Lecture complex

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Lecture complex on discipline "Basics of Internal diseases - 1"

Lecture №1

1. Topic: Acute and Chronic Bronchitis

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

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

3. lecture abstracts:

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

The etiology of acute bronchitis - a number of pathogenic factors affecting the bronchi:

1) physical: hypothermia, inhalation of dust

2) chemical: inhalation of acids and alkaline 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 others play the role of a trigger. There are also predisposing factors: smoking, alcohol abuse, heart disease with stasis in the small circulatory system, chronic inflammation in the nasopharynx, oral cavity, tonsils, genetic inferiority of the mucociliary system of the bronchi.

Pathogenesis of acute bronchitis:

Adhesion of pathogens on the epithelial cells lining the trachea and bronchi, decreased effectiveness of local defense factors (ability of the upper airways to filter inhaled air and free it from gross mechanical particles, changes in air temperature and humidity, cough and sneeze reflexes, mucociliary transport) pathogen invasion hyperemia and swelling of bronchial mucosa, desquamation of the cylindrical epithelium, the appearance of mucous or mucopurulent exudate further violation of mucociliary clearance swelling of the bronchial mucosa, hypersecretion of bronchial glands development of an obstructive component.

Classification of acute bronchitis:

1) primary and secondary acute bronchitis

2) According to the level of the lesion:

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

b) bronchitis with predominant involvement of medium-sized bronchi

c) bronchiolitis

3) by clinical symptomatology: mild, moderate and severe

4) by state of bronchial patency: obstructive and nonobstructive

Clinic and diagnosis of acute bronchitis.

If bronchitis develops against the background of acute respiratory infections, at first there appears hoarseness of voice, pain in the throat when swallowing, feeling of chest tightness, irritating dry cough (manifestation of tracheitis). The cough intensifies, and may be accompanied by pain in the lower regions of the chest and behind the sternum. As the inflammation in the bronchi subsides, the cough becomes less painful, abundant mucopurulent sputum begins to be discharged.Intoxication symptoms (fever, headaches, general weakness) vary greatly and are often determined by the causative agent (with adenovirus infection - conjunctivitis, with parainfluenza virus - hoarseness of voice, with flu virus - high temperature, headache and scarce catarrhal phenomena, etc.).

Laboratory findings are not specific. Inflammatory changes in the blood may be absent. On cytological examination of sputum, all fields of view are covered with leukocytes and macrophages.
Treatment of acute bronchitis.
1. Home regimen, diet, plenty of drinking.
2. Mucolytic and expectorants
3. In the presence of bronchoobstructive 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 a persistent cough with sputum separation for at least 3 months a year for 2 or more years, and these symptoms are not associated with any other diseases of the bronchopulmonary system, upper respiratory tract or other organs and systems.
There is a distinction between CHB:
a) primary - an independent disease, not associated with lesions of other organs and systems, often has a diffuse character
b) secondary - etiologically associated with chronic inflammatory diseases of the nose and sinuses, lung diseases, etc., more often it is local.
Etiology of chronic bronchitis:
1) Exogenous factors - smoking: inhalation of polluted air influence of occupational hazards and cold climate
2) Endogenous factors - reduced immunity, infections, hereditary predisposition (violation of enzyme systems - α1-antitrypsin, local immunity), metabolic disorders (obesity).
Pathogenesis of chronic bronchitis.
1. Disturbance of function of local bronchopulmonary defense system and immune system
2. Structural changes in bronchial mucosa
3. Development of classical pathogenetic triad
Clinical classification of chronic bronchitis
There is no unified classification of chronic bronchitis.
According to the nature of inflammation are distinguished:
- catarrhal;
- purulent.
According to the phase of the disease:
- exacerbation;
- remission.
Clinical picture of chronic bronchitis.
Subjective:
1) cough
2) sputum discharge
3) shortness of breath
Objectively:
1) on examination no significant changes are detected; during exacerbation of the disease sweating, elevation of body temperature to subfebrile numbers may be observed.
2) percussively clear pulmonary sound, with the development of emphysema - cranial sound.
3) auscultatory lengthening of exhalation, rigid breathing ("roughness", "roughness" of vesicular breathing), dry rales (due to the presence of viscous sputum in the lumen of bronchi, in large bronchi - bass low-tone, in medium bronchi - buzzing, in small bronchi - whistling). If there is liquid sputum in the bronchi, wet rales (large bubbly in large bronchi, medium bubbly in medium bronchi, small

bubbly in small bronchi). Dry and moist rales are unstable and may disappear after vigorous coughing and expectoration of sputum.

Diagnosis of chronic bronchitis.

1. Laboratory data:

a) GBT

b) sputum analysis

c) BC

2. Instrumental studies:

a) bronchoscopy

b) bronchography

c) lung X-ray

d) examination of external respiratory function (spirography, peak flowmetry)

Complications of CHB.

1) directly caused by infection: a) pneumonia; b) bronchiectasis; c) bronchoobstructive 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 disease.

Treatment of CHB is different in the period of remission and in the period of exacerbation.

- Elimination of the inflammatory process in the bronchi;
- relief of symptoms of respiratory failure;
- relief of the severity and duration of coughing;
- elimination of symptoms of intoxication, improvement of well-being, normalization of body temperature, recovery and prevention of complications;
- restoration of ability to work.

Outcome of chronic bronchitis: in obstructive form or CB with lesions of the distal parts of the lungs the disease quickly leads to the development of pulmonary insufficiency and formation of pulmonary heart.

4.Illustrative material: slides in MicrosoftOfficePowerPoint.

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.Give the definition of acute and chronic bronchitis?
2.Name the etiological factors of bronchitis.
3.Name the classification of acute and chronic bronchitis
4. Name the syndromes and symptoms of bronchitis.
5.Diagnosis, management tactics

Lecture №2

1. Topic: Pneumonia. COVID-19-associated pneumonia.

2.The purpose: to acquaint the students with an introduction to the section of clinical medicine-pulmonology, to give a general idea about diseases of the respiratory organs.
The lecture contains data on epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.

3. lecture abstracts:

Pneumonia is an acute infectious disease that occurs in an out-of-hospital setting (i.e. out-of-hospital or later than 4 weeks after discharge from the hospital, or diagnosed within the first 48 h of admission, or developed in a patient who has not been in a nursing home/ long-term care unit for ≥14 days accompanied by symptoms of lower respiratory tract infection (fever, cough, sputum discharge, possibly purulent, chest pain, shortness of breath) and radiological evidence of "fresh" focal infiltrative lung changes in the absence of an obvious diagnostic alternative.
Etiology:
Infectious - developed by pneumococci, Klebsiella, staphylococci, streptococci, and other bacteria;
viral genesis - the most common is the herpetic form when affected by Epstein-Barr or cytomegalovirus;
fungal genesis - the causative agents can be fungi - molds (Aspergillus, Mucor), yeast-like (Candida), endemic dimorphs (Blastomyces, Coccidioides, Histoplasma), pneumocysts (Pneumocystis);
mixed type - caused simultaneously by two or more types of pathogens.
Transmission: is transmitted by airborne droplets, by inhalation of germs from a sick person.
Classification of pneumonia:
According to the conditions of occurrence, pneumonia can be: 1) nosocomial; 2) nosocomial; 3) aspiration; 4) in persons with severe immunodeficiencies.
According to the criterion of the spread of the process, pneumonia can be:
1) focal - that is, occupy a small focus of the lung (bronchopneumonia - respiratory departments + bronchi);
2) segmental - spreading to one or more segments of the lung;
3) lobular - involving a lobe of the lung. A classic example of lobular pneumonia is lobar pneumonia - predominantly alveoli and adjacent area of the pleura;
4) confluent - merging of small foci into larger ones;
5) total - if it spreads to the entire lung.
Syndromes and symptoms:
1)Intoxication (general weakness, brokenness, headaches, muscle pain, shortness of breath, palpitations, pallor, decreased appetite).
2)Syndrome of general inflammatory changes (fever, chills, increased body temperature, changes in acute phase blood parameters: leukocytosis, neutrophilic shift, increase in SLE, fibrinogen, α2-globulin levels, appearance of C-reactive protein - CRP).
3) Syndrome of inflammatory changes of pulmonary tissue (appearance of cough and sputum, shortening of percussion sound, increase of vocal trembling and bronchophony, changes in frequency and character of breathing, appearance of wet rales, characteristic radiological changes).
4)Syndrome of involvement of other organs and systems (changes in the cardiovascular system, gastrointestinal tract, kidneys, nervous system).
Diagnosis of pneumonia:
Collection of complaints and anamnesis
Physical examination (auscultation, percussion, palpation of the lungs)
Laboratory tests (GBA, B/X, CRP, Procalcitonin, sputum smear test, coagulogram, ELISA for mycoplasma, acid test, BMV, poisoning gas)
Instrumental examinations (X-ray of the OGC, CT scan of the OGC, ECG, EchoCG)

Treatment
Nonmedicamental treatment:
- to reduce intoxication syndrome and facilitate sputum secretion - maintenance of adequate water balance (adequate fluid intake);
- cessation of smoking;
- elimination of the patient's exposure to environmental factors that cause cough (smoke, dust, sharp smells, cold air).
Drug treatment:
The main drugs for the treatment of community-acquired pneumonia are antibacterial drugs.Usually empirical ABT is carried out.
Hospitalization is recommended only in very severe cases.
Coronavirus infection (COVID19) is an acute infectious disease caused by a new strain of the SARS CoV-2 coronavirus with aerosol-drop and contact-domestic transmission.
Pathogenetically COVID-19 is characterized by viremia, local and systemic immunoinflammatory process, endotheliopathy, hyperactivity of coagulation cascade, which may lead to the development of micro-macrothrombosis and hypoxia. Clinically it runs from asymptomatic to manifest forms with intoxication, fever, predominantly lung lesions and extrapulmonary lesions of various organs and systems
(vascular endothelium, heart, kidney, liver, pancreas, intestine, prostate, central and peripheral nervous system) with high risk of complications (ARDS, ODN, TELA, sepsis, shock, SPON, OSN).
Clinical picture:
Incubation period is 2-14 days.
Increased body temperature (or no fever), general weakness, malaise,
Sweating, myalgia and body aches, headache, sore throat, rare dry cough with small amount of sputum, feeling of tightness, burning, pain, chest tightness (inability to inhale deeply), taste and smell disorders, diarrhea, restless behavior (agitation), conjunctivitis.
Diagnosis:
Collection of complaints and anamnesis.
Physical examination (auscultation, percussion, palpation of the lungs)
Laboratory tests (PCR for COVID-19, OAC, B/X, CRP, Procalcitonin, sputum smear test, coagulogram, ELISA for mycoplasma, BAC, AIM)
Instrumental examinations (X-ray of the OGC, CT scan of the OGC, ECG, EchoCG)
Treatment:
Nonmedicamental treatment: bed rest (depending on the severity of the course, change of the body position in bed is advisable, walking in the room - under the control of the patient's condition (BP, HR, oxygen saturation).
Drug therapy: Currently, there is no convincing evidence of an effective specific therapy for COVID-19-induced disease, so the main principle in the management of patients with a confirmed diagnosis of COVID-19 remains optimal pathogenetic treatment depending on the nature of clinical symptoms, severity of the disease, presence/absence of pneumonia (X-ray and CT/signs), type and degree of complications, comorbidities, which is carried out to relieve symptoms and maintain organ and system function in a more severe course.

4.Illustrative material: slides in MicrosoftOfficePowerPoint.

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.Define pneumonia and COVID associated pneumonia.
2.Name the main etiological factors of pneumonia.
3.Name the major syndromes.
4. State the diagnostic criteria.

Lecture №3

1. Topic: Chronic Obstructive Pulmonary Disease (COPD).
2. Purpose: To acquaint students with an introduction to the section of clinical medicine-pulmonology, to give a general idea about diseases of the respiratory organs.
The lecture contains data on epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. lecture abstracts:
Chronic obstructive pulmonary disease (COPD) is a primary chronic inflammatory disease with predominant involvement of the distal parts of the airways, lung parenchyma and the formation of emphysema; it is characterized by restriction of airflow with the development of irreversible or partially reversible bronchial obstruction. Decreased airway patency is progressive and associated with an inflammatory response of the lungs to dust particles or smoke, tobacco smoking, and air pollution.
Risk Factors
The primary cause of COPD is tobacco smoke (including inhalation of secondary tobacco smoke, or secondhand smoke). Other risk factors include:
- Indoor air pollution (e.g., as a result of using solid fuels for cooking and heating);
- Ambient air pollution;
- presence of dust and chemicals in the workplace (fumes, irritants, and fumes);
- frequent lower respiratory tract infections in childhood.
COPD classification:
1. Stage 0. COPD risk stage - chronic cough and sputum production; lung function normal.
2. Stage I. Mild COPD - unexpressed (mild) bronchoobstruction (SPH1/EL <70%, but SPH1 ≥80% of proper values) and usually, but not always, there is chronic cough and sputum production.
3. Stage II. COPD of moderate severity - progression of bronchoobstruction (50% ≤OEF1<80% of proper values) and usually progression of symptoms, dyspnea develops with exercise.
4. Stage III. Severe COPD - further progression of bronchoobstruction (30% ≤OEF1<50% of proper values), progression of dyspnea and frequent exacerbations that affect quality of life.
5. Stage IV. Very severe COPD - severe bronchoobstruction OEF1<30% of proper values) or the presence of chronic respiratory failure. A patient may have (stage IV) even when the BEF>30% of the proper values, but in the presence of these complications.
Symptoms
- chronic cough (daily, often all day long; occasionally only at night);
- Chronic sputum production (any case of chronic sputum production may indicate COPD);
- Acute bronchitis (repeatedly recurring);
- Dyspnea (progressive, persistent; increased with exercise, respiratory tract infections);

Diagnosis:
Collection of complaints and history
Physical examination (auscultation, percussion, palpation of the lungs)
Laboratory tests (GBA, B/X, CRP, Procalcitonin, sputum smear test, coagulogram, ELISA for mycoplasmas, acid test, AIM)
Instrumental examinations (X-ray of the OGC, CT scan of the OGC, pulse oximetry, ECG, EchoCG)
Treatment:
The basis of symptomatic treatment of COPD is bronchodilators, preferably by inhalation. Chronic obstructive pulmonary disease is incurable. However, available medications and physical therapy can relieve symptoms, improve exercise tolerance and quality of life, and reduce the risk of death. The most effective and cost-effective treatment for COPD in smokers is to stop smoking. This will slow the course of the disease and reduce mortality from COPD-related causes. In some (but not all) COPD patients, prescribing inhaled corticosteroids has a positive effect.
4.Illustrative material: slides in the program MicrosoftOfficePowerPoint.
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 COPD?
2. Name the causes leading to the development of COPD.
3. What testing methods are needed to make a diagnosis of COPD?
4. 4. Name the complications of COPD.

Lecture №4

1. Topic: Bronchial asthma.
2.The purpose: To acquaint the students with an introduction to the section of clinical medicine-pulmonology, to give a general idea of diseases of the respiratory organs.
The lecture contains data on epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. lecture abstracts:
Bronchial asthma (BA) is a heterogeneous disease characterized by chronic inflammation of the airways, the presence of respiratory symptoms such as wheezing, shortness of breath, chest congestion and cough, which vary in time and intensity and occur together with variable airway obstruction.
AD in terms of etiology is divided into allergic (most often begins in childhood, often accompanied by other atopic diseases, usually eosinophilia induced sputum and good response to inhaled GCS) and nonallergic (usually in adults, often worse response to inhaled GCS). In addition, phenotypes of AD are distinguished:
1) with late onset;
2) with permanent bronchial obstruction;
3) accompanied by obesity.

In allergic asthma, allergen binding 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 the allergic reaction, a late phase occurs in which mast cells, basophils and other cells release cytokines and chemokines that multiply the influx of inflammatory cells, particularly eosinophils, into the bronchi. The pathomechanism of nonallergic asthma has not been fully investigated, but the histopathological picture is similar to that of allergic asthma. Damage to the bronchial epithelium stimulates reparation processes, resulting in rearrangement of the bronchial wall, which leads to the fact that in especially severe cases the obstruction becomes irreversible.

Factors that cause attacks and exacerbations of AD or provoke their persistence: allergens, respiratory tract infections (mainly viral), air pollution (including tobacco smoke, aerosols used in households, paint fumes, etc.), physical activity, strong emotions, weather changes, drugs (β-blockers, NSAIDs), food and food supplements.

Factors that increase the risk of AD exacerbations: uncontrolled AD symptoms (including, excessive consumption of short-acting β2-agonists (monthly >1 package containing 200 doses), inappropriate use of inhaled GCS (failure to take the patient's prescribed medication, improper inhalation technique), low SPH1 (especially <60% of proper), serious psychological or socioeconomic problems, exposure to tobacco smoke or allergens (in sensitized individuals), comorbidities (obesity, nasal and paranasal sinus mucosal inflammations, food allergies), sputum or blood eosinophilia, pregnancy, ≥1 severe AD exacerbation in the past 12 months, previous intubation or treatment in an ITU for AD.

Risk factors for bronchial obstruction fixation: non-use of inhaled GCS, exposure to tobacco smoke or other harmful substances (including in the workplace), low baseline SPH1, chronic excessive airway secretion production, sputum or blood eosinophilia.

Clinical picture and natural course

1. Subjective symptoms: paroxysmal dyspnea, mostly expiratory (sometimes felt as chest constriction), which subsides spontaneously or under the influence of treatment; wheezing; dry, intermittent cough (accompanying dyspnea or as the only symptom the so-called cough variant of asthma; isolated cough in adults is rarely a symptom of AD). Patients with allergic AD have concomitant symptoms of other allergic diseases, most commonly allergic rhinitis. Subjective and objective symptoms are variable and may not occur except during episodes of attacks and exacerbations of AD.

2. objective symptoms: diffuse, bilateral wheezing (mostly expiratory) and dry wheezing, prolonged exhalation (sometimes symptoms that are heard only on forced exhalation); during exacerbations, auxiliary muscle work 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 minutes or hours) or gradually (within hours or days) and without treatment can lead to death. Years of uncontrolled AD lead to progressive, irreversible airway obstruction.

Diagnosis

Additional research methods

1. Spirometry: most patients have a baseline spirometry result within normal limits. AF is characterized by obstruction, especially with variable intensity (significant fluctuations between consecutive tests, or under the influence of treatment); with bronchodilation testing, significant improvement in BEF1 and/or EFEL (≥200 ml and ≥12% of proper) and often even elimination of obstruction (in severe AF or in AD with bronchial remodeling, obstruction may be irreversible), and bronchial hyperresponsiveness in provocation testing with methacholine or histamine. In special cases, the diagnosis can be confirmed by specific provocation tests with allergen, acetylsalicylic acid, factors present in the workplace, and physical exertion.

2. Peak expiratory velocity (PEF): the average (over 2 weeks of measurements) daily variability of PEF is characteristic; measurements are used to confirm the diagnosis, monitor the disease (should be considered appropriate in patients with severe bronchial asthma or with poor symptom perception) and identify factors that provoke symptoms (e.g. occupational factors).
3. Chest X-ray: usually normal, with exacerbations there may be signs of pulmonary hyperpneumatization (air traps) and exacerbation complications (e.g. pneumothorax).
4. Pulse oximetry and arterial blood gasometry: use to assess severity and monitor the course of exacerbations → see below.
5. Studies determining IgE-mediated allergies: skin tests, total and specific IgE concentrations - can determine the sensitizing allergen in a patient with allergic asthma.
6. Induced sputum examination for eosinophilia: in centers with experience, may be used to modify treatment in patients with moderate to severe AD.
7. Exhaled nitric oxide concentration (FENO) study: as an additional study in differential diagnosis with COPD. In previously untreated patients, an elevated value (>50 ppb) correlates with a good response to inhaled GCS treatment.
The diagnosis of AD (according to GINA) requires the presence of symptoms of the disease, as well as changes in the severity of bronchial obstruction on functional tests. The severity of the disease must be assessed.
Treatment goals are:
1) achieving and maintaining symptom control and normal vital activity (including the ability to tolerate exercise);
2) minimizing the risk of exacerbations, prolonged bronchoobstruction, and adverse effects of therapy.
4.Illustrative material: slides in the program Microsoft Office Power Point.
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 BA?
2.Name the etiological factors of AD development.
3.Name the main syndromes.
4.State the treatment and further tactics.

Lecture №5

1. Topic: Pleurisy.
2. Purpose: To acquaint students with an introduction to the section of clinical medicine-pulmonology, to give a general idea about diseases of the respiratory organs.
The lecture contains data on epidemiology, etiology and pathogenesis of the disease, its clinical manifestations, differential diagnosis, complications and treatment.
3. lecture abstracts:

Pleurisy - inflammation of the pleura, the membrane covering the lungs and the wall of the chest cavity.When the pleura is irritated, a disease such as pleurisy develops. It leads to inflammation of the membranes that surround the lungs and line the chest cavity. Hippocrates described the clinical signs of the disease as early as 400 B.C. Fluid, called pleural effusion or exudate, may accumulate in the pleural cavity. The most common symptom of pleurisy is sudden onset of pain in the chest. During breathing, this pain is felt as a vague discomfort and may become sharp, intense, and acute. Breathing is usually rapid and shallow, as deep breathing increases the pain.Other symptoms include shortness of breath, coughing, fever, or weight loss, depending on the underlying cause. The most common is a viral infection. Other causes include pneumonia, pulmonary embolism, autoimmune disorders, lung cancer, complications from heart surgery, tuberculosis, pancreatitis, chest trauma (rib fracture), asbestosis, and drug-induced allergic reactions. Sometimes the cause remains unknown. Other conditions that may cause similar symptoms include pericarditis, heart attack, cholecystitis, and pneumothorax.

Diagnosis and treatment:

Collection of complaints and history.

Physical examination.

Laboratory tests (GBA, B/C, CRP, Coagulogram, Immunology ANA; Rheumatoid Factor; OAB)

Instrumental studies (chest X-ray, puncture of the pleural cavity, followed by examination of the pleural contents, CT OGC, ECG, EchoCG, ultrasound of the chest cavity, a functional study of lung function-spirometry).

Treatment:

Treatment of pleurisy depends on its specific cause. For example, antibiotics are prescribed for bacterial infections, but no specific treatment is needed for viral infections. Analgesics such as paracetamol or ibuprofen can help reduce chest pain regardless of the cause of pleurisy.

4.Illustrative material: slides in Microsoft Office Power Point.

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. Give the definition of pleurisy.
2. What are the main types of pleurisy do you know?
3. Give a description of the pain syndrome in pleurisy

Lecture №6

1. Topic: Glomerulonephritis acute and chronic

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

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

3. lecture abstracts:

Glomerulonephritis (GN) is a heterogeneous group of diseases characterized by inflammation exclusively or mainly of the glomerular apparatus of the kidneys. Changes associated with other renal structures (tubules, interstitial substance, vessels) are secondary and result from abnormalities (mainly proteinuria) caused by abnormalities of the glomeruli. At the origin of inflammation are pathological immunological processes, in many cases the causes and pathogenesis are unknown. In the course of GN can occur exacerbations, relapses and remissions.

Primary GN: the disease affects only the glomeruli, and clinical symptoms and laboratory abnormalities are the result of structural and functional abnormalities in the glomeruli. In some cases of primary GN the cause is known (e.g. postinfection, postvaccination GN); in most cases it is idiopathic.

Secondary HN: glomerular damage is the result of another pathological process, often multiorgan 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, because very similar histopathological changes can develop different clinical forms. In addition, the clinical presentation of a particular type of GN varies at different stages (e.g. initially nephrotic syndrome, then chronic GN, or at first asymptomatic microhematuria and then rapidly progressing GN, which is the result of different disease activity and sometimes transformation or crossing of one GN with another. For these reasons, HN is classified on the basis of the histopathological picture, because it indicates the pathogenesis of HN and the nature of damage to the kidney structures, which are crucial for the choice of treatment and prognosis assessment. Most types of GN can be primary or secondary glomerulopathy, and the diseases occur under different clinical forms

Clinical presentation

There are 5 variants of glomerulonephritis:

1. Hypertensive (20% of cases) pronounced hypertension - diastolic pressure higher than 95 mm Hg.
2. Nephrotic (20%) - large loss of protein with urine up to 10-20 grams per day, hypoalbuminemia in the blood, marked edema of the extremities, hydrothorax, ascites, anasarca.
3. Combined form. The most typical combination of the previous two, a steady, progressive course. In all three forms of the disease, there are necessarily changes in the urinalysis (hematuria and proteinuria).
4. Hematuric form. Berger's disease, IgA nephritis (recurrent hematuria, edema and AH).
5. Latent or urinary form. The most common form. Appears only in changes in urinalysis - microhematuria, moderate proteinuria - small amount of protein.

Diagnosis and treatment:

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

2. blood tests: increased serum creatinine levels when HN results in decreased FFR; immunological markers are present in certain variants of HN.

3. renal biopsy: the only accurate way to diagnose HN and its type. Necessary if primary GN is suspected (except: Nephrotic syndrome in children; ≈80% of cases are caused by GN with minimal changes) in a patient with nephrotic syndrome, rapidly progressive renal failure, or microhematuria (after excluding its cause in the urinary tract), because the choice of treatment, including immunosuppressive therapy, and prognosis depend on the histopathologic type of GN, the degree of renal glomerular damage, and the severity of secondary tubulointerstitial changes.

An accurate histopathological diagnosis is also necessary in many cases of suspected secondary glomerulonephritis, e.g. to confirm the diagnosis of a systemic disease or type of glomerulonephritis

in a previously diagnosed systemic disease (systemic lupus erythematosus), or when there is doubt about the nature and extent of renal tubular damage.

1. Primary HN: immunosuppressive therapy, reducing the rate of progression of CKD and controlling the factors that accelerate this progression (arterial hypertension, proteinuria, hyperlipidemia, smoking, urinary outflow obstruction).

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

4.Illustrative material: slides in Microsoft Office Power Point.

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. Name the causes of occurrence of GN.
2. State the diagnostic criteria of glomerulonephritis.
3. State the main principles of treatment.

Lecture №7

1. Topic: Acute Renal Failure

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

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

3. lecture abstracts:

Acute renal failure is a sudden onset, potentially reversible impairment of homeostatic renal function. Currently, the incidence of acute renal failure reaches 200 per 1 million population, with 50% of those in need of hemodialysis. Since 1990s there has been a steady tendency of acute renal failure to become more often not a monoorganic pathology, but a component of multiple organ failure syndrome.

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

Prerenal acute renal failure is caused by impaired hemodynamics and decreased total circulating blood volume, which is accompanied by renal vasoconstriction and decreased renal circulation. This results in renal hypoperfusion, blood is insufficiently purified from nitrogenous metabolites, and 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 of the renal parenchyma, less often by acute renal inflammation and vascular pathology. In 75% of patients with renal acute renal failure the disease occurs against the background of acute tubular necrosis.

Postrenal acute renal failure is more often accompanied by anuria and occurs as a result of obstruction at any level of 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 decreased cardiac output. Another cause may be marked 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 of the liver and the resulting ascites.

Renal acute renal failure is caused by exposure of the kidney to toxic substances: salts of mercury, uranium, cadmium, copper. A pronounced nephrotoxic effect is possessed by poisonous fungi and some drugs, primarily aminoglycosides, the use of which in 5-20% of cases is complicated by moderate acute renal failure and in 1-2% - expressed. In 6-8% of all cases of acute renal failure develops against the background of the use of nonsteroidal anti-inflammatory drugs. X-ray contrast agents have nephrotoxic properties, which requires careful use in patients with impaired renal function. Hemoglobin and myoglobin circulating in the blood in large quantities can also cause the development of renal acute renal failure. This is caused by massive hemolysis caused by incompatible blood transfusions and hemoglobinuria. The causes of rhabdomyolysis and myoglobinuria can be traumatic, such as crush syndrome, and non-traumatic, related to muscle damage in a prolonged alcoholic or drug-induced coma.

Postrenal acute renal failure accounts for about 5% of all cases of renal dysfunction. It is caused by mechanical impairment of the outflow of urine from the kidneys, most often due to obstruction by concrements 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 tuberculosis lesions of the urinary tract, metastases of breast or uterine cancer in retroperitoneal tissue, bilateral sclerotic periureteritis of unknown genesis, dystrophic processes of retroperitoneal tissue.

Pathogenesis. In acute renal failure due to prerenal factors, the cause triggering the pathological mechanism is renal parenchymal ischemia. Even a short-term decrease in blood pressure below 80 mm Hg leads to a sharp decrease in blood flow in the renal parenchyma due to shunt activation in the juxtamedullary zone. Such a condition can occur in case of shock of any etiology, as well as as a result of bleeding, including surgical intervention. In response to ischemia, necrosis and rejection of proximal tubule epithelium begins, and the process often reaches acute tubular necrosis. Sodium reabsorption is sharply impaired, which leads to its increased inflow into the macula densa and stimulates renin production, which supports spasm of bringing arterioles and ischemization of parenchyma.

In toxic lesions, the epithelium of the proximal tubules is also most commonly affected, and in the case of toxic exposure to myoglobin and hemoglobin pigments, the situation is aggravated by the tubule's obturation by these proteins.

In postrenal acute renal failure, impaired outflow of urine from the kidneys causes overstretching of ureters, pelvis, collecting tubules and distal and proximal nephron. Massive interstitial edema is the consequence. If outflow of urine is restored quickly enough, renal changes are reversible, but with prolonged obstruction severe circulatory disorders of the kidneys occur, which may end in tubular necrosis.

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

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

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

Diuretic phase lasts for 9-11 days. The amount of urine gradually begins to increase and after 4-5 days reaches 2-4 liters per day or more. Many patients lose a large amount of urine potassium - hyperkalemia

is replaced by hypokalemia, which may lead to hypotension and even skeletal muscle paresis, disorders of cardiac rhythm. Urine has low density, creatinine and urea content is reduced, but after 1 week of diuretic phase with a favorable course of the disease the hyperazotemia disappears and electrolyte balance is restored.

In the phase of complete recovery there is further restoration of renal function. This period lasts for 6-12 months, after which renal function completely recovers.

Diagnosis. Diagnosis of acute renal failure, as a rule, presents no difficulty. Its main marker is continuous increase of nitrogenous metabolites and potassium level in blood along with decrease of urine output. In a patient with clinical manifestations of acute renal failure it is mandatory to determine its cause. The differential diagnosis of prerenal acute renal failure from renal failure is extremely important, since the first form can quickly transition to the second, which will aggravate the course of the disease and worsen the prognosis. First of all, it is necessary to make differential diagnosis of postrenal acute renal failure from its other types, for which renal ultrasound is performed to determine or exclude bilateral obstruction of the upper urinary tract by the presence or absence of dilatation of the calyx-pelvic system.

Treatment. In the initial phase of acute renal failure, treatment should primarily be aimed at eliminating the cause of the pathological mechanism. In shock, which is the cause of 90% of acute renal failure, the main therapy is aimed at normalization of arterial pressure and replenishment of the circulating blood volume. Protein solutions and large molecular weight dextrans are effective, which should be administered under control of central venous pressure, so as not to cause hyperhydration.

When poisoning with nephrotoxic poisons it is necessary to remove them by gastric and intestinal lavage. Unithiol is a universal antidote for poisoning with salts of heavy metals. Hemosorption taken before acute renal failure develops can be particularly effective.

In case of post-renal acute renal failure therapy should be aimed at early restoration of urine outflow.

In oliguric phase in acute renal failure of any etiology it is necessary to administer osmotic diuretics in combination with furosemide, doses of which can reach 200 mg. Dopamine is indicated in "renal" doses, 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. Patients' diet 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 carry out surgical treatment of wounds with removal of necrotic areas, drainage of cavities. At the same time antibiotic therapy should be carried out taking into account the severity of kidney damage.

Hemodialysis is indicated for increased potassium over 7 mmol/l, urea up to 24 mmol/l, uremic symptoms: nausea, vomiting, lethargy, as well as hyperhydration and acidosis. Nowadays, early or even prophylactic hemodialysis is increasingly used, which prevents the development of severe metabolic complications. This procedure is performed every day or every other day, gradually increasing protein quota to 40 g/day.

Complications. Prognosis. Lethality in acute renal failure depends on the severity of the course, age of the patient, and most importantly - the severity of the underlying disease that caused the development of acute renal failure. In patients surviving after acute renal failure, complete recovery of renal function is observed in 35-40% of cases, partial - in 10-15%, and from 1 to 3% of patients need permanent hemodialysis. The latter figure depends on the genesis of acute renal failure: in renal forms the need for permanent dialysis reaches 41%, whereas in traumatic acute renal failure this figure does not exceed 3%.

The most frequent complication of acute renal failure is urinary tract infection with further development of chronic pyelonephritis and the outcome in chronic renal failure.

4.Illustrative material: slides in Microsoft Office Power Point.

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 do you know the main complaints in diseases of the genitourinary system?
2. What do you need to pay attention to during the general examination of patients with kidney disease?
3 State the reasons for the development of ARF.
4.State the indications for hemodialysis in ARF.

Lecture №8

1. Topic: Chronic Kidney Disease

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

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

3. lecture abstracts:

Chronic renal insufficiency is a syndrome of irreversible impairment of all renal functions lasting for months or years, leading to disorders of water, electrolyte, nitrogen and other types of metabolism, caused by the development of sclerosis of renal tissue due to various renal diseases.

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

Etiology

- Arterial hypertension
- Diabetes mellitus
- Hyperlipidemia
- Obesity
- Smoking
- Lower urinary tract infections and obstruction
- Autoimmune diseases
- Hereditary burden (CKD in relatives)
- Systemic infections, acute renal failure
- Drug-induced renal impairment
- Old age
- Toxic renal damage (alcohol and its surrogates, exposure to lead, mercury, fungicides, disinfectants, heroin, organic solvents)

Pathophysiology

Early stages of CKD are described as reduced renal reserve or renal failure that may progress (terminal renal failure). Initially, the loss of renal tissue function has almost no obvious pathological manifestations because the remaining tissue is working hard (functional renal adaptation).

Reduced 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 joins the decline in the ability to excrete excessive phosphate, acid and potassium. In severe renal insufficiency (GFR ≤15 ml/min/1.73 m2), the ability to effectively dilute or concentrate urine is lost. Thus, urine osmolarity is usually about 300-320 mmol/kg, approaching plasma osmolarity (275-295 mmol/kg), and urine volume does not immediately respond to changes in fluid volume drunk.

Clinical manifestations

With a moderate decrease in renal reserve, the course is usually asymptomatic. Even patients with mild to moderate renal failure may have no symptoms of elevated AMK and creatinine levels. Nycturia is often observed, especially due to the inability to concentrate urine. Apathy, fatigue, lack of appetite, and decreased mental clarity are often the earliest manifestations of uremia. In more severe renal disease (e.g., estimated glomerular filtration rate [rGFR] < 15 mL/min/1.73 m2), neuromuscular symptoms, including marked muscle twitching, peripheral sensory and motor neuropathies, muscle seizures, hyperreflexia, restless leg syndrome, and seizures (usually resulting from hypertensive or metabolic encephalopathy) may appear. Anorexia, nausea, vomiting, weight loss, stomatitis, and a bad taste in the mouth are very common. The shade of the skin may become yellow-brown. Sometimes urea crystals are excreted with sweat to the surface of the skin, forming uremic frosting. Itching can cause serious discomfort. Nutritional deficiencies resulting in generalized tissue loss are a hallmark of chronic uremia.

Diagnosis

Laboratory tests:

- general blood count
- Blood clotting is decreased;
- biochemical blood analysis (creatinine, urea, lipid spectrum, electrolytes (phosphorus, potassium, magnesium, calcium, chlorine)
- acid-base state
- urine analysis

Instrumental studies:

- Renal ultrasound, EchoCG, retrograde pyelography,renal biopsy, radioisotopic renography (scintigraphy)

Treatment

- Treatment of the underlying disease
- Restriction of protein, phosphate, and potassium in the diet as much as possible
- Vitamin D supplementation
- Treatment of anemia
- Treatment of comorbidities (e.g., heart failure, diabetes mellitus, nephrolithiasis, prostatic hypertrophy)
- Hemodialysis for significant declines in FFR if symptoms and signs are not sufficiently amenable to drug intervention

4.Illustrative material: slides in Microsoft Office Power Point.

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. Name the main causes of CKD development.
2. What do you need to pay attention to during the general examination of patients with kidney disease?
3. What are the clinical manifestations of CKD?

Lecture № 9

1. Topic: Arterial hypertension.

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

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

3. lecture abstracts:

Arterial hypertension - office BP ≥140 mmHg, and/or BP ≥90 mmHg.

White coat hypertension - on repeated visits to the medical facility, BP appears elevated, but outside, on CMAD or DMAD, normal. But cardiovascular risk is low compared with patients with persistent AH (no diabetes, target organ damage, cardiovascular disease, or CVD).

Masked hypertension - BP may be normal in the office and abnormally elevated outside the institution, but cardiovascular risk is in the range consistent with persistent AH.

The terms White Coat Hypertension and Masked Hypertension are recommended for patients not receiving treatment.

Resistant AH - treatment with optimal (or maximum tolerated) doses of medications involving a combination of three classes of first-line drugs, including a diuretic (iAPP or ARA II combined with BCC and a thiazide/thiazide-like diuretic), does not reduce BP and BP to values <140 mmHg and/or <90 mmHg, respectively.

Emergency hypertension (hypertensive crisis) - severe hypertension (often grade 3) with signs of acute target organ damage requiring most often immediate, but cautious BP reduction usually by intravenous therapy:

- Hypertensive encephalopathy
- Acute heart failure
- Acute coronary syndrome
- Acute cerebral blood flow disorder
- Acute aortic dissection
- Hypertensive retinopathy (hemorrhages and/or optic disc edema)
- Acute renal failure
- Preeclampsia and eclampsia

Arterial hypertension can be:

- Primary (85% of cases)
- Secondary

Classification of office BP and determining the degree of AH (mmHg)

BP categories	**SBP**		**DBP**
Optimal	< 120	and	< 80
Normal	120 - 129	and/or	80 - 84

High normal	130-139	and/or	85 - 89
AH grade 1	140 - 159	and/or	90 - 99
AH grade 2	160 - 179	and/or	100 - 109
AH grade 3	≥ 180	and/or	≥ 110
Isolated systolic AH	≥ 140	and/or	< 90

Clinical manifestations

Arterial hypertension is usually asymptomatic until complications develop in the target organs. Uncomplicated arterial hypertension can cause dizziness, facial flushing, headache, increased fatigue, nosebleeds, and hyperexcitability. Severe hypertension (hypertensive crisis) can cause serious cardiovascular, neurological, renal, and retinal symptoms (e.g., symptomatic coronary artery atherosclerosis, CHF, hypertensive encephalopathy, renal failure).

The presence of a 4th heart tone is one of the earliest signs of hypertensive cardiomyopathy.

Diagnosis

- Multiple BP measurements to confirm, CMAD.
- Urinalysis and urine albumin/creatinine ratio; if there are abnormalities, consider the need for a renal ultrasound
- Blood tests: fasting lipid, creatinine, potassium.
- Renal ultrasound for elevated creatinine
- Need to assess the presence of aldosteronism if potassium is decreased
- ECG: echocardiography is necessary if left ventricular hypertrophy is present
- Sometimes thyrotropic hormone determination
- Assess for pheochromocytoma or sleep disturbances if BP is labile and rises suddenly or if there is severe arterial hypertension

The more severe the arterial hypertension and the younger the patient, the more extensive the examination should be. Generally, when arterial hypertension is newly diagnosed, routine examinations are performed to:

- Detecting target organ damage
- Determination of cardiovascular risk factors

Examinations include:

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

Prognosis

The higher the blood pressure values and the more severe the retinal changes and if there are other signs of target organ damage, the worse the prognosis. Systolic BP is a predictor of fatal and nonfatal cardiovascular events to a greater extent than diastolic BP. Without treatment, the 1-year survival rate is 10% in patients with retinal sclerosis, cottonwood exudates, arteriolar narrowing, and hemorrhage (grade 3 retinopathy) and 5% in patients with the same changes combined with optic disc edema (grade 4 retinopathy). CHD is the most common cause of death among patients with treated arterial hypertension. Ischemic or hemorrhagic stroke is the most frequent consequence of inadequate treatment of arterial hypertension. However, effective control of arterial hypertension prevents most complications and prolongs life.

Treatment

- Weight loss and physical activity
- Giving up bad habits (smoking, alcohol, etc.).
- Plateau No. 10: more fruit and vegetable intake, less salt, less water, limited alcohol intake.
- Medicines: Depending on BP and the presence of cardiovascular disease or risk factors

Primary hypertension cannot be cured, but some causes of secondary hypertension can be treated. In all cases, blood pressure control can significantly limit adverse effects. Despite the theoretical efficacy of treatment, BP is reduced to the desired level in only one-third of hypertensive patients.

4.Illustrative material: slides in Microsoft Office Power Point.

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. Give the definition of AH.
2. What are the causes of AH?
3. What diagnostic methods are used to determine AH?
4. Name the risk factors for AH.
5. What complications of AH do you know?

Lecture №10

1. Topic: Atherosclerosis

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

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

3.Lecture abstracts: Atherosclerosis - is an umbrella term for several diseases that cause thickening and loss of elasticity of arterial walls.

Atherosclerosis is a variable combination of changes in the inner lining (intima) of arteries, including accumulation of lipids, complex carbohydrates, fibrous tissue, blood components, calcification, and associated changes in the middle lining (median) (WHO).

Risk Factors.

1. Hyperlipidemia - increased plasma levels of total cholesterol (above 5 mmol/L).
2.Dyslipoproteinemia - increased low-density lipoprotein cholesterol (LDL) (above 3 mmol/l), decreased high-density lipoprotein cholesterol (HDL) (below 1 mmol/l in men, below 1.3 mmol/l in women), triglyceride levels above 1.7 mmol/l.
3. Tobacco smoking.
4. arterial hypertension.
5. Family history of cardiovascular disease at a young age (in men under 55 years, in women under 65 years).
6. Age - men over 45 years old, women over 55 years old.
7. Disorder of carbohydrate metabolism.

8. High levels of CRP, fibrinogen, homocysteine, apoliprotein B or Lp-a with normal levels of total and LDL cholesterol.

The presence of one of these risk factors as the only one can lead to the development of atherosclerosis and, as a consequence, cardiovascular disease:

- Total cholesterol > 8 mmol/L;
- LDL cholesterol > 6 mmol/l;
- BP > 180/110 mmHg;
- Type 2 diabetes or type 1 diabetes accompanied by microalbuminuria.

Additional risk factors:

1. Gout.
2. obesity.
3. Hypothyroidism.

Clinical manifestations.

Atherosclerosis initially develops asymptomatically, often over decades. Signs appear when obstructions to blood flow occur. Transient ischemic symptoms (e.g., stable angina pectoris, transient ischemic attacks, intermittent claudication) may develop when stable plaques grow and reduce the arterial lumen > 70%. Vasoconstriction can exacerbate vascular wall lesions (which previously did not restrict blood flow) and lead to severe or complete stenosis. Symptoms of unstable angina, myocardial infarction, ischemic stroke, or resting leg pain may appear when unstable plaques rupture and suddenly close a major artery, with thrombosis or embolism joining in. Atherosclerosis can also cause sudden death without prior stable or unstable angina.

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

Diagnosis

In the initial stage of atherosclerosis, the course is asymptomatic. Depending on arterial lesions by localization - coronary, cerebral, lower extremity arteries, renal arteries, mesenteric arteries - complaints and disease history are formed (CHD, cerebrovascular disease, obliterating endarteritis, vasorenal hypertension, chronic mesenteric ischemia syndrome).

Physical examination - In the initial stage of atherosclerosis, physical examination does not reveal pathology.

OAE, biochemical blood test (LDL, HDL, triglycerides, glucose), USDG of blood vessels, vascular MRI.

Treatment

- Lifestyle changes (diet, smoking, physical activity).
- Drug treatment of the established risk factors
- Antiplatelet drugs
- Statins, possibly ACE inhibitors, beta-blockers

Treatment involves active elimination of risk factors to prevent new plaque formation and reduce existing plaques. Reducing LDL levels below a certain target level is no longer recommended and the current preference is for a "the lower the better" approach.

Lifestyle changes include diet, smoking cessation, and regular physical activity. Medications for dyslipidemia, AH, and diabetes mellitus are often necessary. These lifestyle changes and medications 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 levels. Antiplatelet drugs are effective in all patients with atherosclerosis. Patients with coronary heart disease may benefit further when prescribed ACE inhibitors and beta-blockers.

4.Illustrative material: slides in Microsoft Office Power Point.

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 atherosclerosis?
2. What are the causes of atherosclerosis?
3. What is the difference between HDL and LDL?
4. Name the complications of atherosclerosis.
5. What diagnostic methods are used to determine atherosclerosis?

Lecture №11

1. Topic: Coronary Heart Disease

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

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

3. lecture abstracts:

Coronary heart disease is an acute or chronic myocardial lesion resulting from a reduction or cessation of arterial blood supply to the heart muscle, based on pathological processes in the coronary artery system.

IHD is a widespread disease. One of the main causes of mortality, temporary and permanent disability throughout the world. Cardiovascular diseases are in the first place in the mortality structure, of which IHD accounts for about 40%.

Classification of CHD

Angina

- Stable angina pectoris
- Unstable angina

Primary myocardial infarction

Recurrent myocardial infarction

Old (suffered earlier) myocardial infarction (postinfarction cardiosclerosis)

Sudden cardiac (arrhythmic) death

Heart failure (myocardial damage due to CHD)

The main reason for myocardial oxygen supply failure is the discrepancy between the coronary blood flow and the metabolic needs of the heart muscle. This may be a consequence of:

-Atherosclerosis of coronary arteries with narrowing of their lumen by more than 70%.

-Spasm of unchanged (little changed) coronary arteries.

-Microcirculation disorders in myocardium.

-Increased activity of blood coagulation system (or decreased activity of anti-coagulation system).

The main etiological factor in coronary heart disease is coronary atherosclerosis. Atherosclerosis develops sequentially, in a wave-like and steady manner. As a result of cholesterol accumulation in

the artery wall, atherosclerotic plaque is formed. An excess of cholesterol leads to an increase in plaque size, blood flow is obstructed. Subsequently, under the influence of systemic adverse factors, the plaque transforms from stable to unstable (cracks and ruptures occur). The mechanism of platelet activation and thrombus formation on the unstable plaque surface starts. Symptoms worsen with the growth of atherosclerotic plaque, gradually narrowing the arterial lumen. Decrease of arterial lumen area by more than 90-95% is critical, causes decrease of coronary blood flow and deterioration of well-being even at rest.

Coronary heart disease risk factors:

Gender (male).

Age >40-50 years

Heredity

Smoking (10 or more cigarettes per day for the past 5 years)

Hyperlipidemia (total plasma cholesterol > 240 mg/dL; LDL cholesterol > 160 mg/dL)

Arterial hypertension

Diabetes mellitus

Obesity

Hypodynamia

Clinical picture of coronary heart disease:

The first description of angina pectoris was offered by the English physician William Geberden in 1772: "...pain in the chest, arising while walking and forcing the patient to stop, especially while walking soon after eating. This pain, if continued or intensified, seems capable of taking one's life; at the moment of stopping, all unpleasant sensations disappear. After the pain has continued for several months, it ceases immediately to pass on stopping; and thereafter it will continue to occur not only when one walks, but also when one lies down..." Usually the symptoms of the disease first appear after the age of 50. At the beginning, they occur only with physical exertion.Usually the symptoms of the disease first appear after the age of 50 years. In the beginning they occur only with physical exertion.

THE CLASSIC MANIFESTATIONS OF CORONARY HEART DISEASE ARE:

-Pain behind the sternum, often irradiating to the lower jaw, neck, left shoulder, forearm, hand, and back.

-Pain is crushing, squeezing, burning, choking. Intensity varies.

-Incited by physical or emotional factors. At rest stops on its own.

- Lasts from 30 seconds to 5-15 minutes.

- Rapid effect of nitroglycerin.

Treatment of coronary heart disease:

Treatment is aimed at restoring normal blood flow to the myocardium and improving patients' quality of life. Unfortunately, purely therapeutic methods of treatment are not always effective. There are many surgical methods of correction, such as: aorto-coronary bypass surgery, transmyocardial laser revascularization of the myocardium and percutaneous coronary interventions (balloon angioplasty, coronary artery stenting).

The "gold standard" in the diagnosis of obstructive lesions of the coronary arteries of the heart is considered to be selective coronarography. It is used to find out whether the vessel is significantly narrowed, which arteries are affected, and how many of them, in what place and along what length. Recently, multispiral computed tomography (MSCT) with intravenous bolus contrast media has become increasingly widespread. In contrast to selective coronarography, which is essentially a radiosurgical intervention on the arterial bed and is performed only in a hospital, coronary MSCT is usually performed on an outpatient basis using intravenous injection of contrast agent. Another

fundamental difference may be that selective coronarography shows the vessel lumen, whereas MSCT shows both the vessel lumen and the vessel wall in which the pathological process is localized.

4.Illustrative material: slides in Microsoft Office Power Point.

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. Reference questions (feedback):

1.Give the definition of IBS.
2. State the causes of the development of CHD.
3. Tell the classification of CHD.

Lecture №12

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. Literaturei:

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