemic or hemorrhagic stroke is the most common consequence of the lack of adequate treatment for hypertension. However, effective control of hypertension prevents most complications and prolongs life.

Treatment

• Weight loss and physical activity
•To give up smoking
• Diet: increased consumption of fruits and vegetables, reduced salt intake, reduced alcohol consumption
• Medicines: Depending on blood pressure and the presence of cardiovascular disease or risk factors

Primary hypertension cannot be cured, but some causes of secondary hypertension can be eliminated. In all cases, blood pressure control can significantly limit adverse effects. Despite the theoretical effectiveness of treatment, blood pressure drops to the desired level in only a third of patients with hypertension.

4**. Illustrative material:** presentation
5. Literature: indicated in syllabus
6. Security questions (feedback):

1. What causes pulsation of the cervical vessels?
2. What are the causes of hypertension?
3. What methods are used to determine blood pressure?
4. What are the risk factors for hypertension.

5. What complications of hypertension do you know?

Lecture № 10

1. Topic: Atherosclerosis

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

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

3. Lecture notes: Atherosclerosis is the most common form of arteriosclerosis, which is a general term for several diseases that cause thickening and loss of elasticity of the arterial walls. Atherosclerosis is also the most serious and clinically significant form of arteriosclerosis, as it causes coronary heart disease and cerebrovascular disease. Non-atheromatous forms of arteriosclerosis include Mankeberg arteriosclerosis and arteriolosclerosis.

Atherosclerosis can affect all large and medium-sized arteries, including coronary, carotid and cerebral arteries, the aorta, its branches and large limb arteries. This disease is a leading cause of morbidity and mortality in the United States and most developed countries. The prevalence of atherosclerosis is increasing rapidly in developing countries, and as people in developed countries live longer, the incidence will increase. Atherosclerosis will be the leading cause of death worldwide.

Risk factors

There are a large number of risk factors for development. Certain factors often occur simultaneously with metabolic syndrome, which is becoming more common. This syndrome includes abdominal obesity, atherogenic dyslipidemia, arterial hypertension, insulin resistance and a predisposition to thrombosis and general inflammatory reactions in sedentary patients. Insulin resistance is not synonymous with metabolic syndrome, but may play a key role in its etiology.

Clinical manifestations

Atherosclerosis initially develops asymptomatically, often over decades. Signs appear when obstruction to blood flow occurs. Transient ischemic symptoms (e.g., stable angina pectoris, transient ischemic attacks, intermittent claudication) can develop when stable plaques grow and reduce arterial clearance> 70%. Vasoconstriction can aggravate damage to the vascular wall (which did not limit blood flow before) and lead to severe or complete stenosis. Symptoms of unstable angina pectoris, myocardial infarction, ischemic stroke, or leg pain alone can occur when unstable plaques burst and suddenly close a large artery, with the addition of thrombosis or embolism. Atherosclerosis can also cause sudden death without prior stable or unstable angina pectoris.

Atherosclerotic damage to the arterial wall can lead to aneurysms and stratification of the arteries, which is manifested by pain, throbbing sensations, lack of pulse or causes sudden death.

Diagnostics

The approach depends on the presence or absence of signs of the disease.

Treatment

• Lifestyle changes (diet, smoking, physical activity)
• Medication for established risk factors
• Antiplatelet drugs
• Statins, possibly ACE inhibitors, beta blockers

Treatment involves actively eliminating risk factors to prevent the formation of new plaques and reduce existing ones. Lowering LDL levels below a certain target level is no longer recommended, and currently the “the lower the better” approach is preferred.

Lifestyle changes include diet, smoking cessation, and regular physical activity. Often, medications are needed to treat dyslipidemia, hypertension, and diabetes. These lifestyle changes and drugs directly or indirectly improve endothelial function, reduce inflammation and improve clinical outcome. Statins

can reduce atherosclerosis-related morbidity and mortality even in patients with normal or slightly elevated total cholesterol. Antiplatelet drugs are effective for all patients with atherosclerosis. Patients with coronary heart disease may benefit from the use of ACE inhibitors and beta-blockers.

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. What is atherosclerosis?
2. What are the causes of atherosclerosis?
3. What is the difference between HLVP and HLNP?
4. What are the complications of atherosclerosis.
5. What methods are used to determine atherosclerosis?

Lecture № 11

1. Topic: Coronary heart disease

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

In developed countries, coronary heart disease is the leading cause of death among patients regardless of gender, accounting for approximately one third of all mortality. Mortality is about 1 in 10,000 white men aged 25–34 years and about 1 in 100 aged 55–64 years. Mortality among white men aged 35–44 years is 6.1 times higher compared to women of the same age group. For unknown reasons, these gender differences are less noticeable among fair-skinned and in patients with diabetes mellitus. Mortality in the female subgroup of patients increases after menopause and after 75 years of age, becoming equal to or exceeding that in men.

Etiology

Usually coronary heart disease is caused by

• Atherosclerosis of the coronary artery: subintimal atheroma deposition in large and medium coronary arteries

Less commonly, coronary heart disease is caused by

• spasm of the artery

Violation of the function of vascular endothelium can contribute to the development of atherosclerosis and contributes to spasm of the coronary arteries. Currently, endothelial dysfunction is becoming increasingly important as a cause of angina pectoris in the absence of stenosis or spasm of the superficial arteries of the heart (see Syndrome X).

Rare causes of coronary heart disease can be conditions such as coronary artery embolism, dissection, aneurysm (for example, with Kawasaki disease), vasculitis (SLE, syphilis).

Risk factors

Risk factors for developing coronary heart disease are the same as risk factors for developing atherosclerosis:

• High blood levels of low density lipoprotein (LDL) cholesterol
• High blood levels of lipoprotein a
• Low cholesterol high density lipoprotein (HDL) in the blood
• Diabetes mellitus (especially type 2)
• smoking
• obesity
• Lack of physical activity

• High levels of apoprotein B (apo B)
• High levels of C-reactive protein (CRP) in the blood
Smoking can be a stronger predictor of MI in women (especially those under the age of 45). In addition, genetic factors, various systemic disorders (arterial hypertension, hypothyroidism) and metabolic disorders (for example, hyperhomocysteinemia) are important. High levels of apo B may indicate an increased risk, while total cholesterol or LDL levels remain normal.
A high concentration of C-reactive protein in the blood is a marker of plaque instability and inflammation. This predictor has a closer relationship with acute coronary events than the level of LDL cholesterol. An increase in the level of triglycerides and insulin (as a reflection of insulin resistance) may be a risk factor for the development of the disease, however, for unambiguous judgments, the data obtained are still insufficient. Smoking is a risk factor for CHD; Also, the risk of coronary heart disease increases with certain dietary preferences - high-calorie foods saturated with fats and poor in phyto-compounds (found in fruits and vegetables), dietary fiber, vitamins C, D and E; food is also relatively poor in omega-3 (n-3) polyunsaturated fatty acids (PUFAs - at least in some people); and poor stress tolerance.
Treatment
• Drug therapy, including antithrombotic drugs, lipid-lowering drugs (such as statins) and beta blockers
• Percutaneous coronary intervention
• In acute thrombosis, sometimes fibrinolytic drugs
• Coronary artery bypass grafting
The main goals of treatment are: reducing the workload on the heart by reducing oxygen consumption and improving coronary blood flow, and in the long term, stopping and re-developing the processes of atherosclerosis. Blood circulation in the coronary arteries can be improved with percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). In acute coronary artery thrombosis, the use of fibrinolytic drugs is possible.
4. Illustrative material: presentation
5. Literature: indicated in syllabus
6. Security questions (feedback):
1. IHD. Definition
2. What are the reasons for the development of coronary heart disease.
3. Tell the classification of coronary heart disease.
4. Define angina pectoris.
5. What are the complications of ischemic heart disease?

Lecture № 12
1. Topic: myocardial infarction
2. Purpose: To familiarize students with the introduction to the section of clinical medicine-cardiology, to give a general idea about diseases of the circulatory 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:
In the United States, about 1.5 million cases of myocardial infarction are reported annually. Of these, about 400-500 thousand cases end fatally, with half of the deaths occurring at the prehospital stage. Acute myocardial infarction (MI), along with unstable angina pectoris, is considered as acute coronary syndrome. Acute MI includes both myocardial infarction without ST segment elevation (IMbST) and myocardial infarction with ST segment elevation (IMST). The distinction between IMbST and IMpST is vital because treatment strategies for these two nosologies are different. Myocardial infarction is

defined as myocardial necrosis in a clinical situation corresponding to myocardial ischemia. These conditions satisfy the following requirements: an increase in the level of cardiac biomarkers (cardiac biomarkers) in the blood (preferably troponin - above 99 percent of the upper reference limit [GRP]) and the presence of at least one of the following symptoms:

• Symptoms of ischemia

• Changes in the ECG indicating a new focus of ischemia (significant changes in ST / T or blockade of the left bundle branch block);

• Development of pathological Q waves

• The presence of new sites of myocardial necrosis or abnormal regional wall movement, as evidenced by visual diagnostics

• Evidence of intracoronary thrombus obtained by angiography or autopsy

Heart attack location

IM predominantly affects the LV, while it is possible to spread the process to the pancreas and atria. Right ventricular MI usually develops due to occlusion of the right coronary artery or the dominant artery envelope. The main manifestations are an increase in pancreatic filling pressure, often combined with severe tricuspid regurgitation and a decrease in cardiac output. Lower-posterior localization of MI often leads to pancreatic dysfunction in about half of patients and is manifested by hemodynamic disturbances in 10-15%. Pancreatic dysfunction should be suspected in each patient with inferior localization of myocardial infarction and increased central venous pressure with hypotension or shock. The development of pancreatic myocardial infarction with LV myocardial infarction significantly increases the risk of death. MI of anterior localization is usually higher in prevalence and has a worse prognosis compared to inferior MI. The development of anterior MI is usually associated with occlusion in the basin of the left coronary artery, especially the anterior descending artery; inferior posterior MI is associated with obstruction of the right coronary artery or the dominant envelope of the artery.

Heart attack prevalence

Myocardial infarction may be

• transmural

• Non-transmural

Transmural MI involves all layers of the myocardium from the epicardium to the endocardium and is characterized by the appearance of abnormal Q waves on the ECG. Non-transmural or subendocardial heart attacks do not penetrate the ventricular wall and only lead to the appearance of ST-segment anomalies and T wave (ST-T anomalies). Subendocardial infarction usually affects the inner third of the myocardium, where wall tension is highest, and myoardial blood flow is most vulnerable to changes in blood circulation. The development of this type of heart attack can follow after prolonged periods of hypotension. Since the transmural spread of myocardial infarction cannot be accurately verified by clinical methods, myocardial infarction is usually classified as myocardial infarction and myocardial infarction depending on the presence of elevation of the ST segment or Q waves on the ECG. The volume of the damaged myocardium can be approximately estimated using the magnitude and duration of the increase in creatine phosphokinase or, most often, the peak level of troponins.

Myocardial infarction without ST segment elevation (myocardial infarction without ST, subendocardial MI) is cardiac muscle necrosis (confirmed by analysis of cardiomarkers in the blood: troponin T or troponin I and CPK levels are elevated), which is not accompanied by acute ST segment elevation on the ECG. IMBST is characterized by ECG changes such as ST segment depression, T wave inversion, or a combination thereof.

Myocardial infarction with ST segment elevation (IMpST, transmural MI) is cardiomyocyte necrosis, accompanied by persistent ST segment elevation on the ECG, which does not disappear after taking nitroglycerin. Levels of cardiac markers such as troponin I or troponin T and CPK are elevated.

Clinical manifestations

Symptoms of IMcST and IMbST are similar. A few days or weeks before a coronary event, about 2/3 of patients report prodromal symptoms, which include unstable or progressive angina pectoris, shortness of breath, or fatigue.

Usually, the first symptom of MI is intense pain deep behind the sternum, described by patients as compression or aching pain, often radiating to the back, lower jaw, left arm, right arm, shoulders, or all of these areas. In its characteristics, pain is similar to that of angina pectoris, but usually more intense and prolonged, often accompanied by shortness of breath, sweating, nausea and vomiting, taking nitroglycerin and rest have only a partial and temporary effect. However, the pain syndrome may be less pronounced, about 20% of acute MI are asymptomatic (the clinic is absent or manifests non-specific symptoms that are not perceived by the patient as a disease), which is most often manifested in patients with diabetes mellitus. Patients often interpret the pain in the chest as a digestive disorder, partly due to the positive effect after burping air and taking antacids.

In some patients, the disease manifests itself as loss of consciousness.

Women are characterized by frequent development of an atypical MI clinic. Elderly patients are more likely to have complaints of shortness of breath than angina pain. In severe cases of ischemia, the patient often has a pronounced pain syndrome, accompanied by anxiety and fear of death. Nausea and vomiting may occur, especially with myocardial infarction. Shortness of breath and weakness due to left ventricular failure, pulmonary edema, shock, or clinically severe arrhythmia predominate.

The skin can be pale, cold, wet with sweat. The appearance of peripheral or central cyanosis is possible. The appearance of a filiform pulse, fluctuations in the level of blood pressure, although arterial hypertension is recorded in many patients during a pain attack.

These auscultations of the heart are characterized by the appearance of distant noises; in almost all patients, the 4th heart tone is recorded. The occurrence of soft systolic blowing noise with a maximum at the apex of the heart, reflecting the occurrence of papillary muscle dysfunction, may be noted. During the initial examination, a friction noise or a more expressive noise may indicate a previously existing heart disorder or other diagnosis. Determination of pericardial friction noise during the first few hours after the onset of symptoms of MI indicates acute pericarditis rather than MI. However, the occurrence of pericardial friction noise, often intermittent, is typical 2-3 days after IMCST. In 15% of patients, sensitivity to palpation of the chest is noted.

Diagnostics

• ECG in dynamics

• Measurement of the level of cardiomarkers in dynamics

• Coronary angiography according to emergency indications (if fibrinolytics were not used) in patients with UTI or its complications (persistent anginal pain, hypotension, a significant increase in the level of cardiac markers, rhythm disturbances)

• Delayed coronary angiography (within 24–48 h) for patients with IMbST without assessment The assessment begins with the initial ECG and ECG in dynamics, as well as with the determination of the dynamics of cardiac markers, which helps to differentiate unstable angina, myocardial infarction with ST elevation and myocardial infarction without lifting ST. This difference is key in finding solutions, since fibrinolytics are indicated for patients with UTI, but may increase the risk for patients with UTI and unstable angina. In addition, patients with the acute form of IMpST are shown to have emergency CG, while patients with IMPST usually do not.

ECG

An ECG is the most important test and should be done within the first 10 minutes of admission.

Anterior MI of the left ventricle (ECG done several hours after the first symptoms of the disease)

The diagnosis of myocardial infarction is not necessary for the presence of pathological Q waves. A careful interpretation of the ECG is necessary, since the ST segment elevation can be mild, especially

in the leads characterizing the lower LV wall (II, III, aVF), and sometimes the doctor's attention is more focused on leads with depression ST segment. In the case of a typical clinic, the ST segment elevation on the ECG is characterized by a specificity of 90% and a sensitivity of 45% in the diagnosis of MI. The study of ECG in dynamics (registration every 8 hours during the first day, then daily) allows you to confirm the diagnosis in the case of the development of pathological Q waves within a few days from the onset of the disease.

If there is a suspicion of right ventricular (RV) myocardial infarction, ECG recording is required in 15 leads: additional lead electrodes are located in position V4-6R and for the diagnosis of posterior MI in positions V8 – V9.

ECG diagnosis of MI is difficult if the patient has a complete blockade of the left leg of the bundle of His, since the form of the QRS complex resembles changes in MI. Elevation of the ST segment, concordant with the QRS complex, is a clear sign of MI if there is an elevation of ST> 5 mm in at least 2 chest leads. In any case, a patient with an appropriate clinical picture and newly arising blockade of the left leg of the bundle of His (or lack of data on the history of blockade registration) is considered as a patient with IMCST.

Myocardial Damage Markers

Cardiac markers (serum markers of damaged cardiomyocytes) are cardioenzymes (e.g., CPK-MB) and cell contents (e.g., troponin T, troponin I, myoglobin) that are released into the systemic circulation during necrosis of cardiomyocytes. Markers are found in the blood at different times from the moment of necrosis, and their levels increase at different rates. The specificity and sensitivity of these markers to damage to cardiomyocytes is significantly different, but troponins are the most sensitive and specific, and are currently preferred markers. Recently, several new, highly sensitive methods for the analysis of cardiac troponin have become available, which also provide very accurate results. These methods can reliably examine troponin levels (T or I) in such low ranges as 0.003-0.006 ng / ml (3 to 6 pg / ml);

Mortality during the first year after MI is from 8 to 10%. Most deaths occur in the first 3-4 months after discharge from the hospital. High-risk indicators include persistent ventricular arrhythmias, heart failure, decreased contractile function of the left ventricle, and recurrence of myocardial ischemia. Most experts recommend performing stress echocardiography before the patient is discharged from the clinic or in the first 6 weeks. after discharge. Good tolerance of physical activity without the occurrence of ECG changes is associated with a favorable prognosis, while further studies are optional. Poor exercise tolerance is an indicator of high risk.

Treatment

• Prehospital care: oxygen, aspirin, nitrates and / or opioids for pain and referral to the appropriate medical center

• Medication: Antiplatelet agents, antianginal drugs, anticoagulants and, in some cases, other drugs

• Reperfusion therapy: fibrinolytics or angiography with percutaneous coronary intervention or coronary artery bypass surgery

• Post-hospital rehabilitation and ongoing management of coronary heart disease

Hospitalization

• Stratify the patient by risk and choose a reperfusion strategy

• Drug therapy with antiplatelet agents, anticoagulants and other drugs based on the reperfusion strategy

Upon arrival at the emergency department, the patient's diagnosis is confirmed. Drug therapy and the timing of revascularization depend on the clinical presentation and diagnosis.

4. Illustrative material: presentation

5. Literature: indicated in syllabus

6. Security questions (feedback):

1. Give a classification of coronary heart disease.
2. Give a definition of IM.
3. What are the types of MI.
4. What is the most informative method for diagnosing MI.
5. What are the complications of myocardial infarction?

Lecture №13. Chronic heart failure.

1. Topic: Chronic heart failure.
2. Purpose: to acquaint students with the introduction of cardiology to the Department of Clinical Medicine, to give a general idea of the diseases of the circulatory system.
The lecture contains information about the epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. Abstracts of lectures:
Heart failure is understood as the insufficiency of the circulatory system (including the heart), which is based on the intake of substances necessary for the proper functioning of organs and tissues and the production of metabolic products. Chronic heart failure is not a separate nosology; it often develops as a secondary syndrome to various diseases. Chronic heart failure often develops in diseases of the cardiovascular system, lungs, liver, kidneys, endocrine diseases (diabetes mellitus, thyrotoxicosis, myxedema, obesity).
CAUSES (ETIOLOGY) OF CHRONIC HEART FAILURE:
1. MYOCARDIAL (myocardial insufficiency, damage to the heart muscle) Primary myocardial insufficiency (myocarditis, dilated cardiomyopathy) Secondary myocardial insufficiency (postinfarction and diffuse cardiosclerosis, hypothyroidism, alcoholic heart damage, heart damage in DTA)
2. CIRCULATION (excessive force on the heart muscle) Pressure force (systolic force on the ventricles) - stenosis of the right and left AV valves, aorta, pulmonary artery, arterial hypertension (systemic, pulmonary) Volumetric force (diastolic force on the ventricles) - heart valve insufficiency , intracardiac shunts Mixed insufficiency (complex heart defects, loss of pressure and volume of the heart).
3. DISTURBANCE OF VENTRICULAR DIASTOLIC FILLING Arterial hypertension, "hypertonic heart" Hypertrophic and restrictive cardiomyopathy Adhesive pericarditis Hydropericardium
4. DISEASES WITH INCREASED HEART RATE Thyrotoxicosis Severe obesity Severe anemia Arrhythmic cardiomyopathy (tachysystolic arrhythmias)
ETIOLOGY OF CHRONIC HEART FAILURE AS A PERCENTAGE
CVD (myocardial infarction) - 60%
Heart problems - 15%
Dilated cardiomyopathy - 11%
Arterial hypertension - 4%
Other reasons - 10%
CLASSIFICATION OF CHRONIC HEART FAILURE ("NYHA" OF THE NEW YORK HEART ASSOCIATION, 1969)
FUNCTIONAL CLASS I - symptoms of heart failure (shortness of breath, palpitations, weakness) do not appear with daily physical exertion.
II FUNCTIONAL CLASS - slight limitation of physical activity, symptoms of heart failure (shortness of breath, palpitations, weakness, cardialgia) are not visible at rest, but are visible with daily exertion.

III FUNCTIONAL CLASS - a clear limitation of effort, symptoms of heart failure (shortness of breath, palpitations, weakness, cardialgia) are not visible at rest, appear with less effort than with daytime effort.

IV FUNCTIONAL CLASS - inability to perform any effort, symptoms of heart failure (shortness of breath, palpitations, weakness, cardialgia) appear at rest, intensify with each effort.

One of the characteristic manifestations of chronic heart failure is orthopnea - the forced sitting of the patient to facilitate breathing with severe shortness of breath. Difficulty breathing in the supine position is due to the accumulation of fluid in the capillaries of the lungs, due to an increase in hydrostatic pressure.

In this case, in the prone position, the diaphragm (diaphragm) rises slightly and makes breathing difficult. Paroxysmal dyspnea at night (cardiac asthma). The reason for this is interstitial pulmonary edema. At night, during sleep, bouts of severe shortness of breath are accompanied by coughing and wheezing in the lungs. With the further development of heart failure, alveolar pulmonary edema develops. Fatigue of patients - due to heart failure, insufficient blood supply to skeletal muscles with oxygen. Patients with heart failure are also concerned about the following complaints: nausea, loss of appetite, abdominal pain, bloating (ascites) caused by stagnation of blood in the portal vein in the liver.

Diagnostics.

ECG

It is possible to identify blockade of the left or right bundle of His bundle, ventricular or atrial hypertrophy, pathological Q wave, arrhythmias. A normal ECG raises the suspicion of chronic heart failure.

echocardiography

It allows you to study the function of the heart and clarify the etiology of heart failure. The main manifestation is the expansion of the left ventricle, a decrease in the fraction of heart contractions.

X-ray

The lungs fill with fluid, causing symptoms of interstitial edema or pulmonary edema. Hydrothorax is detected (in most cases right-sided). Cardiomegaly is diagnosed when the transverse size of the heart exceeds 15.5 cm in men and 14.5 cm in women.

Treatment.

The goal of treating chronic heart failure is to reduce the likelihood of further development of the disease, reduce its symptoms and improve the quality of life of patients. Treatment is carried out with constant monitoring of the patient's condition. Treatment outcomes often depend on the patient and the preventive measures listed below. Drug treatment for chronic heart failure includes the following drugs: Angiotensin-converting enzyme (ACE) inhibitors block the action of a hormone produced by the kidneys, thereby dilating blood vessels and lowering blood pressure. The drugs of this group are indicated for long-term use in all patients; Angiotensin II receptor inhibitors are prescribed for intolerance to AAF inhibitors; Saluretics - diuretics, which are used when there are signs of fluid retention in the body; Aldosterone antagonists - drugs that lead to an increase in the excretion of sodium, chlorine and water from the body, inhibition of the excretion of urea and potassium in the kidneys; Nitrates are drugs that reduce the need of the heart muscle for oxygen and increase its delivery to the heart; Long-term use is advisable only in cases of double angina pectoris. In severe cases of chronic heart failure, doctors may recommend surgery. Three main surgical methods of treatment: Traditional cardiac surgery (correction of valvular insufficiency, surgical correction of the ventricles of the heart, installation of pacemakers); The installation of cardiac implants is an operation to introduce additional circulatory devices (ventricles of the heart) into the human body, which help to partially restore the work of the heart. Heart transplant (replacement). For heart failure, pacemakers and defibrillators may be used.

Disease prognosis:

In general, the prognosis of the disease in patients with HCV is negative, but patients can live for many years. The risk of sudden death in patients with heart failure is 3 times higher than in others. Therefore, it is very important to detect the disease in time and start appropriate treatment. Possible complications:
Enlargement of the chambers of the heart (cardiomyopathy); Violation of the heart rhythm; Stroke; thromboembolism (acute blockage of a blood vessel by a thrombus (thrombus); sudden death.

4. Illustrated materials: presentation

5. Literature: indicated in the syllabus.

6. Control questions (feedback):

1. Name the causes of CHF.
2. Name the functional classes of CHF.
3. Name the most informative diagnostic method.
4. Name their complications.