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METHODOLOGICAL MATERIALS ON PULMONOLOGY IN THE COURSE OF FACULTY THERAPY

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Methodological materials are intended for teaching 4th year students (7.8 semesters) of the medical faculty of the Federal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation in the discipline "Faculty therapy".

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ACUTE PNEUMONIA.

Acute pneumonia is an acute exudative-inflammatory process of various etiology and pathogenesis with a predominant lesion of the respiratory parts of the lungs and the involvement of all other anatomical structures in the pathological process.

The incidence is very high - every year 1 in 100 people get pneumonia, and in during the flu epidemic, this percentage increases.

In our country, the generally accepted classification of pneumonia, developed by N.S. Molchanov in 1962 and supplemented by O.V. Korovina, which is being built onvarious criteria for assessing the pathological process.

Classification of acute pneumonia (V.P. Silvestrov, 1987).

I. By etiology (indicating the pathogen): 1. Bacterial.
2. Microplasma.
3. Viral.
5.Mixe

II. By clinical and morphological characteristics:
1.Parenchymal (croupknowing the share).
2.Focal (lobular, bronmonia).
3.Interstitial.

III. Downstream: 1. Sharp. 2. Protracted.

IV. By localization: 1. Right lung.
2. Left lung.
3. Bilateral defeat.
4. Share, segment.

V. By the presence of functional disorders of external respiration and blood circulation: 1. Without functional disorders.
2.With functional impairments (their characteristicsstick, severity).

Vi. By the presence of complications: 1. Uncomplicated.
2. Complicated (pleurisy, exudative, abscess, bacterial toxic shock, myocarditis, endocarditis, etc.).

It is also advisable to reflect the severity of acute pneumonia.

Pathogenesis.

Speaking about the pathogenesis, it should be noted that the question of the mechanism of infection of the lung tissue is important. With the constant inhalation of microorganisms, the contents of the bronchi in a healthy person remain practically sterile. It accomplishesXia due to the presence of a number of protective mechanisms:

a) local - adequate work of the epithelium of the nasopharynx; mucociliary clearance, i.e. cleansing of the bronchial tract by the friendly work of the ciliated epithelium with a preserved cough reflex and a normal composition of mucus (so in a healthy person, the composition of the bronchial secretion is updated every 3

hours, and all pathogenic components are eliminated); phagocytic function of cellular elements - neutrophils; components of local immunity - immunoglobulin A, lysozyme, interferon; surfactant; alveolar macrophages.

b) general mechanisms of cellular and humoral immunity.

Thus, for the infection of lung tissue, bronchogenic penetration of microflora is most often required in violation of local and general immunity. Changes in immunity may precede acute pneumonia or occur during an acute viral infection preceding the development of acute pneumonia.

In addition to the bronchogenic route of infection, there is a hematogenous and lymphogenous route of infection.

Clinic of acute pneumonia.

Croupous pneumonia.

The clinical picture (patient complaints, physical data) corresponds to pathomorphological changes and proceeds in several stages:

1. The stage of bacterial edema or the tidal stage lasts from 12 hours to 3 days and is characterized by sharp hyperemia and exudation of the lung tissue with the formation of a zone of edema.

2- Compaction stage, which is divided into 2 periods: a) the period of red hepatization of the lung tissue (from the 3rd day the alveoli are filled with sweating plasma rich in fibrin and erythrocytes, the so-called erythrocyte diapedesis); b) the period of gray hepatization (3 days after the period of red hepatization), while erythrocytes are destroyed, hemoglobin turns into hemosiderin, leukocytes accumulate in the alveoli, fibrin massively falls out in the form of a film.

3- Resolution stage (dissolution of fibrin under the action of leukocyte proteases and gradual resorption of exudate).

The disease begins acutely, patients can often indicate not only the day, but also the hour of the onset of the disease. The onset of the disease is characterized by tremendous chills and an increase in body temperature up to 40 ° C. After a while, pain in the chest joins when breathing. In the first hours of the disease, the patient is worried about a dry cough, which increases chest pain. After 2 days, when coughing, "rusty" sputum begins to stand out.

On a general examination, flushing of the cheeks, shortness of breath, pallor and cyanosis of the skin, herpetic eruptions in the area of the lips and wings of the nose, tachycardia corresponding to the level of body temperature are noted. When examining the chest in the first days, one can notice the lag of one half of it during breathing. Vocal tremor over the affected half of the chest is enhanced, and percussion and auscultatory data correspond to the stage of the process. At the onset of the disease (edema stage), the percussion sound gives a tympanic tone, because at this time, both air and liquid are in the alveoli at the same time. Auscultatory - increased vesicular respiration and initial crepitus, which occurs when the alveoli collapse at the inspiratory height in the presence of a small amount of exudate in them. At the height of the stage - the dullness of sound is determined percussion. On auscultation - bronchial breathing,

In the stage of resolution, air again enters the alveoli, therefore tympanic tone of percussion sound and crepitus. The temperature reaction lasts about 5 days.

Focal pneumonia.

It is much more common than lobar pneumonia.

The clinical picture of focal pneumonia is characterized by a gradual onset of the disease, tk. focal pneumonia usually develops against the background of ARVI or exacerbationchronic bronchitis. Focal pneumonia is characterized by: low-grade fever, cough with scanty mucopurulent sputum, moderate shortness of breath, general weakness, sweating. On examination, there is an increase in vocal tremor over the lesion, a limited zone of dullness of percussion sound, an increase in vesicular breathing, fine bubbling moist rales. According to the localization of pneumonia, damage to the lower lobe of the right lung is more often noted (due to the anatomical features of the right bronchus).

Additional research methods.

Clinical blood test.

When examining the peripheral blood of a patient with acute pneumonia, the same type of changes are determined, but their severity in lobar and focal pneumonia is different. Focal pneumonia is characterized by moderate neutrophilic leukocytosis, a stab shift to the left, and a moderate increase in ESR.

With lobar pneumonia, there is a more significant leukocytosis, palcano-nuclear shift, toxic granularity of neutrophils, increased ESR, eosinopenia, lymphopenia.

Clinical analysis of sputum. The patient's sputum contains a large number of leukocytes, macrophages. With lobar pneumonia, sputum includes red blood cells.

Sputum bacteriological analysis.

To clarify the etiology and determine the tactics of treatment, sputum culture with identifying the pathogen and determining its sensitivity to antibiotics.

Blood chemistry. Nonspecific changes are noted (increased levels of fibrinogen, sialic acids, C-reactive protein).

X-ray research methods.

Fluoroscopy (graphy) of the chest organs can confirm the diagnosis, to clarify the localization and length of pathological changes.

With focal pneumonia, small foci of inflammatory infiltration of the lung tissue appear within the segment.

With a lobe, there is a total homogeneous darkening within the lobe.

The course of acute pneumonia.

Clinicians distinguish an acute course of pneumonia (with resolution within 3-4 weeks) and a protracted course, which ends in 1.5-2 months. treatment of the patient. A protracted course is often associated with the background pathology of the patient, the development of complications (lung abscess, pleurisy, acute

respiratory failure, myocarditis).

Principles of treatment of acute pneumonia.

1. Compulsory hospitalization of patients in the case of large-focal, pleuropneumonia, as well as children, elderly and debilitated patients with concomitant pathology.

2. Strict bed rest during the entire febrile period.

3. The patient's diet is predominantly liquid, easily digestible food at the onset of the disease. Then - an increase in the energy value of food.

4. Drug therapy.

- Etiotropic treatment with antibiotics, sulfa drugs. Antibiotic therapy should be early; taking into account the pathogen, and carried out in an adequate dose. Violation of these requirements leads to the fact that pneumonia acquires a protracted course.

The duration of therapy is determined according to the scheme: "febrile period of the disease plus 5-7 days."

-Pathogenetic therapy is carried out with mucolytics and expectorants. These drugs are administered in the form of tablets, drops, inhalation.

-Symptomatic therapy is carried out by infusion of fluids, the use of cardiotonic drugs.

5. Physiotherapy treatment is carried out after normalization of temperature, in the absence of hemoptysis.

The main treatment for acute pneumonia is the prescription of antibacterial agents. Treatment for pneumonia begins immediately after diagnosis. Bacteriological control is required to determine the pathogen and its sensitivity to antibiotics.

According to the consensus of the Russian National Congress of Pulmonologists, the duration of antibiotic therapy is determined by the type of pneumonia pathogen. Uncomplicated bacterial pneumonia is treated for another 3-4 days after the normalization of body temperature (subject to the normalization of the leukocyte formula).

In the absence of the effect of the antibiotic within 2-3 days, it is changed, ifIn the course of pneumonia, antibiotics are combined.

A combination of a group of antibiotics (penicillins, cephalosporinew and glycopeptides) with sulfa drugs. The mechanism of the bactericidal action of these antibiotics is associated with the effect only on the multiplying bacteria. Sulfanilamide drugs inhibit this process.

For community-acquired pneumonia, which is most often caused by pneumococci, haemophilus influenzae, and less often gram-negative flora or mycoplasma, treatment start with antibiotics of the 1st line (a group of penicillins - benzylpenicillins, oxacillin, ampicillin, amoxicillin, agumentin (amoxicillin and clavulanic acid suppress I-lactamase and protect penicillin), piopen, azlocillin, temocillin).

An alternative drug of the macrolide group: old ones - erythromycin, oletetrin; new - sumamed, macropen.

Hospital-acquired pneumonia are often caused by resistant strains of

staphylococci and oxacillin is recommended for treatment, a combination of antibiotics withgibitors I-lactomases, ciproflaxin.

For aspiration pneumonia associated with gram-negative flora or anaerobes, aminoglycosides are recommended (1 poc - kanamycin, 2 poc - gentamicin, 3 poc - amikan) or a combination of 3rd generation cephalosporins (claforan) with metronidazole.

If it is assumed that the pathogen is Pseudomonas aeruginosa, then amnoglycosides, 6th generation penicillins and 4th generation cephalosporins.

Atypical pneumonia caused by mycoplasma, legionella, chlamydia. Macrolides are used for treatment.

In patients with immunodeficiency states, pneumonia is often caused by opportunistic flora and protozoa. Biseptolum is used for their treatment,bactrim.

Further correction of antibiotic therapy is carried out depending on the clinical effect and the results of laboratory tests.

Important factors indicating the threat of a protracted course of pneumonia are late hospitalizations and a high percentage of globulins on the day of hospitalization. A positive relationship between the duration of illness and the number of antibiotics used seems natural. But their excessive use can contribute to the sensitization of patients, the provocation of drug fever and vasculitis in them, which leads to the preservation of infiltrative changes in the lungs and their transformation into fibrosis, especially against the background of immune pathology.

It must be remembered that the darkening in the lungs, which is documented radiographically, with late hospitalization in the absence of fever, shortness of breath, leukocytosis, may be caused by infiltration of the lung tissue no longer by neutrophilic leukocytes, which are mandatory at the onset of pneumonia, but by immunocytes (lymphocytes and monocytes) replacing them, or fibroblasts. Indirect evidence of this may be hyper-y-globulinemia and a high incidence of fibrosis in patients with polysegmental pneumonia.

As it has now become obvious, antibacterial therapy alone cannot ensure the success of treatment; it must be combined with measures aimed at stimulating the body's protective and adaptive reactions.

The arsenal of means used to treat pneumonia is so extensive that a rational choice for a practitioner in many cases is difficult. Domestic researchers offer a practically grounded approach to the treatment of pneumonia, when therapy is changed taking into account changes in clinical manifestations.

At the onset of the disease in the phase of bacterial aggression, antibiotics and detoxification therapy are the basis. Along with this, sanitary and hygienic measures are carried out (regime, food for the patient). Symptomatic therapy is carried out, which provides for the elimination of painful symptoms of the disease. As for antipyretics and analgesics, in view of the possible negative effect on immune reactivity, they are used only with severe hyperthermia and pleural pain.

With a strong dry cough, antitussive non-narcotic drugs are prescribed that reduce cough, but have a secretolytic effect and do not reduce the drainage

function of the bronchi (baltix, stop-tussin).

During this period, various changes in clinical and laboratory parameters occur: hemodynamic disturbances, microcirculation, patency of small bronchi, leukocytosis, hyperfibrinogenemia, increased lipid peroxidation, etc.

Correction of lipid peroxidation processes is carried out regardless of the etiology of pneumonia. In the acute period, at the height of intoxication, water-soluble antioxidants (ascorbic acid, unitiol) are prescribed.

To prevent hemodynamic disorders, analeptics, cardiac glycosides, more often korglucon are used.

Acute pneumonia is characterized by various disorders of immunological reactivity, the features of which depend on the etiology, pathogenetic manifestations of pneumonia, the prevalence of the process, and the severity of the course of the disease. Transient disorders of the immune response, observed in acute pneumonia, usually do not require the appointment of immunomodulators. Immunocorrective therapy is carried out in cases of pronounced imbalance in the regulatory link or depression of the macrophage and effector killer link of immunity.

In acute situations with extremely severe pneumonia as a remedy for replacement therapy of secondary antibody deficiency syndrome, sterile lyophilized endobulin is used until acute symptoms disappear, at a dose of 100 mg per 1 kg of body weight. Can be repeated at weekly intervals.

For the prevention of DIC syndrome, heparin is administered subcutaneously during the febrile period. With psychomotor agitation and delirium, chlorpromazine or haloperidol is prescribed.

Second phase the course of acute pneumonia - the phase of clinical stabilization - is characterized by the formation of an infiltrate while maintaining intoxication. During this period, antibacterial treatment continues, active anti-inflammatory therapy is carried out using both medicinal and physiotherapeutic procedures.

Elimination of fever indicates the transition of the disease to the phase of morphological recovery, during which the exudate is absorbed. Antibiotics are canceled, anti-inflammatory and absorbable therapy is continued. Non-steroidal anti-inflammatory drugs are used, and if indicated, and***corticosteroid medications.***

One of the reasons for the protracted course of pneumonia is the syndrome of bronchial obstruction, which is often combined with the preservation of unabsorbed remnants of the infiltrate or its fibrous transformation. To normalize bronchial patency, bronchodilators, sputum-thinning and expectorants are indicated. In the first days of the disease, anticholinergics (atropine, platifillin) or combined preparations containing them (solutan) are prescribed. In the future, adreiergic bronchodilators (berotek) and expectorants are combined. They use drugs that stimulate the synthesis of a surfactant (bromhexine). Prescribe multivitamin pre***parats.***

Resorption of inflammatory infiltrates means the transition of the disease to the phase of functional recovery and thereby during the period of convalescence.

During the period of pneumonia resolution, methyluracil, pentoxil, biogenic stimulants (aloe, vitreous) are used to stimulate the regeneration processes, adaptogenicus (tincture of ginseng root).

The formation of local pneumofibrosis is considered as a form of recovery, albeit incomplete. By this time, patients are discharged from the hospital and re- they are taken to the outpatient-polyclinic stage of treatment.

The outcomes of pneumonia depend on the timeliness of diagnosis, treatment tactics and an individual approach to each patient.

Recovery criteria.

Clinical - disappearance by the end of the 7-10th day of all physical symptoms diseases.

Laboratory - the disappearance of the inflammatory reaction of the blood until the end of the 2nd weekwhether the disease.

X-ray - disappearance of radiological signs of acute pneumonia by 21 days.

CHRONICAL BRONCHITIS

Etiology and pathogenesis.

Emergence and the development of chronic bronchitis has been linked to exposure to three environmental factors: smoking, air pollution, and infection. With prolonged exposure to irritating factors, primary chronic bronchitis is formed,with infection, acute bronchitis often becomes chronic.

The frequency of chronic bronchitis in smokers is 2-5 times higher than in non-smokers. Mortality from chronic bronchopulmonary diseases among smokers of more than 25 cigarettes per day is 30 times higher than among nonsmokers. Tobacco smoke disrupts the mucociliary clearance of the bronchial mucosa long before the development of clinical signs of bronchitis.hit. Tobacco smoke paralyzes the cilia of the bronchial mucosa.

There is mucostasis and increased reactivity of bronchial receptors, causing a violation of bronchial patency. Smokingand air pollution manifestedthey are synergistic effect on the respiratory system.

Significant role in shaping and the progression of chronic bronchitis belongs to infection (BE Votchal). Infection (usually viral) acts as an independent cause of acute bronchitis with the transition to chronic (up to 10% of cases).

In the pathogenesis of chronic bronchitis, the pathology of the nasopharynx, focal infection in the upper respiratory tract, which is a source of bacterial contamination and infectious sensitization, is important. Violation of nasal breathing impairs the functions of cleansing, warming, humidifying the inhaled air, and also contributes tomaintaining the inflammatory process. Among the infectious agents of exacerbation of chronic bronchitis, the leading role belongs to pneumococcus and Haemophilus influenzae. Pneumococcus was isolated from sputum in a diagnostic titer in about 80% of patients with chronic bronchitis in the acute phase (L.A. Vishnyakova). The frequency of excretion of Haemophilus

influenzae at the beginning of an exacerbation (with the 1st degree of activity of the inflammatory process) was established in approximately 20% of patients with chronic bronchitis. With a high degree of activity of the inflammatory process, the frequency of release of the hemophilic bacillus increases and determines the course of inflammation (M.E. Faustova, 1987).

Violation of bronchial drainage creates conditions for the vegetation of microorganisms, which determines the high frequency and concentration of these bacteria in obstruction, bronchiectasis. Associations of pneumococcus and Haemophilus influenzae significantly increase with purulent bronchitis and persistent obstructive phenomena in the lungs.

The manifestation of obstructive syndrome and the course of chronic bronchitis are influenced by climatic and weather factors, especially in meteorological patients (V.A.Kantur, 1987). It has been established that the risk factors for meteorological stability in chronic bronchitis are:

1. Age (50 and older).
2. Gender (more often in men).
3. The frequency of exacerbations (in persons with 2-3 exacerbations per year or more).

There are four main stages in the development of chronic bronchitis:

1. A threat situation with the presence of risk factors (smoking, air pollution, pathology of the nasopharynx and impaired breathing through the nose, repeated acute respiratory viral infections, acute bronchitis, bronchopneumonia).
2. Formation of pre-disease - pre-bronchitis.
3. Detailed clinical picture of the disease.
4. Complicated disease.

The main manifestations of pre-bronchitis can be:

- smoker's cough;
-cough due to irritation of the respiratory tract (gas pollution, dustiness of the inhaled air);
-cough due to pathology of the nasopharynx, which makes it difficult to breathe through the nose;
- prolonged and recurrent course of acute bronchitis;
- a feeling of respiratory discomfort upon contact with unfavorable microclimatic conditions (ecology of the environment, temperature drops, excessive humidity).

To distinguish between the stages of pre-disease (pre-bronchitis) and disease (chronic bronchitis), you can focus on the criteria developed by WHO experts.

In chronic bronchitis, cough (sputum separation) lasts at least 3 months. for two years in a row, while at the stage of pre-illness, cough (with or without sputum separation) still meets the specified criteria. It can be shorter or repeated at longer intervals.

At the stage of a detailed clinical picture, chronic bronchitis is characterized by:

1. A type of inflammation of the bronchial tree (catarrhal, purulent, hemorrhagic).

2. The absence or presence of bronchial obstruction (degree and level of obstruction).

3. The presence of complications (pulmonary heart failure, bronchiectasis, diffuse or focal pneumosclerosis, emphysema).

4.

Chronic bronchitis. Classification.

I. Clinical forms: 1. Simple (catarrhal) uncomplicated, non-obstructive (with secretion of mucous sputum, without ventilation disorders).

2. Purulent non-obstructive (with the release of purulent sputum without ventilation disorders).

3. Simple (catarrhal) obstructive bronchitis (with mucous sputum and persistent ventilation disorders).

4. Purulent obstructive bronchitis.

5. Special forms: hemorrhagic, fibrinous.

II. **Level of defeat:** 1. Bronchitis with predominant involvement of large bronchi (proximal).

2. Bronchitis predominantly affecting small bronchi (distal).

III. The presence of bronchospastic (asthmatic) syndrome.

IV. **Flow:** 1. Latent.

2. With rare exacerbations.

3. With frequent exacerbations.

4. Continuously relapsing.

V. **Process phase:** 1. Aggravation.

2. Remission.

VI. **Complications:** 1. Emphysema of the lungs.

2. Hemoptysis.

2. Respiratory failure (indicating the degree).

3. Chronic cor pulmonale (compensated, decompensated).

Here are examples of the formulation of the diagnosis:

1. Chronic catarrhal diffuse non-obstructive (functionally stable) bronchitis, exacerbation phase, DN0.

2. Chronic catarrhal diffuse obstructive bronchitis, exacerbation phase, pulmonary emphysema, DN II, chronic compensated cor pulmonale.

3. Chronic suppurative diffuse obstructive bronchitis, exacerbation phase. Diffuse pneumosclerosis, pulmonary emphysema, DN III, chronic decompensated pulmonary heart disease, NKIIa.

Clinic. Diagnostics.

The clinical picture of chronic bronchitis is determined by the phase of the disease (exacerbation, remission), the nature of the inflammatory process, the depth and level of damage to the bronchial tree, the presence of bronchial

obstruction.

The leading complaint of patients with chronic bronchitis is cough (dry or with sputum), shortness of breath, often expiratory in nature.

In patients with chronic bronchitis with a predominant lesion of the small bronchi, exacerbation often proceeds with shortness of breath, the cough syndrome is mild, often without sputum secretion. This is due to the lack of receptors for the cough reflex in the small bronchi (B.E. Votchal). Clinically, there are signs of bronchial obstruction in the form of high-pitched dry wheezing, mainly with forced expiration, against the background of vesicular, less often weakened breathing.

In patients with chronic bronchitis with damage to large and medium bronchi, the leading complaint is a cough with sputum production. On objective examination, dry rales of low timbre (humming) are heard over the lungs, breathing is hard or weakened. Cracking moist rales can be heard, more often in the lower lateral parts of the lungs. The appearance of wet rales is due to the accumulation of sputum in the bronchi.

Depending on the presence and nature of sputum, chronic bronchitis is divided into: dry, catarrhal and purulent.

The addition of an obstructive component in any form of chronic bronchitis is prognostically unfavorable for the patient. With obstruction, the drainage function of the bronchi worsens, hypertension of the pulmonary circulation develops, and cor pulmonale is formed.

Objective disorders in chronic bronchitis tend to progress, therefore, it is necessary to identify them as early as possible, to carry out timely correction of these disorders as a measure of prevention of exacerbations of the inflammatory process.

The clinical signs of obstructive syndrome in patients with chronic bronchitis include the following:

1. The appearance of shortness of breath during physical exertion, a change in the temperature of the inhaled air (when leaving a warm room to the cold). Shortness of breath in patients with obstructive bronchitis is characterized by difficulty in exhaling and variability: "Day after day does not occur."
2. A painful paroxysmal cough with difficult sputum, increased shortness of breath after coughing fits (I.P. Zamotaeva).
3. The presence of high-pitched whistling dry wheezing arising during exhalation.
4. Elongation of the expiratory phase (mainly forced), the appearance of a boxed percussion sound, swelling of the cervical veins during exhalation as a result of intrathoracic pressure.
5. According to spirography data: changes in the spirogram depend on the severity of impaired respiratory function, usually a decrease in VC, an increase in the MOU, a decrease in the O2 coefficient is possible. Spirographic manifestations of bronchial obstruction - a decrease in the forced vital capacity of the lungs (FVC) and maximum ventilation of the lungs.
6. According to pneumotachometry data, a decrease in the maximum

expiratory flow rate.

The selection of obstruction options is possible taking into account a comprehensive analysis of clinical data, laboratory parameters, and the results of X-ray bronchological functional studies. For the diagnosis of the predominance of the spastic component, in addition to clinical data, pharmacological tests with bronchodilators are of primary importance, revealing the lability of the functional parameters of external respiration, complete or partial reversibility of obstruction.

To objectify discrimination, an important role is played by a macroscopic examination of sputum, an assessment of its viscosity and elasticity. Hyperplastic changes are evidenced by the data of bronchoscopy, the study of biopsy specimens of the mucous membrane.

The presence of tracheobronchial dyskinesia (expiratory collapse of the trachea and large bronchi) can be suspected by the nature of the cough: bitonal, low timbre, sometimes accompanied by fainting at the height of the attack. The diagnosis is confirmed by the data of bronchoscopy, which reveals the mobility of the posterior membrane wall of the trachea and large bronchi, its sagging into the lumen during exhalation and coughing.

The activity of the inflammatory process in chronic bronchitis is determined by a combination of clinical and laboratory data. Clinical signs of exacerbation of chronic bronchitis include subfebrile temperature, daily temperature fluctuations up to 1 ° C within the normal range, chilling, night sweating of the upper half of the body (shoulder girdle, neck, nape - a symptom of a wet pillow), increased shortness of breath, bronchial obstruction, an increase in signs of insufficiency circulation in chronic pulmonary heart disease.

Changes in the composition of peripheral blood during exacerbation of chronic bronchitis are manifested by a moderate stab shift, an increase in ESR. In patients with chronic bronchitis, complicated by decompensated pulmonary heart disease, there is a tendency to erythrocytosis, ESR is slowed down.

Biochemical indicators of acute phase reactions are more informative in assessing the activity of the inflammatory process in chronic bronchitis:

C-reactive protein, sialic acids, fibrinogen, serum protein spectrum (a1-, a2- g-globulins), lactate dehydrogenase (LDH) with isoenzymes. An important role is played by cytological examination of sputum. With exacerbation of chronic bronchitis, cellular elements are represented mainly by leukocytes, dystrophic altered cells of the bronchial epithelium, eosinophilia and the presence of macrophages. Bacteriological examination of sputum is of great importance in assessing the inflammatory process and making an etiological diagnosis.

Sputum should be delivered to the laboratory no later than 1-2 hours from the moment of collection, because longer exposure of the material at room temperature leads to the death of pathogenic pathogens. Sputum is collected in the amount of 2-3 spits into a Petri dish or sterile dish. Before bacterioscopic and bacteriological examination, the purulent lump is washed three times with sterile saline (Mulder's method). This method of sampling and sowing sputum is easy to perform, informative and most accurately reflects the microbial landscape of the lower respiratory tract (V.I.Sokolova). Bacterioscopy of a smear stained

according to Gram and Ziehl-Nielsen is an available method for an approximate assessment of microflora. Bacteriological research (culture) allows you to assess the qualitative composition and quantitative ratio of sputum microflora. It is known

Excretion of pathogens in concentrations of 105 and higher microbial bodies in 1 ml of sputum is considered to be etiologically significant. After identification of the pathogen, the sensitivity of the isolated microorganisms to antibiotics is determined. In some cases, a serological study of paired sera is carried out in relation to viral, legionella and mycoplasma infections.

The possibilities of X-ray examination in the diagnosis of chronic bronchitis are very limited. In the early stages of the disease, changes in the lungs may not be detected, but X-ray examination allows you to exclude another pathology of the lungs. In the later stages of chronic bronchitis, on the general images of radiographs of the lungs, deformation and strengthening of the pulmonary pattern are revealed due to perilobular, peribronchial, perivascular pneumosclerosis. With the development of obstructive emphysema, an increase in the transparency of the pulmonary fields is found.

The method of choice in the diagnosis of chronic bronchitis is bronchography. Bronchographic signs of bronchitis include transverse striation of the bronchial mucosa, bronchiectasis, moderate cylindrical expansion of the bronchi, uneven contours, multiple breaks of the branches of the I-Ip order, uneven lumen of the bronchi.

In terms of differential diagnosis of lung diseases, tomography and bronchoscopy are essential, which make it possible to clarify the localization, degree of inflammation and the prevalence of the process.

The main complications of chronic bronchitis include: peribronchotic pneumosclerosis, obstructive pulmonary emphysema, respiratory failure, cor pulmonale. You should take into account the possibility of pneumonia, which take a protracted course and often turn into chronic forms of the disease (lung abscess, chronic pneumonia).

Respiratory failure degree evaluated by clinical manifestations:

1 degree - shortness of breath with previously available physical activity (DNI).

2 degree - shortness of breath during habitual exertion (DN II).

3 degree - dyspnea at rest (DN III).

With the development of decompensated pulmonary heart disease, the stage of circulatory failure is classified according to Strazhesko-Vasilenko (HI, H Pa, H Pb, H III).

Treatment...

When drawing up a treatment plan, it is necessary to take into account the etiological factor of exacerbation, the nature of the inflammatory process, the presence or absence of broncho-obstructive syndrome, drug tolerance and the state of the patient's immune status.

1. Antibacterial therapy.

2. Immunocorrectors.
3. Expectorants, mucolytics.
4. Bronchodilators.
5. Physiotherapy treatment.

1. Indications for the appointment of antibacterial drugs are: purulent sputum, symptoms of intoxication.

Despite the large selection of antibiotics and sulfa drugs, the treatment of chronic bronchitis presents difficulties due to the variable sensitivity of certain pathogens and microbial associations to them.

In case of purulent bronchitis and peribronchitis with symptoms of pronounced intoxication syndrome, bronchorhea (200-500 ml / day), parenteral therapy with metrogil 100 ml 2 times a day intravenously is recommended. Sometimes combination therapy is used, the effect of which is aimed at a specific isolated or putative pathogen (aerobic-anaerobic association). From this point of view, metrogil (100 ml 2 times a day intravenously) is prescribed in combination with aminoglycosides (gentamicin 160-200 mg / day, netromycin - 200 mg / day, sisomycin, amikacin, etc.). The main goal of combination therapy is to fill the gaps in the spectrum of action of each of the components.

Fluoroquinolones (ofloxacin, ciprofloxacin, norfloxacin, etc.) are opening up a new perspective in the treatment of chronic bronchitis. They are active against both gram-positive and gram-negative pathogens, including "problem" types of microorganisms (materials of the symposium, 1986). The experience of treatment with ofloxacin (tarivid) has shown that the drug is highly effective in the treatment of chronic bronchitis of various etiologies. He is prescribed 200 mg 2-3 times a day for 7-10 days. Tarivid actively penetrates into sputum and its concentration is 1-5 mg / l, exceeding the MIC value by 10 times. Fluoroquinolones can be administered orally and parenterally. It is especially recommended to use them in the treatment of persistent infections (of unclear etiology) and severe intolerance to penicillins and cephalosporins (V.I.Sokolova).

2.In recent years, in the treatment of patients with respiratory diseases, more and more attention is paid to immunocorrective drugs (levamisole, diucifon, kemantan, mildronate, etc.). Indications for their use are the presence of clinical signs of immunodeficiency:

- a sluggish inflammatory process;
- frequent relapses of respiratory diseases;
- ineffective treatment of chronic bronchitis with antibiotics and chemotherapy drugs due to the fact that some antibiotics are themselves immunosuppressants.

Levamisole (decaris)used in patients with chronic bronchitis with severe T-cell deficiency and impaired synthesis of immunoglobulins. The drug is prescribed in a daily dose of 2.5 mg per 1 kg of body weight; or an average of 150 mg / day in a row 3 days a week for 3-4 weeks. Levamisole can cause nausea, skin rashes and the worst complications, leukopenia and thrombocytopenia, so blood should be monitored at least once a week. When these side effects appear, treatment with

levamisole is discontinued. It should be noted that an increase in the percentage of E-ROC and immunoglobulins A and G corresponds to good clinical dynamics. Nonspecific protective factors (lysozyme, complement, bactericidal activity of blood serum, etc.) respond adequately to a 1-2 month course of treatment with levamisole.

In recent years, a new immunomodulatory drug, katergen, has been successfully used. It is prescribed orally 500 mg 3 times a day with meals for 10-14 days. The drug has an antioxidant, membrane stabilizing effect, stimulates the immune antiviral and reticuloendothelial systems.

The use of immunocorrective drugs does not exclude the use of antibiotics, sulfa and antimicrobial drugs. The empirical appointment of immunocorrectors is unacceptable.

Treatment of chronic bronchitis should be complex due to the fact that the mechanisms and levels of bronchial obstruction are essential.

3.Among ***expectorants*** in the treatment of chronic bronchitis, drugs of reflex action are widely used - infusions of ipecacuanha, thermopsis.

From the means of direct action on the mucous membrane of the respiratory tract, which have a secretory and mucolytic effect, a 3% solution of potassium iodide, ammonium chloride, terpinhydrate is used.

The most effective mucolytic agents are cis-theine derivatives - acetylisteine, mucosolvin. Recently, bromhexine, bisolvon, lasolvan have been widely used. The drugs have a secretolytic, secretomotor and antitussive effect. They reduce the viscosity of sputum 50 times compared to the baseline.

Bisolvon prescribed by mouth, in the form of inhalation, intramuscular and intravenous injections.

Lasolvan - one of the metabolites of bromhexine, has a mucolytic effect, activates the function of the ciliated epithelium and ultimately provides mucociliary clearance of the bronchial tree. The drug is administered orally, intramuscularly and by inhalation. As studies carried out in our clinic have shown, lasolvan gives a distinct expectorant effect in 85% of patients with chronic bronchitis with impaired drainage function of the bronchi. The effect is more pronounced with the combined use of the drug inside and in aerosols. In severe cases of bronchial obstruction, it is advisable to prescribe lasolvan in the form of injections in combination with oral administration.

Proteolytic enzymes are also used - trypsin, chymotrypsin, chymopsin. ribonuclease in the form of inhalation, which has a mucolytic effect. The spectrum of action of these drugs is limited due to the possible increase in bronchospasm and hemoptysis in some patients.

4. Of great importance in the restoration of drainage and evacuation functions are warm-humid inhalation of physiological solution, 2% sodium bicarbonate solution, 10-20 ml 3-4 times a day.

An important role in the treatment of chronic obstructive bronchitis belongs to bronchodilators. Three groups of drugs are most widely used in clinical practice: adrenomimetics, anticholinergics, and phosphodiesterase inhibitors.

I-adrenomimeticsinclude: izadrin, novodrin, euspiran. isoprenaline. With the inhalation method of administration, the effect occurs after 2-3 minutes and lasts about 2 hours.

Izadrin has a moderate bronchodilatory effect. The optimal dose for inhalation use is 0.5 mg. Side effects are possible in the form of palpitations, rhythm disturbances associated with the excitation of R1 receptors. The frequency and severity of side effects increase with increasing dose.

Alupent (asthmopent) - isomer of isadrin, has a longer effect (up to 4 hours), has a pronounced bronchodilatory effect and to a lesser extent stimulates R1-adrenergic receptors. Alupent dosage forms: metered aerosols, 0.02 g tablets, solutions for stationary inhalers of 2% and 5%, ampoules of 1 ml of a 0.05% solution. Alupent is diluted 20 times (0.5 ml per 10 ml of saline) and inhaled for 5-7 minutes.

Ipradolthe magnitude of bronchodilation is approximately equal to that of alupent. The most effective dosage form of ipradol is metered-dose aerosols. The onset of action of the drug is after 2 minutes, the duration is about 5 hours.

R1 - the stimulating effect of ipradol is so pronounced that it can be used in patients with bradycardia and sick sinus syndrome. Of the side effects, in addition to palpitations, there is a pronounced tremor even with inhalation of therapeutic doses - 2 breaths.

Berotec (fenoterol)- the most effective adrenergic agonist with R1- and R2-stimulating action. Side effects include tachycardia, palpitations, tremors.

Ventolin (salbutamol) - Selective H2-stimulant, not metabolized by catecholo-methyltransferase, due to which it has a long-lasting bronchodilator effect, approaching berotek.

Terbutaline (bricanil) - selective Y2-stimulant. Its bronchodilator activity is close to that of ventolin. With an inhalation method of administration, the duration of action is up to 6 hours. There are practically no side effects.

As the bronchodilatory effect decreases, adrenomimetics in metered aerosols are distributed as follows: berotec, ventolin, alupent, ipradol, terbutaline, euspiran.

Anticholinergics act on the vagal component of bronchial obstruction. This group of drugs includes belladonna, atropine, atrovent, troventol.

The effectiveness of atropine is higher with the inhalation route of administration than with the parenteral one. Inhalation of 0.2-0.3 mg of atropine at a dilution of 1: 5, 1:10 for 3-6 minutes gives a bronchodilatory effect equal to intravenous administration of 0.24-0.48 g. aminophylline. The duration of the drug action is up to 5-6 hours.

Atrovent and troventolare produced in the form of metered aerosols. The optimal therapeutic dose is 1-2 breaths. Troventol and atrovent have approximately the samebronchodilatory activity equal to 22% of the initial parameters of the respiratory function. According to A.N. Tsoi 1990, troventol is most effective in the treatment of asthmatic bronchitis. The effect of the drug occurs in 3-4 days, the maximum effect develops on the 10th day. A slowdown in MCC and an increase in sputum viscosity is noted on the 3-4th day of treatment

with M-anticholinergics.

Berodualis a combined drug consisting of R2-adrenergic agonist (beroteka) and anticholinergic (atrovent). In one aerosol dose of the combined preparation, the content of berotek is 25%, the dose of atrovent is the same as in the metered aerosol of atrovent. Clinical observations have shown that Berodual is a highly effective bronchodilator. By the strength of the bronchospasmolytic action, the drug significantly surpasses atrovent and approaches berotek. However, berodual compares favorably with berotek both with a longer action and less pronounced side effects. The use of berodual significantly expands the therapeutic possibilities of influencing the multifaceted mechanisms of bronchial obstruction.

Euphyllin - a soluble preparation of theophylline, which is used as for the purchasetreatment of acute attacks of bronchospasm, and in the complex therapy of broncho-obstructive syndrome. The drug is administered orally, intravenously, rectally. The duration of the therapeutic effect when taken orally is 4-6 hours, when used in suppositories, up to 10 hours. Possible side effects: insomnia, palpitations, extrasystole, dyspeptic disorders. Dosage: aminophylline is administered at 10-20 mg / day of 2.4% solution intravenously; inside - 0.15 g every 6 hours.

At present, interest has increased in methylxanthines and especially in prolonged theophylline preparations (theopec, theobilong, durophyllin, theodur, ritafillin, etc.), which are capable of sustained release and maintenance of uniform therapeutic concentrations in the blood. Methylxanthines relax the smooth muscles of the bronchi, stimulate mucociliary clearance and improve the functioning of the respiratory muscles.

In case of difficult separation of viscous sputum before positional drainage, it is recommended to take expectorants for one hour, for example, a decoction of thermopsis S glass 3-4 times per hour.

Prevention.

The complex of measures for the prevention of chronic bronchitis provides for maintaining a healthy lifestyle, improving working conditions, rehabilitating foci of chronic inflammation in the nasopharynx, rational treatment of acute respiratory diseases, acute bronchitis.

BRONCHIAL ASTHMA

Bronchial asthma (BA) is a chronic recurrent disease with a predominant lesion of the bronchi, characterized by pathological hyperreactivity of the bronchi, a mandatory symptom of which is an attack of suffocation (G.B. Fedoseev, 1982).

Epidemiology.

Epidemiological studies conducted for many years in different parts of the

world have shown that about 3% of humanity suffers from bronchial asthma, and in 2% it is associated with a profession. According to L.G. Chuchalin, bronchial asthma can develop at any age, however, in about half of cases it begins in childhood and in another 30% - before the age of 40. In general, without differentiation by age group, women get sick more often than men.

Over the past 50 years, the incidence of bronchial asthma has increased 10 times. There are many reasons for the increase. This is the increasing use of chemistry in everyday life and at work, the use of a large amount of drugs, repeated cases of a respiratory infection, especially a viral one, an increased pace of life and the associated neuropsychic stress, smoking.

Etiology.

In the occurrence of bronchial asthma, a hereditary predisposition matters. Internal and external factors play a role in the development of the disease.

Internal factors - these are biological defects of the immune, endocrine systems, autonomic nervous system, sensitivity and reactivity of the bronchi, vascular endothelium of the lungs, the rapid response system.

External factors include:

1) infectious allergens (viruses, bacteria, fungi, yeast);
2) non-infectious allergens (pollen, dust, industrial, medicinal, food, insect and animal allergens).

In addition to these environmental factors, the following should be distinguished:

1) mechanical and chemical irritants (metal, wood, cotton dust, vapors of acids, alkalis, fumes);
2) meteorological and physicochemical factors (changes in air temperature and humidity, fluctuations in barometric pressure, Earth's magnetic field, physical effort); stressful, neuropsychic effects.

Infectious agents, in addition to their allergenic action, can also play another role:

A) reduce the threshold of the body's sensitivity to non-infectious allergens, increase the permeability for them of the mucous membrane of the respiratory system;

B) form a non-immunological way of changing the reactivity of target cells and effector systems.

Pathogenesis.

The central link in the pathogenesis of bronchial asthma is an altered reagent*bronchial stiffness,* which can be primary and secondary.

Primary change in reactivity there is congenital and acquired, due todirect effects of chemical and mechanical, physical factors and infection.

Secondary changes in bronchial reactivity are a manifestation of changes in the

reactivity of various body systems: immune, endocrine, nervous.

Bronchial reactivity changes under the influence of both immunological and notimmunological mechanisms.

Immunological mechanisms.

In a significant part of patients with bronchial asthma, changes in the reactivity of the bronchi are caused by disorders of the immunocompetent system, proceeding according to I, III, IV types of hypersensitivity reactions, i.e. with changes in humoral and cellular immunity. Immune reactions take place in the mucous membrane of the respiratory tract.

Type 1 (atopic, reaginic or anaphylactic) characterized by increased production of IgE (in response to exoallergens: pollen, drugs, bacteria). Ig E is fixed on mast cells and the immunological stage unfolds. Following this, a pathochemical stage unfolds - degranulation of mast cells occurs with the release of a large amount of substances with vasoactive, bronchospastic and chemotactic properties (histamine, serotonin) - primary mediators. Eosinophils and non-nitrophils secrete secondary mediators. Under the influence of an excess of biologically active substances, the permeability of the microvasculature increases, edema, serous inflammation, bronchospasm and other manifestations of the pathophysiological stage develop. Clinically, this is manifested by an acute violation of bronchial patency and the development of an asthma attack.

Reaction III type (immunocomplex type or Arthus phenomenon) occurs in the area of excess antigen with the participation of precipitating antibodies. The reaction develops under the influence of exoallergens and endoallergens. In type III, IgG and IgM are formed. The damaging effect of the formed antigen-antibody complex is realized mainly through the activation of complement, the release of lysosomal enzymes, under the influence of which damage to the basement membranes, spasm of bronchial smooth muscles, vasodilation, increases the permeability of the microvasculature.

Type IV (cellular), in which sensitized lymphocytes have a damaging effect, they are referred to as HRT, because allergic reactions develop in a sensitized patient 24-48 hours after exposure to an allergen.

In the formation of altered bronchial reactivity, a known role belongs to the local "breakdown" of the immune system. Thus, in AD, both congenital and acquired (due to repeated respiratory processes) decrease in secretory Ig A is noted, which reduces the ability to protect the mucous membrane from the damaging effects of bacteria and viruses. The phagocytosis system is disturbed, which in the respiratory organs is provided mainly by alveolar macrophages. The latter are not only involved in the capture of microorganisms, but also synthesize interferon. If their function is disturbed, the antiviral defense of the body is sharply reduced.

Non-immunological mechanisms.

When exposed to physical, mechanical, chemical stimuli, infectious agents, the reactivity of target cells and, above all, mast cells located along the respiratory

tract, decreases, which is accompanied by excessive production of biologically active substances, in response to which bronchospasm and mucosal edema develop. All this causes an attack of suffocation.

As a result of numerous studies of the immune-enzyme status of the body, it has been established that bronchial asthma occurs in the presence of primary or secondary immunodeficiency. There were identified 3 main groups of factors contributing to the onset of immunodeficiency: stress factor, vitamin imbalance and microelements. Lack of vitamin A increases the lability of lysosomes, etc.

Classification.

I. Stages of bronchial asthma.

1) *Predastma* - vasomotor disorders of the respiratory tract mucosa, acute and chronic pneumonia with elements of bronchospasm and allergy symptoms.

2) *Stage I*- pulmonary insufficiency is absent or there is I or II degree. It is divided into 3 degrees of severity: mild, when remissions are frequent; moderate severity (possibly asthmatic) - remissions are not frequent; severe - often an asthmatic condition and remissions are rare.

3) *Stage II* -protracted attacks of bronchial asthma, asthmatic condition. The disease is progressing.

II. Stages of development of bronchial asthma.

1) *Biological defects* in practically healthy people.

2) *Pre-asthma condition*... This is not a nosological form, but a sign of the threat of a clinically expressed bronchial asthma.

3) *Clinically formalized bronchial asthma* - after 1 attack or status of bronchial asthma.

III. *Forms of bronchial asthma.*

1) *Immunological.*

2) *Non-immunological.*

IV. Clinical and pathogenetic classification.

1) Atopic - indicating the allergenic allergen or allergens

2) Infectious-dependent - with an indication of infectious agents and the nature of infectious dependence, which can be manifested by the stimulation of atopic reactions, infectious allergies and the formation of a primary altered bronchial reactivity.

1) *And utoimunny.*

2) *Hormonal* - with an indication of the endocrine organ, the function of which is changed and the nature of dyshormonal disorders.

3) *Neuropsychic.*

4) *Adrenergic imbalance.*

5) *Primary altered bronchial reactivity*, which is formed without the participation of altered reactions of the immune, endocrine and nervous systems, can be congenital, manifest under the influence of mental, physical, mechanical factors of infectious agents. It is characterized by attacks of suffocation during exercise, exposure to cold, medications (acetylsalicylic acid).

V. According to the severity of the course.

1) *Easy flow.*
2) *Moderate course.*
3) *Heavy course.*

Vi. Phases.

1) *Aggravation.*
2) *A fading exacerbation.*
3) *Remission.*

Vii. Complications.

1) *Pulmonary:* emphysema, pulmonary insufficiency, atelectasis, pneumothorax.
2) *Extrapulmonary:* myocardial dystrophy, cor pulmonale, heart failure.

The clinical picture.

The main clinical manifestations of asthma are an attack of expiratory suffocation, cough, and shortness of breath.

When collecting anamnesis, it is necessary to clarify what precedes the attacks of suffocation. In patients with atopic asthma, attacks are preceded by contact with non-infectious allergens. In patients with an infectious-dependent type - inflammatory diseases of the respiratory system. In hormone-dependent asthma - with a decrease or withdrawal of steroid hormones or in connection with the menstrual cycle. Also preceded by an attack: neuropsychic stress, physical activity, inhalation of cold air, pungent odors, etc.

In the development of an attack, three periods are distinguished: the period of precursors, the height and the reverse development.

1.Period of seizure harbingers- occurs a few hours, minutes, sometimes days before the attack and is manifested by the following symptoms: vasomotor reactions from the nasal mucosa, discharge of abundant liquid watery secretion, sneezing, dry nose, itchy eyes, cough, difficulty in sputum discharge. In addition, headaches, excessive diuresis, nausea, fatigue. Some patients have an elevated mood.

2. ***The peak period***... Asphyxiation is of an expiratory nature and is accompanied by a feeling of compression, compression behind the sternum, which does not allow breathing freely. The inhalation is short, the exhalation is slow, convulsive, the duration of the inhalation is 2-4 times longer, accompanied by wheezing. Trying to make breathing easier, the patient takes a forced position (orthopnea). The patient sits, leaning forward, resting his elbows on his knees and resting his hands on the edge of the chair. The face is puffy, pale, with a bluish tinge, covered with cold sweat. The wings of the nose are inflated upon inhalation. The muscles of the shoulder girdle, back and abdominal wall are involved in breathing. Wheezing during an attack is interrupted by a cough, which in some patients is accompanied by sputum discharge, after which it becomes easier. The sputum is sticky, viscous, frothy. It may contain white, dense balls and threads, which are casts of mucus. Breathing 10-12 in 1 minute. Above the lungs, there is a percussion sound with a tympanic tinge. The lower borders of

the lungs are omitted, the mobility of the pulmonary edges is limited. Above the lungs, against the background of weakened breathing during inhalation and especially during exhalation, many dry, wheezing rales of various shades are heard. Relative cardiac dullness is reduced in size. Pulse of weak filling, quickened. With prolonged attacks of suffocation, there are signs of failure of the right ventricle, an enlarged liver, and bloating.

3. ***Reverse development period.*** All clinical manifestations of an attack of bronchial asthma gradually disappear.

Diagnostics.

From general clinical laboratory researcheosinophilia of peripheral blood, leukopenia, lymphocytosis should be noted. In the sputum of patients, many eosinophils, Charcot-Leiden crystals, Kurshman's spirals are detected. Neutrophilic leukocytes are found in large quantities in patients with infectious-dependent bronchial asthma. ESR increased.

ECGduring an attack, the T wave rises in all leads, the P waves in leads II and III often rise. In some patients, depression of the ST line in lead I is noted, which is explained by hypoxia of the heart muscle, which occurs due to respiratory failure.

X-ray examination increased transparency of the pulmonary fields is determined. The pulmonary pattern is strengthened, the shadows of the lung roots are expanded and strengthened.

Differential diagnosis.

When carrying out a differential diagnosis of BA with other diseases, resistanceobstruction of the bronchi, as the main symptom should be used an attack of suffocation, typical for asthma.

A differential diagnosis should be made between infectious-dependent asthma and chronic obstructive bronchitis. In contrast to BA patients, shortness of breath and shortness of breath in patients with chronic obstructive bronchitis does not have paroxysmal character, persists constantly, intensifies after physical exertion and is accompanied by a cough with sputum secretion, and bronchial asthma is characterized by time-limited attacks of suffocation with sputum secretion at the end. attack.

They also differentiate with acute respiratory viral diseases, with tracheobronchial dyskinesia, etc.

Complications.

A formidable complication of asthma that can lead to death is status asthmaticus. The factors predisposing to its development are almost always the result of inadequate therapy. Most often these are: uncontrolled intake of symptomatic drugs and corticosteroids, exacerbation of a chronic or acute inflammatory process in the bronchopulmonary apparatus, abuse of hypnotics and sedatives.

Treatment.

Among the various methods of treating AD, there are methods aimed at eliminating the causes of the disease (etiological treatment) and its manifestations (pathogenetic treatment).

As measures aimed at etiological factors, first of all, organizational measures are taken: elimination of allergens from the environment of patients, sanitation of foci of infection.

The main task of pathogenetic, symptomatic treatment is to restore bronchial patency, prevent asthma attacks. Use broichospray drugs. They are divided into 3 groups:

1) stimulants of adrenergic receptors (sympathomimetics);

2) phosphodiesterase inhibitors;

3) acetylcholine blockers (anticholinergics).

Sympathomimetics are used:

4) stimulants @ iYa receptors (adrenaline, ephedri);

5) stimulators of Y1 and Y2 receptors (izadrin);

6) selective stimulants of Y2 receptors (astmonent, salbutamol, berotek).

Light attack patients stop themselves, using tablets of theofedrine, aminophylline, 0.1-0.15 g 2-3 times a day, or in inhalers (asthmopent, berotek), or subcutaneous administration of adrenaline (0.3 ml of 0.1% solution) or ephedrine (0.5 ml of 5% solution) in combination with papaverine (1 ml of 2% solution) and antihistamines (1 ml of diphenhydramine or suprastin).

With a moderate to severe attackaminophylline is administered intravenously, and in the absence of effect, 60-90 mg of prednisolone is administered intravenously in a stream. Infusion therapy is carried out with sodium bicarbonate.

With a prolonged attackstatus asthmaticus may begin. Then you need to start intensive therapy:

7) *oxygen therapy;*

8) *infusion therapy*, in which glucose, insulin, heparin, sodium bicarbonate are administered;

9) administration of corticosteroids: prednisolone 60-90 mg every 4 hours, hydrocortisone 1 mg per hour per 1 kg of body weight. In order to avoid undesirable side effects associated with prolonged use of hormones, it is recommended to prescribe high doses (40 mg / day) from the very beginning and then, after eliminating the symptoms of the disease, quickly (5-7 days) reduce these drugs to the minimum dose or completely withdraw these drugs.

During the period of exacerbation, they are also used:

1) ***broicholytics*** parenterally, in suppositories or orally. Eufillin 10 ml 2.4% solution intravenously stream or drip; in powder together with papaverine, platifillin and diphenhydramine;

2) ***sympathomimetics*****:** *berotec, salbutamol*1-2 breaths 4-5 times a day. Continuous use of inhalers is not recommended in order to avoid side effects (tachycardia, arrhythmia, increased bronchospasm);

3) *mucolytics - mucosolvin, acetylcysteine, mucaltin*6-8 tablets per day, infusions and decoctions of thermopsis, coltsfoot, hot alkaline drink, iodine preparations (3% solution of potassium iodide, 1 tablespoon 3-4 times a day). A special solution is prepared from potassium iodide, distilled water, aminophylline and ephedrine - 1 tablespoon 3 times a day;

1) antibiotic therapy with infectious BA;

4) *therapeutic bronchoscopy* under anesthesia with intrabronchial administration of antibacterial and mucolytic drugs;

5) physiotherapeutic methods of influence, breathing exercises, chest massage;

6) *Ketotifen*- has anti-anaphylactic activity, inhibits the release of histamine. Inside, 0.001-0.002g .;

2) *Ethanol* - i / v drip - defoamer;

nine) *Antimediators - diphenhydramine* (1 ml of 1% solution in / m), suprastin (2 ml of 2% solution in / m), blocking H1 -receptors, reduce the effect of histamine. Antiserotonin: stugerone, cinnarizine;

10) *Ca antagonists: isoptin, finoptin,* inhibit the transmembrane Ca flux, which leads to a decrease in the release of mediators.

In addition, unloading and dietary therapy, psychotherapy, acupuncture *Exercise therapy (breathing exercises), spa treatment.*

Pleurisy (P)

Pleurisy is an inflammation of the pleural sheets, accompanied by the formation of fibrinous overlays on their surface (dry or fibrinous pleurisy), or the accumulation of exudate in the pleural cavity (exudative pleurisy).

Etiology of pleurisy (P)

By etiology, all pleurisy are divided into I) infectious and II) non-infectious, as well as idiopathic (with unknown etiology). I. Causes of infectious P:

1. Bacterial (staphylococcal, pneumococcal, gram-negative microbes, etc.).
2. Tuberculous (in 20% -25% of patients with pleurisy)
3. Fungal (coccidioidosis, actinomycosis, etc.).
4. P. of viral, mycoplasma and rickettsion origin.

five. Syphilis, typhoid and typhus / tularemia brucellosis. II. Causes of non-infectious P.

1.Tumor processes:

a) primary tumors of the pleura (mesothelioma),

b) metastatic malignant tumors of the pleura,

c) Meigs syndrome (pleurisy and ascites with ovarian neoplasms).

d) tumor diseases of the lymphoid system (LHM, lymphosacrcoma, etc.)

2. DBST group (SLE, rheumatism, RA, dermatomeasitis).

In the overwhelming majority, P is a process that complicates the course of

certain diseases of the lungs, chest, diaphragm and subphrenic space, as well as a number of systemic or oncological diseases.

3.Systemic vasculitis (periarteritis nodosa).

4. Thromboembolism of the branches of the pulmonary artery, infarction - pneumonia.

5. Surgery on the chest and rib injuries.

6.Group P of other origin:

but) postinfarction dressler syndrome,

b) hemorrhagic diathesis (Wergolf's disease).

in) pancreatitis (the so-called enzyme P), '

d) periodic illness.

Pathogenesis of P.

Two closely related factors play a role in the development of P

I. direct impact on the tissues of microbes and their metabolic products.

II.pathological changes in the general and local reactivity of the patient. Direct penetration of microbes into P can be as follows:

a) by contact, from subpleural foci (pneumonia, tuberculosis, bronchiectasis, etc.).

b) lymphagenous way...

c) hematogenous

d) direct infection from the external environment.

In the realization of the picture P, an important role is played by:

1. Increased permeability of blood and lymph vessels in systemic vasculitis and under the influence of toxic products (tumor and endogenous intoxication) and proteolytic enzymes (in acute pancreatitis).

2. Disruption of lymph circulation as a result of blockage of its outflow pathways.

3. Development of local and general reactions of allergic origin.

Classification P (Putov N.V., 1984).

I. Etiology:

1. Infectious P (indicating the pathogen - pneumococcal, staphylococcal, tuberculous, etc.).

2. Non-infectious (indicating the main process, the appearance or complication of which is P - lung cancer, SLE, rheumatism, systemic vasculitis, etc.).

3. Idiopathic P (of unknown etiology).

II. The nature of the exudate:

1.Fibrinous.

2.Grey.

3. Serous - fibrinous.

4. Purulent.

5.Hiley.

6.Holystirinic.
7. Eosinophilic
8. Putrid.
nine.Hemorrhagic.

III... Flow

1.Sharp.
2. Subacute.
3. Chronic.

IV. Localization

1. Diffuse
2. Encapsulated: a) apical; b) diaphragmatic; c) parietal; d) inter-share; e) paramediastinal; f) bone-diaphragmatic.

The main clinical syndromes of P.

one. Dry pleurisy: chest pain worsening with forced breathing, cough: the irradiation of pain depends on the localization of pleurisy - with diaphragmatic pleurisy, pain is given to the upper abdomen or to the neck, i.e. along the phrenic nerve. Apical pleurisy is accompanied by soreness of the trapezius and pectoralis major muscles (symptoms of Sternberg and Pottenger). Pain syndrome is combined, as a rule, with limited mobility of the lungs, low-grade fever, general weakness, during ascultation - pleural friction noise.

In the KLA - moderate leukocytosis, accelerated ESR, LAC - an increase in sialic acids, fibrin, seromucoid. X-ray dry pleurisy is not recognized, but signs of the underlying disease (pneumonia, tumor, tuberculosis, etc.)

2. Exudative pleurisy: general weakness, increased shortness of breath, fever with chills, sweating, anorexia, lagging behind the sick half in the act of breathing, and evilfemininity of the intercostal spaces. With the accumulation of exudate in total from 500 ml. and more signs of dullness of percussion sound appear. The upper level of dullness in the accumulation of exudate in the pleural cavity has the form of a parabola, called the Ellis-Damoiseo line. The lowest point on this line is located at the back of the spine, and from here the line rises in an arc up to the corner of the scapula, then drops slightly, and at the level of the middle aximilar line rises again, so that it again goes down in an arc-like manner to the lowest point in front of the sternum. The reason for such an arcuate direction of the dullness level is the unequal compliance of the areas of light pressure of the liquid. The upper level of dullness is higher in those places where the compliance is greater and vice versa. At the upper border of the exudate, both pleural sheets stick together and therefore the configuration of dullness does not change when the patient's position changes. In case of exudative pleurisy on the healthy side, behind and below the spine, a Rauchfus rectangle with a percussion sound is determined. Its hypotenuse is the continuation of the Ellis-Damoiseau line on the healthy half of the chest, one leg is the spine, and the other is the lower edge of the lung. The reason for the appearance of the Rauchfus rectangle lies in the displacement to the healthy side of the aorta, which, when percussed,

gives a dull sound, as well as in the ability of the spine to conduct percussion vibrations to the direction of the exudate and, as a result, mixing this sound from the exudate with the normal pulmonary sound on the healthy side. In cases of encapsulated mediastinal pleurisy - facial edema, hoarseness, dysphagia, neck swelling. Percussion - a dull sound, auscultation - a sharp weakening or lack of breathing, tachycardia, deaf heart sounds.

In the KLA - leukocytosis, accelerated ESR, anemia, toxic granularity of leukocytes.

LHC: increased content of sialic acids, seromucoid, and globulins. Study of pleural fluid: protein more than 3%, relative density 1.018, positive Rivalta test, LDH content of more than 1.6 mmol (l. Ch.), Neutrophils predominate in the remainder, straw-yellow color, with empyema pus.

Instrumental research.

X-ray studies: intense darkening with an oblique upper border. displacement of the mediastinum in the opposite direction.

Ultrasound procedure: fluid in the pleura

Differential diagnosis of various exudative pleurisy

The main criteria are clinical and paraclinical features. So parapneumonic Ps are hidden by the main process and are, as a rule, with small effusion.

Severe pain syndrome in the onset of pneumonia requires the search for P, which can begin in the form of dry, and then transformed into exudative.

With heart attacks, it is easy to P exudate, usually hemorrhagic and in small quantities.

Carcinomatous P in the early stages is accompanied by pain syndrome, hemorrhagic exudation with the presence of atypical cells.

In cases of compression of the thoracic lymphatic duct by metastases, the effusion is chylous.

Pleurisy of tuberculous genesis requires taking into account the following factors:

but) relatively young age of patients,

b) positive tuberculin tests,

in) severe intoxication against the background of a moderate temperature reaction

d) contacts with a patient with tuberculosis Algorithm diagnostic search for exudative P. is presented:

1) clinical and X-ray examination before pleural puncture.

2) pleural puncture with inoculation of punctate on nutrient media and establishment of etiology, Rivalta's test, specific gravity, amount of protein, cytology of sediment, analysis on VC, lupus cells, atypical cells.

3) repeated X-ray examination after puncture.

You need to know that the nature of the exudate is determined by its origin, the

rate of accumulation and resorption of the effusion, the duration of its existence, etc.

So with moderate effusion and good resorption, fibrinous pleurisy develops,with a predominance of resorption over exudation, adhesions and moorings are formed, and with a predominance of exudation over resorption, serous or serous-fibrous P. In case of hemorrhagic disease, one should think about oncological processes, pleural mesothelioma, hemorrhagic diathesis, pulmonary infarction or trauma, pancreatitis or anti-coagulitis. With the predominance of the allergic onset, eosinophilic exudate is obtained, as a rule, with compression of the thoracic duct, chyle exudate, and with chronic long-term P. (in particular, with tuberculosis) - cholesterol effusion.

Recommended research volume for pleurisy

one. General analysis of blood and urine.

2... LHC: protein and its fractions, sialic acids, fibrin, seromucoid.

3... Radiography of the lungs (with exudative P. before and after pleural puncture)

four... Studies of pleural fluid: a) Rivalta's test, b) specific gravity, amount of protein, cytology of the sediment, analysis for CD, lupus cells, atypical cells.

Treatment

Treatment of sick P. includes:

1. Treatment of the underlying disease (impact on etiological factors)
2. Pathogenetic treatment taking into account the effusion and clinical - morphological P.
3. Fight against the main clinical syndrome (intoxication, allergic manifestations, correction of protein metabolism disorders)
4. Increased general reactivity and immunomodulatory therapy.

I) Etiological treatment (treatment of the underlying disease) often leads to the elimination or reduction of symptoms of P. Therefore, the treatment of the underlying disease, which is given in the above classification, is the basis.

II) The use of anti-inflammatory and desensitizing therapy, anti-inflammatory drugs have an analgesic and resorption effect.

Assign: 1) Acidum acetylsalicylicym 1.0 X 3-4 times a day after meals

2) Paracetamol 0.5 X 3 times a day

3) painful cough Tab. Codeini phosphorici 0.01X 2-3 timesday, as well as non-narcotic antitussives (do not cause addiction and do not depress the respiratory center):

but) glaucine hydrochloride 0.05 2-3 times a day

b) ledin 0.05 2-3 times a day

in) bitiodine 0.01 2-3 once a day

d) libexin 0.1 3-4 times a day

e) tusuprex 0.01 3 times a day

five. in case of severe pleural pain, Metindol is recommended - retard 0.075X 1-2 times a day, voltaren 0.025 X 2 - 3 times a day.

III) Evacuation of exudate.

The evacuation of exudate has two goals: a) prevention of the development of empyema, elimination of compression and displacement of vital organs. To avoid collapse, no more than one and a half liters of liquid should be removed. Taking into account the indicated indications, pleural puncture is performed even in the early period of exudative P. (if the dullness border in front reaches 2 ribs, dyspnea is increased, the heart is displaced). In other cases, puncture with removal of exudate is best done in the phase of stabilization or resorption, since early evacuation leads to an increase in negative pressure in the pleural cavity and accumulation of exudate.

At P. of nonspecific infectious etiology after removal of exudate in the cavity is injected with antibacterial agents. Acute pleural empyema requires removal of purulent exudate followed by the introduction of antibiotics into the cavity.

IV) Increase of general reactivity and immunomodulatory therapy.

These measures are performed with prolonged fibrinous P. Immunomodulatory therapy normalizes the system of general and local immunity. It is advisable to carry out this therapy after studying the immune status, phagocytosis and bronchopulmonary defense function. These drugs include: a) decaris - 100–150 mg per day for 2–3 days, then a 4-day break and such cycles are 8–12. B) T - activin 100 subcutaneously 1 time per day for 3–4 days) katergen - 0.5 X 3 times a day during meals for two weeks, d) prodigiosan - is prescribed in gradually increasing doses from 25 to 100 with an interval of 3-4 days (course of 4-6 injections), e) ribomunyl - tab. by 0.00025 and applied according to the following scheme: 3 table. in the morning on an empty stomach every four days for 3 weeks of the first month, then 3 tables. the first 4 days of each month for 5 months, f) lycopid - a daily dose of 1 mg in chronic exacerbation and unstable remission. With sluggish, often recurrent P is most appropriate to apply a daily dose of 10 mg. g) oxymetacil - 0.25 X 3 times a day after meals for 3-4 weeks h) UV - blood, laser blood irradiation.

To increase the nonspecific resistance of the body, adaptogens are used (setting ginseng, extract of Eleutherococcus, aralia, radiola rosea, pantacrine, saparal).

V) **Detoxification and correction of protein metabolism disorders**

These measures are carried out with exudative P and pleural emphysema.

Assign: 1) hemodesis

2) p - p Ringer

3) 5% glucose solution

4) solution of Albumin 10% once 2 - 3 days only 3-4 times

five) 400 ml of active fresh frozen plasma 1 time in 2 - 3 days only 2-3 tons times.

6) retabolil - 1 ml / m 1 time in 2 - 3 weeks only 2-3 times

VI) Physiotherapy, LF1S, massage

Common diseases, heating compresses, calcium chloride electrophoresis. With effusion pleurisy, physiotherapy is carried out in the resolution phase in order to accelerate the resorption of exudate in pneumonia, and to reduce pleural adhesions (Electrophoresis with calcium chloride, heparin, decimeter waves, paraffin therapy). As the acute phenomena subside, manual chest massage, sanatorium treatment in local suburban sanatoriums and in the resorts of the southern coast of Crimea are shown.

IDIOPATHIC FIBROSING ALVEOLITIS

Idiopathic fibrosing alveolitis (synonym: Hammen-Rich disease, Scadding's syndrome, Osler-Charcot disease, sclerosing alveolitis, fibrous pulmonary dysplasia, etc.) is a peculiar pathological process in the lungs of an unclear nature, accompanied by increasing respiratory failure (DN) due to progressive pneumofibrosis ...

Etiology

The etiology of idiopathic fibrosing alveolitis (ELISA) is unknown. Suggestions are made about the possible viral nature of the disease. However, the largest number of supporters has a hypothesis about the polyetiology of this disease, according to which factors of a bacterial, viral, allergic, toxic or other nature can play the role of a trigger moment that causes a stereotypical reaction of the lung tissue. There are reports of a genetic predisposition to this disease.

Pathogenesis

The pathogenesis of ELISA is insufficiently developed. An important role is played by disorders in the collagen-collagenase-collagenase inhibitors system. The release of neutrophilic chemotactic factor by alveolar macrophages causes an increased influx of neutrophils into the lungs, as a result of the breakdown of which proteolytic enzymes (primarily collagenase) are released and collagen is cleaved by activated neutrophilic collagenase, followed by enhanced resynthesis of pathological collagen. In the pathogenesis of the disease, a significant role is

also assigned to lymphocytes, which produce the so-called "migrating inhibitory factor", which normally inhibits the synthesis of collagen by 30-40%. Disruption of the suppressive effect of this factor may be one of the causes of collagen overproduction. Damage to the lung tissue by circulating immune complexes (CICs) is possible.

The picture of progressive respiratory failure with ELISA is determined by the thickening and thickening of the interalveolar septa, as well as obliteration of the alveoli and capillaries by fibrous tissue. An increase in the elastic resistance of the lung tissue leads to a decrease in extensibility and, accordingly, insufficient expansion of the alveoli,deterioration of alveolar ventilation and an increase in the work of breathing. Hypoxemia in patients with ELISA depends not only on the functional properties of the alveolar-capillary membrane itself, but also on the reduction of the capillary bed, which leads to an increase in the blood flow rate in the functioning capillaries of the lungs and thereby to a decrease in not only the area of failure, but also the time of contact of the erythrocyte with the alveolar air. Violation of antihypertensive-perfusion relations is aggravated by a decrease in the permeability to gases of the alveolar epithelium due to metaplasia of its cubic, as well as damage to small airways by the type of obliterating bronchiolitis. Hypoxemia is aggravated, probably by reflex vasoconstriction of the lungs due to endocapillary hypoxemia (Euler-Liliestrand reflex),

Pathological picture

Pathomorphological changes in the lung parenchyma with ELISA can be represented in the form of three interrelated phases (stages):

- interstitial edema;
-interstitial "inflammation" (alveolitis);
-interstitial fibrosis.

The central place in the modern concept of the pathogenesis of the disease is given to the stage of alveolitis. This stage is characterized by: the development of interstitial edema, exudation of serous-fibrinous fluid into the alveolar septa and into the alveoli, lymphocytic infiltration of the interalveolar septa. The progression of the process leads to disruption of the structure of endothelial cells of pulmonary capillaries, basement membrane, alveolar cellsI and II types, lining the alveoli. Desquamated, alveolar cells expose the basement membrane, forming an "alveolar ulcer". The alveolar epithelium lining the thickened interalveolar septa gradually acquires the signs of a cubic epithelium, unable to provide gozo exchange. The transition of the process to the stage of interstitial fibrosis is irreversible and leads to progressive respiratory failure.

There are two clinical and morphological forms of ELISA: mural (with a predominant lesion of the interstitial tissue of the lungs) and desquamative (with damage mainly to the alveoli).

There are 5 degrees of histological changes:

1) connective tissue thickening and infiltration of the alveolar septa;

2) filling the alveolar lumens with secretions and cells;
3) involvement of bronchioles in the process with the formation of small cysts and destruction of the structure of the alveoli;
4) the normal structure of the lung tissue is completely steamed cavities gradually increase;
5) the formation of the so-called "honeycomb (or cellular) lung.

Clinical picture

The disease is more common in females (3: 1). The average age of patients is 41.8 ± 1.4 years.
There are no pathognomonic, characteristic only for ELISA signs of the disease. In approximately 37% of patients, the onset of the disease is acute and is characterized by an increase in body temperature to 38-40 ° C, in the rest (63%) of patients, the onset of the disease is gradual.

Steadily progressive shortness of breath is one of the most common and persistentclinical signs of ELISA - is detected in all patients. Shortness of breath that has arisen once tends only to increase, and the rate of its progression depends on the activity of the pathological process. Most patients note a characteristic feature of shortness of breath - the inability to breathe deeply. In patients with a pronounced degree of it (dyspnea at rest), tachycardia up to 90-130 per minute is detected. Cough is observed in 90% of patients. As the disease progresses, it intensifies, provoked by an attempt to take a deep breath. One of the signs of the disease is weight loss (in 36% of patients). In 47% of patients, aching pains are noted at the lower angles of the shoulder blades, aggravated by a deep breath. An increase in body temperature to subfebrile or fibril indicates the activation of the pathological process. Arthralgias and Reyio's syndrome are relatively rare. General weakness and fatigue are noted in all patients. The frequency and severity of cyanosis depend on the severity of the disease. Almost half of the patients (46%) show characteristic changes in the nail phalanges like drumsticks and watch glasses.

With percussion of the lungs, the dullness of the percussion sound over the affected area is determined (as a rule, these are the lower pulmonary fields). One of the characteristic auscultatory signs of ELISA is the shortening of the inspiratory and expiratory phases. Crepitation is heard, as a rule, on both sides, mainly along the posterior and mid-axillary lines, as well as between the shoulder blades. This sound phenomenon detected by>50-87% of patients in the literature are called cellophane crackle.

Against the background of hard breathing, dry wheezing can be heard, with forcednom breathing, the number of wheezing increases.

In patients with ELISA, another auscultatory phenomenon is revealed - the so-called squeak, which by its nature resembles the sound of friction of a plug. It is heard on inspiration, mainly over the region of the upper pulmonary fields (along the anterior surface of the chest). The presence of "beeping" only in patients with widespread pneumofibrosis allows us to consider this sound phenomenon as a sign of pronouncedpleuropneumosclerotic changes.

Complications of ELISA include: pneumothorax, formation of cor pulmonale, pleural exudates, pulmonary embolism.

Diagnostics

CBC changes are nonspecific. Leukocytosis with a shift to the left, an increase in ESR, dysproteinemia with a2- and y-globulinemia, a moderate increase in sialic acids, seromucoid, haptoglobin, a positive reaction to CRP may indicate an inflammatory stage of the disease (alveolitis stage). In patients with IF, antinuclear, antipulmonary antibodies, rheumatoid factor, CEC are found. The analysis of X-ray changes makes it possible to distinguish between groups of patients with a predominant lesion of the interstitial tissue of the lungs (mural variant of the disease) with a predominant lesion of the alveoli (desquamative variant of the disease) and with radiological signs corresponding to the picture of the cellular lung. Changes on radiographs are bilateral, with the greatest severity in the lower lobes of the lungs. As the disease progresses, the volume of the lower lobes decreases, which is accompanied by a rise in the diaphragm. At angiopulmonography in patients with ELISA, the expansion of the central branches of the pulmonary artery is revealed, indirectly indicating a higherpressure in the pulmonary circulation.

In patients with ELISA, the partial tension of oxygen in arterial blood (PaO2) decreases. Violation of the ventilation capacity of the lungs with ELISA leads to an increase in the respiratory rate (RR), a decrease in the volume of inspiration, a decrease in the vital capacity of the lungs, to a lesser extent in the residual volume of the lungs, an increase in the elastic resistance of the lungs, a decrease in the diffuse capacity of the lungs (DL) and the diffusion capacity of the alveolar-capillary membrane , absence of bronchial disorderspatency.

For the purpose of cytological diagnosis of ELISA, a study of BALF is carried out. This disease is characterized by an increase in the number of neutrophils in the BALF with a normal or slightly increased content of lymphocytes, it is determined that it is absent in it.collagenase is normal.

The highest diagnostic informational content of open biopsy, which with ELISA is about 95%. Transbronchial lung biopsy with ELISA maloinformative.

X-ray computed tomography(RCT) helps to detect changes before they appear on radiographs, often reveal the symptom of "ground glass" (gentle homogeneous darkening of the pulmonary fields), interstitial changes. Based on the results of CT, it is possible to accurately determine the lesion site for taking a biopsy. The study in dynamics provides fairly accurate information about the progression.

Scintigraphy with 67Ga - non-specific method to assess the degree inflammatory response.

Differential diagnosis

The nature of dyspnea is extremely important differential diagnostic value: once it arises, it tends only to progression. Dyspnea does not depend or depends little on the time of day, ambient temperature and other factors. A distinctive feature of the breathing of patients is the shortening of the inhalation and exhalation phases and, in connection with this, increased respiration per unit of time, hyperventilation syndrome. Another characteristic is that trying to inhale deeply causes coughing. The listed characteristics of shortness of breath allow already at the first interview of the patient to exclude such diseases as bronchial asthma, aboutstructural bronchitis, pulmonary emphysema.

Differential diagnosis of ELISA should be carried out with bilateral pneumonia, chronic pneumonia, sarcoidosis, bronchoalveolar cancer, silicosis, as well as with the syndrome of fibrosing alveolitis in diffuse diseasesunderwear.

Treatment

Patients with ELISA with early diagnosis (edema phase and alveolitis phase), as well as in cases of predominantly desquamative forms of the disease, corticosteroids are prescribed in maximum doses (40-50 mg in terms of prednisone) for 3-10 days. This dose of the drug gradually over 6-8 months. (depending on the effect) is reduced to maintenance (2.5 - 5 mg per day). The duration of treatment is on average 18-20month

With the transition of the pathological process to the stage of interstitial fibrosis, the appointment of penicillamine in combination with corticosteroids is indicated, the initial dose of cosome in these cases is 15-20 mg per day.

Two treatment regimens for ELISA with penicillamine have been developed:

1) in the 1st week - 0.3 g per day, in the 2nd week - 0.6 g / day, in the 3rd - 1-2 g / day, then the dose is reduced in the reverse order. The maintenance dose is 0.15-0.3 gper day for 1-2 years.

2) 0.3 g of penicillamine per day for 4-6 months, then 0.15 per day for within 1-1.5 years.

The indications for treatment according to the first scheme are the acute course of the disease and the exacerbation of the pathological process. The second scheme is used in the chronic course of ELISA without clearly expressed exacerbations. In the presence of pronounced changes in the immunological status, the appointment of azathioprine is indicated, less often cyclophosphamide, chlorobutin. Azathioprine is prescribed according to the scheme: 150 mg / day for 1-2 months, then 100 mg / day for 2-3 months and then a maintenance dose (50 mg / day) for 3-6 months. Duration of administration is 1.9 years. ,

Another ELISA treatment regimen has been developed, including the simultaneous appointmentcorticosteroids, penicillamine and azathioprine.

In order to reduce the catabolic effect of corticosteroids, it is advisable to use anabolic hormones (retabolil, nerobol). To avoid a deficiency of potassium, calcium, magnesium, Panangin (asparkam), calcium preparations are prescribed. For the prevention of osteoporosis, as well as its treatment, calcitrin is prescribed at 3 IU / m throughday, in courses of 1-1.5 months and vitamin D.

Long-term use of azathioprine and (or) penicillamine leads to a deficiency of vitamin B6, therefore, it is necessary to include pyradoxin in treatment (daily dose from 10 to 80 mg).

In the complex treatment of patients with ELISA, it is advisable to include veroshpiron, which, in addition to the diuretic and potassium-saving effect, has a positive effect on lung perfusion, reduces interstitial edema, and has immunosuppression.pressing action.

Of the antioxidants, vitamin E is prescribed (50% solution, 0.2-0.6 g per day), sodium thiosulfate intravenously (5-10 ml of a 30% solution) for 10-14 days.

Hemosorption is indicated for pronounced shifts in the immune status of patients. ELISA.

Dispensary observation

The scheme of dispensary observation of patients with ELISA depends on the degree of compensation of the disease. Thus, group I (with a compensated course of the disease) includes patients with grade I DN (dyspnea with moderate exercise), group II (with a subcompensated course) - patients with grade II DN (dyspnea with light exercise) and group III (with decompensated course) - patients with DNIII degree (dyspnea at rest, cyanosis).

The maintenance dose of the main (basic) drugs should depend on the degree ofpenalties for compensation of the disease (Table 1).

Table 1.

Daily doses of essential drugs with maintenance therapy.

Dispensary observation group			
	(based on		
II-subcompensated flow			
III-decompensated course			

Dispensary observation of patients with ELISA is carried out by a pulmonologist, however, in the absence of relapses of the disease within 3 years, the patient can be transferred toobservation of the local (shop) therapist.

Forecast

The life expectancy of patients with ELISA is on average 4-6 years, but varies widely: from several months to 20 years or more. Early diagnosis and the appointment of adequate treatment can significantly improve the prognosis.

SARCOIDOSIS

Sarcoidosis (Benier-Beck-Schaumann disease) is a chronic multisystem disease of unknown etiology, characterized by the accumulation of T-lymphocytes and mononuclear phagocytes, the formation of non-caseinfected epithelioid granulomas and disruption of the normal architectonics of the affected organ. Everyone can be amazedorgans other than the adrenal glands.

Prevalence. Sarcoidosis occurs in all countries of the world, but the highest incidence is recorded in the northern countries, in particular North America and Europe, the incidence of the disease in which averages 10-40 cases per 100 thousand population. The highest prevalence of sarcoidosis is in the Scandinavian countries (64 per 100 thousand population). The disease is less common in southern countries with hot climates. The prevailing age of patients is 20-40 years old, the disease rarely affects children and the elderly. There are currently no reliable epidemiological data in Russia.

The etiology is unknown. Most authors consider sarcoidosis to be a polyetiological disease associated with impaired responsiveness and genetic factors.

The etiological factors of sarcoidosis presumably include chlamydia, Lyme borreliosis, latent viruses, Mycobacterium tuberculosis, but the lack of identification of any infectious agent and epidemiological relationships casts doubt on the infectious etiology of sarcoidosis.

Inhalation of metal dust or smoke can cause granulomatous changes in the lungs similar to sarcoidosis. The dust of aluminum, barium, burrillium, cobalt, copper, gold, rare earth metals, titanium and zirconium has antigenic properties, the ability to stimulate the formation of granulomas. Academician A.G. Rabukhin considered pine pollen as one of the etiological factors, however, the relationship between the frequency of the disease and the area dominated by pine forests is not always found.

Pathogenesis. The earliest change in sarcoidosis of the lungs is lymphocytic alveolitis caused by alveolar macrophages and T-helper cells that release cytokines (associated with the development of pulmonary fibrosis in sarcoidosis). Alveolitis is required for the subsequent development of granulomas. Sarcoidosis is considered a granulomatosis mediated by an intense cellular immune response at the site of disease activity. Granulomas can form in various organs (lungs, skin, lymph nodes, liver, spleen). They contain a large

the number of T-lymphocytes. At the same time, patients with sarcoidosis are characterized by a decrease in cellular and an increase in humoral immunity: in the blood, the absolute number of T-lymphocytes is usually reduced, while the level of B-lymphocytes is normal or increased. It is the replacement of lymphoid tissue with sarcoid granulomas that leads to lymphopenia and anergy to skin tests with antigens (AH).

In 60% of patients with sarcoidosis, the production of angiotensin-converting enzyme (ACE) by epithelioid cells of noncaseated granuloma is increased. In the early stages of the disease, elevated serum ACE levelsaccompanies violations of patency at the level of small bronchi (decrease in instantaneous volumetric velocities -MOC? s) -

Increased blood lysozyme levels found in 50% of patients with sarcoidosis (secreted by macrophages and giant cells in the granuloma). In the fluid of bronchoalveolar lavage (BAL) of patients with sarcoidosis, an increase in the level of hyalungonate (hyalunic acid) was revealed at the stage of fibrosis formation. According to a number of authors, this indicates the activation of fibroblasts.

Disorders of calcium metabolism (hyperkalemia, hypercalciuria) were found in patients with sarcoidosis, which is caused by dysfunction of blood monocytes, macrophages in the lung tissue and the formation of granulomas in the lung. In the development of hypercalcemia, a certain role is played by hypersensitization or increased production of vitamin D, in which alveolar macrophages and sarcoid granulomas are actively involved. There are reports that a rapid increase in blood and calcium levels can cause severe illness in sarcoidosis patients, stimulating an acute abdomen,polyuria, and polydipsia.

Due to prolonged calcemia, the formation of calcifications is observed in the kidneys (nephrolithiasis), lymph nodes, in the mucous membrane of the stomach, lungs and other organs.

Pathomorphology. there are three pathological stages of sarcoidosis: pregranulomatous, i.e. alveolitis, granulomatous and fibrous.

The main pathomorphological substrate of sarcoidosis is epithelioid granuloma, which consists almost exclusively of epithelioid cells, single giant cells of Pirogov-Langhans, with a narrow rim of lymphocytes around the tubercle, without foci of cheesy necrosis in the center and perifocal inflammation around. A characteristic feature of sarcoid granuloma is the presence of sinusoidal or capillary blood vessels in it, which distinguishes it from tuberculoustubercle.

Classification.

Until now, there is no universal classification of sarcoidosis. In 1994, a classification of intrathoracic sarcoidosis was developed (Table 1).

Table 2.

Classification of intrathoracic sarcoidosis (OPSharma, 1994).

Stage I - bilateral mediastinal lymphadenopathy
Stage II - bilateral mediastinal lymphadenopathy and parenchymal infiltration...
Stage III - parenchymal infiltration in the form of dissemination
Stage IV - widespread interstitial fibrosis and bullous transformation.

The Central Research Institute of Tuberculosis of the Russian Academy of Medical Sciences together with Hungarian specialists (A.G. Khomenko et al., 1982) proposed the following classification:

I. Clinical and radiological forms:

1. Sarcoid intrathoracic lymph nodes.
2. Sarcoidosis of the lungs and intrathoracic lymph nodes.
3. Sarcoidosis of the lungs.
4. Respiratory sarcoidosis combined with lesions (single) other organs. to
five. Generalized sarcoidosis with respiratory involvement.

P. Development phase of the process:

1. Active.
2. Stabilization phase.
3. Regression phase.
4. Chronic.

Sh. The nature of the process:

1. Abortive.
2. Slow motion.
3. Progressive.
4. Chronic.

IV. Complications: bronchial stenosis, hypopneumatosis, atelectasis, respiratory and cardiac failure, etc.

V. Residual changes after stabilization or cure of the disease:

1. Pneumosclerosis.
2. Emphysema of the lungs (bullous, diffuse).
3. Adhesive pleurisy.
1. Fibrosis of the roots of the lungs (with or without calcification intrathoracic lymph nodes).

Clinic.

Sarcoidosis affects various organs and systems. Most often (in 90% of patients), lung lesions develop. The most common concerns are increased fatigue (71% of patients), shortness of breath (70%), arthralgia (52%), muscle pain (39%), chest paincage (29%), general weakness (22%). Skin lesions are found in 25% of patients with sarcoidosis.

The most common manifestations include erythema nodosum, plaques, maculopapular rash, and subcutaneous nodules. Along with erythema nodosum, swelling or hyperthermia of the joints is noted. Most often, a combination of these signs appears in the spring. Arthritis in sarcoidosis usually has a benign course, does not lead to joint destruction, but recurs. Changes in peripheral lymph nodes, especially cervical, axillary, elbow and inguinal, are noted very often. On palpation, the nodes are painless, mobile, and compacted (they resemble rubber in consistency). Unlike tuberculosis, they do not ulcerate with sarcoidosis.

Pulmonary manifestations of sarcoidosis.

In the early stages of the disease, the percussion sound during examination of the lungs is not changed. With severe lymphadenopathy of the mediastinum in thin people, you can find dullness of the percussion sound over the dilated mediastinum. With the development of emphysema of the lungs, the percussion sound becomes boxy. There are no specific auscultatory signs in sarcoidosis. Possibly weakened vesicular or hard breathing, wheezing is not typical. Blood pressure usually does not change, even in patients with elevated ACE levels.

Extrapulmonary manifestations of sarcoidosis.

Musculoskeletal changes in sarcoidosis (occurring in 50-80%) are most often manifested by arthritis of the ankle joints, myopathies. Sarcoidosis of the eyes is noted in about 25% of patients, of which 75% have anterior uveitis, 25-35% have posterior uveitis, and infiltration of the conjunctiva and lacrimal glands is possible. Sarcoidosis of the eye can lead to blindness. Neurosarcoidosis affects less than 5% of patients. The disease can manifest itself as cranial nerve palsy, polyneuropathies, meningitis, epileptiform seizures, masses in the brain, pituitary-hypothalamic syndrome, and memory impairment. Heart damage (less than 5%), for example in the form of arrhythmias, blockades, pose a threat to the patient's life (50% of deaths from sarcoidosis are associated with heart damage). Sarcoidosis of the larynx (more often its upper part) is manifested by hoarseness, coughing, dysphagia and increased breathing due to obstruction of the upper airways. Renal involvement in sarcoidosis is most commonly associated with nephrolithiasis. In sarcoidosis, characteristic-syndromes:

Löfgren's syndrome - fever, bilateral lymphadenopathy of the roots of the lungs, polyarthralgia and erythema nodosum - a good prognostic sign of the course sarcoidosis.

Hefordt-Waldenstrom syndrome is diagnosed with fever, parotid lymph node enlargement, anterior uveitis, and facial paralysis. nerve.

Diagnostics.

In the general analysis of blood: lymphopenia, monocytosis, moderate eosinophilia, increased ESR. With biochemical blood tests, it is possible to detect hypercalpiemia, hypercalciuria, an increase in the ACE content, and hyperglobulinemia. For painsarcoidosis is characterized by a decrease in the number of T-lymphocytes and an increase in B-lymphocytes and immunoglobulins Ig G and Ig M in the blood, a positive Kveim test is a reaction to intradermal administration of a suspension of sarcoid tissue. Serologic tests with tuberculin in patients with sarcoidosis are negative. An increase in the content of serum lysozyme is possible.

In modern international practice, radiological signs of sarcoidosis of the chest organs are distinguished into 5 stages: stage 0 - there are no changes;
stage I - thoracic lymphadenopathy, the lung parenchyma is not changed; stageII- lymphadenopathy of the roots of the lungs and mediastinum in combination with changes in the lung parenchyma;
stage III - the pulmonary parenchyma is changed, lymphadenopathy of the roots of the lungs and mediastinum is absent;
stage IV - irreversible pulmonary fibrosis;

RKT- a highly informative method for diagnosing sarcoidosis and monitoring its course. Focal opacity of the frosted glass type ("alveolar sarcoidosis") may be the only manifestation of the disease in 7% of patients, which corresponds to early alveolarstages of the process.

Bronchoscopy- important in the initial diagnosis of sarcoidosis. During bronchox, bronchoalveolar lavage can be performed to exclude granuloma:infectious nature.

Biopsy the mucous membrane of the bronchi, lymph node, lung allows you to detect non-zeinfected granulomas, consisting of epithelioid cells and single giant of Pirogov-Langhans cells.

ECG - an important component in the examination of patients with sarcoidosis, since late diagnosed myocardial sarcoidosis can cause arrhythmias and sudden stops;hearts.

Scintigraphy with gallium allows you to determine the localization of sarcoidosis - in the mediastinal lymph nodes, the parenchyma of the lungs, submandibular and parotid glands.

Kidney ultrasound indicated for the timely detection of nephrolithiasis.

Differential diagnosis.

In the presence of bilateral lymphadenopathy on a chest x-ray, dio: renal diagnosis is carried out between sarcoidosis and lymphoma, tuberculosis, grie5 infections, lung cancer and eosinophilic granulomas.

Treatment.

In stage 1-II sarcoidosis, 60-70% of patients have the likelihood of spontaneous stable remission, while the use of systemic glucocorticoids can be accompanied by frequent subsequent relapses, therefore, after the detection of the disease, it is recommendedobservation is carried out for 2-6 months.

The most commonly used glucocorticoids. In stage I-II sarcoidosis, especially with verified obstructive syndrome, experience has been gained with the use of budesonide, flunisolide. In severe cases, systemic use of glucocorticoids is indicated. There are several treatment regimens with prednisone. The first is characterized by long-term daily intake of prednisolone at a dose of 20-40 mg. per day for 3-4 months. Then the doses are gradually reduced, prescribing 15-10 mg per day for another 3-4 months, and then a maintenance dose of 5-10 mt per day is used for 4-6 months. The duration of treatment with steroid drugs lasts 6-8 months. and more depending on the clinicaleffect.

Another regimen involves intermittent use of prednisolone (every 1-2 days). Treatment also begins with a dose of 20-40 mg per day, gradually decreasing it. Intermittent treatment is prescribed for patients with poor tolerance to steroid drugs, whenadverse reactions.

With severe violations of the function of external respiration, especially obstructive, with sarcoidosis of the bronchi, it is possible to use pulse therapy with prednisolone (10-15 mg / kg methyl prednisolone IV drip, every other day, 3-5 times) or flunisolide (1 mg 3 times a day for 3-6 months) followed by treatment low doses.

If hormones are ineffective or poorly tolerated by patients, presocil, chloroquine, methotrexate are prescribed instead. ACTH is also recommended for the treatment of sarcoidosis,colchicine, vitamin E, intravenous sodium thiosulfate.

Transplantation.

Nowadays, patients with terminal stages of sarcoidosis, with the ineffectiveness of drug therapy, are transplanted lungs, as well as heart and lungs, liver and kidneys. The immunosuppressive therapy carried out at the same time is the treatment of sarcoidosis. The three-year survival rate is 70%, the five-year survival rate is 56%. However, it is possiblerelapse of the disease in the transplanted lung.

Forecast fromarcoidosis is very variable and depends, in particular, on the stage of the disease.

Clinical examination. Constant observation by a pulmonologist is necessary (visits are not less often once every 6 months).

ABSCESS AND LUNG GANGREN

Lung abscess and gangrene, combined by the terms "acute pulmonary suppuration", "acute infectious destruction of the lungs", "destructive pneumonitis", etc., are, as a rule, severe, often life-threatening pathological conditions characterized by a fairly massive necrosis and subsequent purulent or putrefactive decay (destruction) of lung tissue as a result ofactions of certain

infectious pathogens.

It is advisable to distinguish not two, but three main clinical and morphological forms of infectious and destructive processes in the lung tissue: abscess, gangrenous abscess and gangrene of the lung. A lung abscess is understood as the formation of a more or less limited cavity in the lung tissue as a result of its necrosis and purulent fusion Lung gangrene is a much more severe pathological condition with massive necrosis and ichorous decay, rapid purulent fusion and rejection of lung tissue without a tendency to clearly delineate from its viable parts. Gangrenous abscess, as a rule, is less extensive and more prone to delineation than with widespread gangrene, necrosis of lung tissue, during the demarcation of which a cavity is formed with parietal or free-lying sequesterslung tissue and a tendency to constantly cleanse.

Prevalence. Lung abscesses 3-5 times more often than in the general population are observed in men aged 20-50 years. Over the past 40 years, the frequency of lung abscesses has decreased 10 times, while the mortality rate among patients has decreased only by 5-10% and is 4-7%. When aspiration of liquids containing gram-negative microflora, mortality can reach 20% or more, especially if the reaction of the liquidsour.

Most often, deaths of lung abscess are associated with seeding of Pseudomonas aeruginosa, Staphylococcus aureus, and Klebsiella pneumoniae.

Classification of infectious lung destruction (N.V. Putov, 1984)

I. By etiology (depending on the type of microbial pathogen).
II. By pathogenesis:
1. Bronchogenic (including aspiration).
2. Hematogenous (including embolic).
3. Traumatic.
4. Lymphogenous

III. By the type of pathological process:
1. The abscess is purulent.
2. Gangrenous abscess.
3. Lung gangrene.

IV. In relation to the anatomical elements of the lung:
1. Peripheral.
2. Central.

V. By the prevalence of the lesion:
1. With segment lesions
2. With the defeat of the lobe
3. With more than one lobe or all of the lung involved
4. Single
5. Plural
6. Unilateral
7. Bilateral

Vi. According to the
severity of the course:

1.Lungs
2. Medium severity
3. Heavy
Vii. Depending on the absence or presence of
complications;1. Uncomplicated
2. Complicated, including pyopneumothorax, pleural empyema, pulmonary bleeding, sepsis.

Etiology. The cause of the development of infectious destruction of the lungs can be practically any microorganisms or their associations.

Anaerobes. Among the anaerobic microflora, the species Peptostrepto-coccus (anaerobic gram-negative cocci), Fusobacterium nucleatum, Fusobacterium necrophorum, Porphyromonas and Prcvotella melaninogcnica species (formally related to genus Bacteriodes).

Aerobes. Among aerobes, the most common lung abscess is caused by Staphylococcus aureus, Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus pyogenes, Pseudomonas pseudomallei, Haemophilus influenzae (especially type B), Legionella pneumophila, Nocardia asteroides, Actinomyces species, and rarely pneumococci.

The simplest. The destruction and formation of an abscess can be caused by the protozoa Paragonimus nestermani and Entamoeba histolytica, as well as mycobacteria.

Risk factors... For the development of destructive pneumonitis, factors are necessary that reduce the defenses of the human body and create conditions for the entry of pathogenic microflora into the respiratory tract or aspiration. These factors include alcoholism, drug overdose, surgical interventions usinggeneral anesthesia, prolonged vomiting, neurological disorders (disorders of cerebral circulation, myasthenia gravis, amyotrophic lateral sclerosis, etc.), epilepsy, neoplasms in the lungs, foreign bodies in the respiratory tract, diabetes mellitus, immunodeficiency states.

Pathogenesis. The causative agents of infectious destruction of the lungs penetrate the lung parenchyma through the respiratory tract, less often hematogenous, lymphogenous, by spreading from neighboring organs and tissues. With transbronchial infection, the source of microflora is the oral cavity and nasopharynx. An important role is played by aspiration (microaspiration) of infected mucus and saliva from the nasopharynx, as well as gastric contents. In addition, lung abscesses can occur with closed injuries (bruises, compression, concussion) and penetrating chest wounds. With an abscess, initially there is a limited inflammatory infiltration with purulent fusion of the lung tissue and the formation of a decay cavity surrounded by a granulation shaft.

Subsequently (after 2-3 weeks), a purulent focus breaks out in the bronchus; with good drainage, the walls of the cavity collapse with the formation of a scar or areapneumosclerosis.

With gangrene of the lung, after a short period of inflammatory infiltration due to exposure to the waste products of microflora, vascular thrombosis develops extensive necrosis of the lung tissue without clear boundaries. In the necrotic tissue, many foci of decay are formed, which are often drained through the bronchus. The most important pathogenetic factor is also a decrease in the function of general immunity and local bronchopulmonary protection.

Clinic. The process of abscess formation lasts up to 10-12 days, during which the clinical picture of the disease is most often associated with the course of pneumonia. In the initial period of the disease, patients note general malaise, weakness, chills, cough with scanty sputum, sometimes hemoptysis and chest pain. Body temperature is usually high. Even with small processes, shortness of breath is observed due to intoxication. With gangrene of the lungs, these signs are more pronounced. Sudden discharge of a large amount (with a full mouth) of fetid sputum is a sign of a breakthrough of an abscess in the bronchus. An improvement in the patient's condition, a decrease in body temperature are characteristic. With gangrene of the lung, sputum is putrid in nature. The average daily amount of sputum with an abscess is 200-500 ml, but it can increase to 1000 ml or more with gangrene.

Objective research. Before the abscess breaks out, slight cyanosis of the face and extremities can be detected. With extensive damage and involvement of the pleura in the process, the lag of the affected half of the chest in the act of breathing is visually determined. The patient takes a forced position on the sore side. In chronic abscess, the fingers take the form of "drumsticks", signs of right ventricular failure are formed. Tachypnea and tachycardia are characteristic. The first period lasts from 4 to 12 days. The transition to the second period - the beginning of the emptying of the destruction cavities - is accompanied in typical cases by an improvement in the patient's condition.

Palpation allows you to detect soreness along the intercostal space on the diseased side, which indicates the involvement of the pleura and the intercostal neurovascular bundle. With a subpleural location of the abscess, the voice tremor is enhanced. When a large abscess is emptied, it may become weakened.

Percussion. In the initial phase on the side of the lesion, percussion sound can be somewhat shortened. With a deep location of the abscess, the percussion sound does not change. At the first stage of the course of destructive pneumonitis, the physical picture is similar to that in confluent pneumonia. At the second stage, the intensity and area of the shortening of the percussion sound decrease. Superficially located large empty abscesses are accompanied by a tympanic percussion sound.

Auscultation in the first period of the abscess, it reveals hard breathing, sometimes bronchial and weakened breathing, against which dry or moist rales are possible. In some cases, wheezing may not be present. When the picture of pneumonia prevails, crepitus is heard. After opening the abscess, you can hear moist rales of various calibers, bronchial and, quite rarely, amphoric breathing.

Diagnostics.

X-ray examination of the chest organs in frontal and lateral projections is an obligatory component of the diagnosis of a lung abscess, which is more often localized in the posterior segment of the upper lobe (S2) and the upper segment of the lower lobe (S6), as well as in the segments S8, S9, S10. In the first phase, during X-ray examination, intense infiltrative darkening of various lengths (from several segments to a lobe or more) is determined. In the second phase, against the background of decreasing infiltration, the density of a rounded shape with a fairly even inner contour and a horizontal liquid level begins to be determined. Sometimes there are several of these cavities. With good drainage, the level is determined only at the bottom of the cavity, andthen disappears altogether. The presence of effusion in the pleural cavity indicates the involvement of the pleura in the process. In a chronic abscess, the cavity has dense walls, surrounded by an infiltration zone. Sequesters can be seen in the cavity of a chronic abscess.

With gangrene of the lung after a breakthrough in the bronchus, multiple enlightenments of a directed form (sometimes with fluid levels) are determined against the background of massive darkening.

X-ray computed tomography(RCT) allows you to accurately determine the localization of the cavity, the presence of even a small amount of fluid in it, sequestration, to assess the involvement of the pleura. With gangrene of the lung, RCT provides more reliable information about sequestration.

Examination of the function of external respiration (FVD). In patients with lung abscess reveal mixed or retrictive ventilation disorders.

Laboratory diagnostics... A general blood test reveals an neutrophilic leukocytosis with a shift of the leukocyte formula to the left, an increase in ESR, and hyperalbuminemia. In severe cases, iron deficiency anemia and moderate albuminuria are observed. Leukocytes may appear in the urine. When standing, the phlegm stratifies: the upper layer is a frothy serous fluid, the middle layer is liquid, contains many leukocytes, erythrocytes, bacteria (the most significant in volume), the lower layer is purulent. Microscopy of sputum detects neutrophils, various types of bacteria.

Bronchoscopyis diagnostic and therapeutic in nature. Aspiration allows you to obtain material for the determination of microflora and its sensitivity to antibiotics.

Differential diagnosis. Differential diagnosis of an abscess is carried out primarily with cavities of various natures, which can be detected on radiographs and during CT. These include tuberculosis, disintegrating lung tumors, suppurating cysts, actinomycosis, etc.

In the differential diagnosis of lung abscess with tuberculous cavities take into account the presence of contact with bacilli-releasing agents. On average, tuberculous

lesions flow more torpidly, with a less pronounced temperature reaction, and at the same time with greater sweating. The daily amount of sputum in tuberculosis patients rarely exceeds 50-100 ml.

Tuberculous cavities are more often located in the S1, S2 and S6 segments. A horizontal liquid level is rarely observed in them. I consider the appearance of shedding foci in the lungs typical for tuberculosis. The destructive forms of tuberculosis are usually accompanied by bacterial excretion, detected by smear microscopy during Ziehl-Nielsen staining, bacteriological examination, and in highly specialized institutions - with the DNA-polymer method for identifying mycobacteria. In doubtful cases, bronchoscopy and bacteriological examination of the contents of the bronchi should be performed.

Empyema of the pleura

A parietal abscess is differentiated from pleural empyema. Conducting CT allows you to accurately determine the topography of the cavity formation, its belonging to the parenchyma of the lungs or pleural cavity.

Lung cancer

Of great practical importance is the differential diagnosis of abscess and cavity form of peripheral lung cancer. The age of the patient (over 50), the absence of an acute period of the disease, the scarcity of sputum, and, if present, the absence of odor, testify in favor of the tumor. During radiation examination, the tumor is characterized by the presence of a clear outer contour with its bumpy outlines. The inner contour of the cavity, in contrast to the abscess, is indistinct; there is little liquid inside the cavity, and more often it is absent. When cytological examination of sputum or bronchial contents, or in a biopsy material, tumor cells are found.

Lung cysts

Festering congenital lung cysts are rarely observed. Suppuration in a cyst usually proceeds without high body temperature and intoxication, there is little sputum, it is mucopurulent. On the roentgenogram, a festering cyst looks like a thin-walled round or oval formation with a horizontal fluid level without perifocal infiltration

Treatment

Intensive treatment is carried out simultaneously in several main directions-yam:

1) maintenance and restoration of the general condition, and correction of the disturbed homeostasis;

2) ensuring optimal drainage of destruction foci in the lung (and

pleura);

3) suppression of microorganisms - pathogens of the infectious process (including viruses);

1) correction of the immunological reactivity of patients.

Patients with lung abscess require intensive treatment in a hospital setting. Patients are provided with a diet with an energy value of up to 3000 kcal / day, a high protein content (110-120 g / day) and a moderate restriction of fat (80-90 g / day). They increase the amount of foods rich in vitamins A, C, group B (decoctions of wheat bran, rose hips, liver, yeast, fresh fruits and vegetables, juices), calcium, phosphorus, copper, zinc salts. Limit table salt to 6-8 g / day, liquid.

Conservative therapy lung abscess is based on the use of antibacterial agents up to clinical and radiological recovery (often 6-8 weeks). The choice of the drug is based on the results of bacteriological examination of sputum, blood and determination of the sensitivity of microorganisms to antibiotics (Table 3).

Table 3.

Antibiotic prescription after microbiological identification pathogen

Microbiological data analysis	**Antibacterial agents**
Staphylococci	Aminopenicillins with inhibitors (β-lactamase Cephalosporins of the first and second generation. Aminoglycosides Fluoroquinolones. Vancomycin (with
Haemophilus influenzae	Aminopenicillins with β-lactamase inhibitors. New macrolides (claritomycin, azithromycin).
Klebsiella pneumoniae	First and second generation cephalosporins Aminoglycosides. Fluoroquinolones.
Pseudomonas aeruginosa	Third generation cephalosporins. AminoglycosidesFluoroquinolones.
Proteus vulgaris	Second and third generation cephalosporins
Escherichia coli	Aminoglycosides. Fluoroquinolones.
Legionella pneumophila	Macrolides. Fluoroquinolones
Mycoplasma pneumoniae. Clamydia pneumoniae	Macrolides Doxycycline

Intravenous administration of antibacterial agents is most effective. PreIntravenous penicillin or clindamycin are considered paras of choice. High doses of intravenous penicillin were effective in 95% of cases. We recommend benzyl penicillin sodium, 1-2 million IU IV every 4 hours until the patient's condition improves. The growth of penicillin-resistant strains makes it possible to recommend clindamycin 600 mg IV every 6-8 hours, then 300 mg orally every 6 hours for weeks. With microbiological identification of the pathogen, it is necessary to correct the etiotropic therapy in accordance with the identified pathogen and its sensitivity.

Severe violations of water-electrolyte and protein balance, intoxication, exhaustion and progressive anemia require massive infusion therapy.

It is advisable to maintain the energy balance by introducing concentrated glucose solutions with the addition of an appropriate amount of insulin; I.S. Kolesnikov and sotr. (1983) proposed the following therapeutic composition: for 1 liter of 40% glucose solution, 5 g of potassium chloride, 2 g of sodium chloride, 1 g of calcium chloride, 0.5 g of magnesium chloride and 8 IU of insulin. The addition of insulin at a dose of 1 U per 3-4 g of glucose not only facilitated the utilization of the latter, but also facilitates the penetration of fecal ions into the cells, normalizing the disturbed electrolyte balance. This solution is poured in a volume of 1-3 l / s. Protein losses are replenished by the infusion of protein hydrolysates - aminocrovin (clarified). infusoalin, as well as solutions of amino acids - polyamine, panamish amikon, etc. In case of severe hypoalbuminemia, an infusion of human albumin (100 ml 2 times a week) is useful. The assimilation of parenterally administered protein is improved with the simultaneous use of apabolizing hormones (regabol-nerabol). For detoxification, low molecular weight drugs such as rheopolyglucin and hemodez are used. Severe anemia makes it necessary to transfuse fresh donor blood or erythrocyte mass, 250-500 ml 1-2 times a week.

In order to restore and stimulate the factors of the body's immunological defense, the following are used: measles gamma globulin, antistaphylococcal gamma globulin, methyluracil, T-activin (administered subcutaneously at night 1 time per day for 40-100 m, for 5-7 days). Thiamine is injected intramuscularly in 0.25% novocaine solution at 10-30 mg of the drug for 5-20 days.

Expectorants are prescribed. To facilitate the outflow from a purulent focus in the lung, it is recommended to use the so-called postural drainage.

Surgical treatment is indicated in about 10% of cases with ineffective antibiotic therapy, pulmonary bleeding, inability to exclude lung cancer, with an abscess larger than 6 cm, with an abscess breaking into the pleural cavity with the development of empyema, as well as with chronic abscesses.

Outcomes of acute infectious lung destruction

Complete recovery is noted in 25-40% of patients (observed with purulent abscesses); clinical recovery - in 35-50%, transition to a chronic form - in 15-20% (observed with gangrenous abscesses) and death - in 5-10%.

Widespread gangrene of the lungs gives a mortality rate of at least 40%.

Follow-up after discharge from hospital

Follow-up after discharge from the hospital is carried out by a pulmonologist at the place of residence. 3 months after clinical recovery, it is necessary to conduct a concontrol X-ray examination.

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