

**MINISTRY OF HEALTH CARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

"Approved"

at the meeting of the internal
medicine №1 department

The Head of the Department

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**GUIDELINES
FOR the STUDENTS
INDEPENDENT WORK
FOR THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Basics of Internal Medicine
<i>Content module</i>	Fundamentals of diagnostics, treatment and prevention of respiratory diseases
<i>Study subject</i>	Pleurisy and pleural effusion. Infectious and destructive lung disease. Pulmonary insufficiency
<i>Course</i>	IV
<i>Faculty</i>	of foreign students training

Poltava 2016.

1. Relevance of the topic: pleurisy syndrome is quite common in the clinic of internal diseases and its differential diagnosis is a complicated pulmonology problem. Exudates pleural cavity is a sign of such common diseases as the hospital and community-acquired pneumonia, tuberculosis, systemic connective tissue diseases, cancer, and others. The study of this subject is necessary for the differential diagnosis of many diseases accompanied by syndrome of fluid accumulation in the pleural cavity.

2. The main goal: To be able to assess the typical clinical picture of pleurisy, to determine tactics of treatment and prophylaxis.

The student should know:

1. Classification of pleurisy
2. Etiological factors that contribute to pleurisy.
3. Clinical features of exudative and adhesive pleurisy.
4. Features of the X-ray diagnosis of exudative and adhesive pleurisy.
5. The differential diagnostic differences exudate and transudata.
6. Clinical pharmacology of antibiotics and anti-inflammatories.

The student should be able to:

1. Choose from complaints and medical history information reflecting the presence of fluid in the pleural cavity.
2. Identify the characteristics of exudative and dry (fibrinous) pleurisy an objective examination of the patient.
3. Compose a plan of laboratory and instrumental tests and interpret their results.
4. According to the biochemical and cytological examination of the pleural cavity effusion distinguish exudate from transudate.
5. Determine the principles of treatment of patients with different origin pleurisy.

3. Basic knowledge, abilities, skills (interdisciplinary integration)

Discipline	To know	To be able to
Anatomy	The structure of the bronchopulmonary apparatus, blood supply, innervation	
Histology	The structure of the wall of the trachea, bronchi, alveoli in health and disease	
Microbiology	Properties pathogens that are etymological origin with pleurisy	
Regional anatomy	Interposition of the chest organs	
Normal physiology	Indicators of respiratory function, their value	To determine the function of external respiration
morbid anatomy	Changes in the structure of the wall of bronchopulmonary tissue in pleurisy	
Pathological physiology	Indicators pneumotachometry, spirometry depending on the type of ventilation insufficiency	Analyze the performance of external respiration
Radiology	Radiographic changes with pleurisy	Analyze the radiological picture pleurisy
Pharmacology	The mechanism of action, indications and side effects of antibacterial drugs	Prescriptions

Propaedeutic therapy	Pleurisy symptomatology and its complications	Perform a physical examination of the patient, analyze the clinical and laboratory data of the patient
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Theoretical questions to the lesson::

1. Definition of pleurisy
2. Classification.
3. The etiology of infectious and non-infectious pleurisy.
4. Pathogenic mechanisms leading to accumulation of fluid in the pleural cavity (inflammatory and non-inflammatory nature).
5. Clinical features of the course, diagnosis and differential diagnosis of exudative pleurisy in pneumonia, tuberculosis, systemic connective tissue diseases, cancer, pleural empyema.
6. Clinical features of the course, diagnosis and differential diagnosis of dry (fibrinous pleurisy).
7. Treatment of pleurisy different genesis.

Topic content.

Definition. Pleurisy (pleuritis) - an inflammation of the pleura with fibrinous layers formation on its surface or with fluid accumulation in the pleural cavity.

Epidemiology. In many countries the most common cause of exudative pleurisy is tuberculosis, which usually occurs on the background of pulmonary tuberculosis.

The most common cause of pleural effusion identify the population of developed countries is left ventricular congestive heart failure, malignancy, infections, pulmonary embolism.

Etiology. Pleurisy mainly occurs as complication of the pathological process of the lungs, mediastinum, diaphragm, thorax (pneumonia, tuberculosis, infection destructive lung diseases, malignancies, systemic connective tissue diseases).

Etiological factors can be both of infectious and non-infectious nature. Infectious etiological agents includes bacterial pathogens such as *Streptococcus pneumoniae*, *Klebsiella pneumoniae*, *Staphylococcus aureus*, *Haemophilus influenzae*; *Mycobacterium tuberculosis*; fungi; viruses; protozoa; vermin.

Aseptic pleurisy occurring malignant tumors of various origins, pulmonary thrombosis with development of pulmonary infarction, Dressler's syndrome, acute pancreatitis, chronic renal failure, nephrotic syndrome, systemic connective tissue diseases such as systemic lupus erythematosus, dermatomyositis, scleroderma, rheumatoid arthritis; systemic vasculitis, hemorrhagic diathesis, injuries of the chest due to trauma or surgical treatment, drug reactions.

Congestive heart failure, chronic renal failure, liver cirrhosis can cause pleural effusion without inflammation.

Pathogenesis. Physiologically pleural cavity contains a small amount of serous fluid secretion products by parietal pleura, which has the blood supply through the vessels of big blood circulation, fluid is reabsorbed mainly by visceral pleural leaf, blood supply of which is associated with big blood circulation (system of bronchial arteries) and small blood circulation (branches of pulmonary artery). Significantly higher hydrostatic pressure in the capillaries of the parietal leaf than visceral difference oncotic pressure provide a constant fluid motion. Imbalance between the rate of secretion and reabsorption of pleural fluid is derived from the hydrostatic and oncotic pressure in the capillaries of the pleural layers, impaired vascular permeability may cause excessive accumulation of fluid in the pleural cavity.

Penetration infectious agent in the pleural cavity can occur by direct contact (infection in trauma, after chest or abdominal surgical treatment), infection lesions of the lung tissue associated with pneumonia, abscesses, gangrene, tuberculosis; hematogenous or lymphogenous ways. Increased permeability of blood vessels, lymphatic capillaries dilate leads to development of the pleura edematous infiltration with formation of a small amount of inflammatory exudate in the pleural cavity. In the case of sufficient lymphatic drainage, performed mainly by the vessels

of visceral pleural leaf, the fluid is reabsorbed and covering of fibrin on the surface of pleural leaves contribute to the dry pleurisy development. In the case of considerable intensity of the inflammatory process with a lot of fluid or at lower lymph outflow pleural effusion should be formed.

Underlying disease pathogenesis causes the development of noninfectious etiology pleurisy. In the case of trauma in the area of injury limited hemothorax occurs. In the malignant tumors metastasis to the pleura increased vascular permeability, fluid flow is reduced, there is a disorder of lymph outflow due to metastases in the lymph nodes, circulatory problems for metastatic lesions of vessels. Autoimmune mechanisms are crucial in the accumulation of pleural effusion in systemic connective tissue diseases, systemic vasculitis, Dressler's syndrome. In acute pancreatitis pleurisy as a complication can occur due to pathogenic influence of pancreatic enzymes fall into the pleural cavity through the diaphragm lymphogenous way.

Pathomorphology. Pathomorphological changes characterized by dilatation of the lymph capillaries, increased vascular permeability, pleural layers edema, infiltration of subpleural layers, deposition of fibrin on the pleural leaves surface, accumulation of pleural effusion depending on the pathophysiological mechanisms of formation - exudate or transudate. Sero-fibrinous character fluid appears in pneumonia, tuberculosis (sometimes hemorrhagic); hemorrhagic character - malignant tumors, lung infarction; purulent - with abscess and other infectious destructive lung diseases.

Classification. The nature of the process are distinguished dry (fibrinous) and exudative pleurisy or pleural effusion, but in most cases there are the subsequent stages of common pathological process. The affection described as purulent pleurisy or pleural empyema if the pus formation in pleural cavity occurs. According to course - acute, subacute, chronic.

Clinic. The leading complaints of the patient's with dry pleurisy are intense stabbing chest pain, usually on one side, associated with the act of breathing, aggravated during cough and during pressing on the corresponding area of the chest, accompanied by fever, general weakness.

Objectively determined the lag of affected half of the chests in breathing act; pain on palpation detected in several intercostal spaces, not in one, amplifies at an inclination to a healthy side, reduced while limiting the mobility of the chest, sometimes pleural friction rub determined by palpation and during auscultation over the lesions area.

In exudative pleurisy leading syndrome associated with the presence of pleural effusion is shortness of breath, the severity of which is connected with the volume and speed of the fluid accumulation rather than hypoxia. Not very frequent symptom of pleural effusion is light nonproductive cough. Other clinical signs appears depending on the underlying disease, which caused pleurisy.

Severe cough appearance with purulent or bloody sputum may indicate pneumonia or endobronchial lesion as the cause of the pleural effusion presence. Constant invariable chest pain can be a sign of chest infestation by bronchogenic carcinoma or malignant mesothelioma. Severe intoxication syndrome with fever, weight loss and inanition observed in empyema.

Objectively asymmetry of the chest manifested the presence of pleural effusion, vocal fremitus decreased or defined over the area of effusion, dull percussion sound determined, decreasing or absence of breathing sounds during auscultation. The shift of the mediastinum to the opposite side if effusion volume over 1000 ml, or in the direction of pleural effusion should be observed in the endobronchial lobar bronchus obstruction more often associated with malignancy process or with the presence of a foreign body.

The evacuation of pleural effusion partially reduces clinical symptoms and makes it possible to determine the cause at the repeated X-ray examinations.

Diagnosis. At infectious inflammatory origin of pleurisy in general blood test revealed nonspecific inflammatory syndrome signs - neutrophilic leukocytosis with a shift to the left, increased ESR. The changes in the biochemical blood analysis characterized by increasing levels of acute-phase reactants (C-reactive protein, haptoglobin, sialic acids seromucoid etc.), dysproteinemia due to hypoalbuminemia and hyper alpha-1- and alpha-2-globulinemia.

Aseptic pleural effusion accompanied by laboratory changes which characterised the underlying diseases.

X-ray examination has decisive importance. Dry pleurisy accompanied by the signs of diaphragm cupula high standing with limitation of diaphragm mobility at the affected side, thickening of the parietal pleura with regular monotonic lung fields transparent decreasing above the diaphragm and at costodiaphragmatic recess with vascular pattern conservation.

X-ray study in patients with pleural effusion should be performed before and immediately after pleural puncture (thoracentesis) to determine the possible nature of the pathological process that could be the cause of the pleural effusion. X-ray picture depends on the amount of liquid, location, availability of free or encysted pleurisy. Representative is the presence of dense homogeneous darkening at the bottom and the flank of the chest, obliteration of acute costodiaphragmatic angle with the upper level of liquid placed obliquely from top to down and from the outside to inwards, which should be replaced with body position changes.

More sensitive methods are ultrasound and computed tomography of the chest. CT with contrast allows to differentiate malignant pleural lesions in which nodular thickening of the parietal pleura or total pleural thickening should be determine.

The presence of free fluid can be detected in patients with pneumonia at lateral decubitus radiography (radiographic study in lateroposition of the patient at the affected side), computed tomography of the chest or ultrasound.

The indication for thoracentesis is ascertained free fluid in the pleural cavity more than 10 mm during lateral decubitus radiography. The procedure of thoracentesis is performed with diagnostic and therapeutic purposes.

First of all it is necessary to determine the transudate or exudate nature of effusion.

The leading causes of exudative pleural effusion are malignant tumors (metastasis, pleural mesothelioma), infectious diseases (tuberculosis, bacterial, fungal, viral infections, parasitic), pulmonary embolism, pathology of the digestive system (rupture of the esophagus, diaphragmatic hernia, lesions of the pancreas, subdiaphragmatic abscess), systemic connective tissue diseases and others.

Transudative pleural effusions associated with the left ventricular congestive heart failure, liver cirrhosis, nephrotic syndrome, obstruction of the vena cava superior, myxedema, urinotorax. The necessity for differentiation is determined a circumstance to be taken into consideration that additional diagnostic procedures can help to detect the cause of exudative pleural effusion.

Pleural effusion is considered to be exudate if: pleural fluid protein / serum protein > 0,5; pleural fluid LDH / serum LDH > 0,6; pleural fluid LDH more than two thirds from the normal upper limit in serum. But according to these criteria pleural effusion in patients with congestive heart failure, who has to intake massive diuretic therapy for a long time may mistakenly recognized such as exudate. More specific is the definition of the difference between protein levels in serum and pleural effusion, at the limit gradient > 31 g / L (3.1 g / dL), pleural effusion is a transudate and suggested criteria can be ignored.

In the case of exudative character pleural effusion it is necessary to determine the glucose level, to conduct the microbiological studies, differential cell count, cytology.

Pleural fluid lymphocytosis suggests tuberculosis, lymphoma, sarcoidosis, chronic rheumatoid pleurisy, chylitorax. Pleural fluid eosinophilia most often is caused by presence of blood or air in the pleural space but it may be associated with nonmalignant diseases also including parasites, fungal infection, medication reactions.

The presence of localized effusion with a pH <7.2, glucose levels <3.3 mmol / L, Gram-positive culture and purulent nature requiring a more invasive interventions, such as pleural cavity drainage.

Differential diagnosis. The differential diagnosis should be conducted with pathological conditions with similar clinical symptoms.

Chest pain may be a sign of **myositis**, **intercostal neuralgia**, but unlike to pleurisy the pain will be associated with the movements, amplifies at an inclination to the side of lesion. Intercostal nerve exit painful points determined at palpation; the pleural friction rub noise is absent.

In **acute coronary syndrome** or **angina pectoris** attack the complaints and medical history, ECG changes (ST-segment elevation, depression, arrhythmias and conduction disorders), positive functional exercise tolerance or pharmacological tests can indicate the presence of coronary **ischemic heart disease**.

Chest pain that is irradiated at the anterior abdominal wall in a diaphragmatic dry pleurisy location must be differentiated with "**acute abdomen**", acute pancreatitis, acute cholecystitis, acute appendicitis, perforation of stomach ulcers.

In many countries the most common cause of exudative pleurisy is **tuberculosis**. The most frequent signs in this group of patients are fever, weight loss, shortness of breath, constant cough, pleuritic chest pain. The diagnosis verified by the results of research methods such as pleural effusion lymphocytosis, high level of tuberculosis specific markers or receiving Mycobacterium tuberculosis culture in pleural effusion, target biopsy, thoracoscopy results.

The most common cause of pleural effusion identify the population of developed countries is left ventricular **congestive heart failure**. Usually adequate etiopathogenetic treatment can be effective. Diagnostic thoracentesis should be indicated to confirm the presence of transudate appointed only if the process is not bilateral, accompanied by fever or if the patient has pleuritic chest pain.

Among the main causes of exudative pleural effusion **malignant tumors (lung carcinoma, breast carcinoma, lymphoma)** are detects. Significant breathlessness is primarily disturbs the patients. The severity of dyspnea is not depends on the amount of fluid in the pleural cavity. The diagnosis is confirmed by cytological study of pleural contents. In the case of a negative cytological study result for the purpose of malignant tumor invalidation thoracoscopy with abrasion and subsequent histological examination of the material obtained.

Pleural mesothelioma develops from mesothelial cells that cover the inner surface of the pleural layers clinically accompanied by chest pain, shortness of breath. Radiologically pleural effusion, diffused pleural thickening, half chest shrinkage are detected . Verification of diagnosis is possible by the results of target biopsy or thoracoscopy procedure.

Pulmonary embolism with infarction, embolization of the pulmonary artery branches can be one of the causes of exudative pleural effusion, the main complaint of the patients is shortness of breath, pleural fluid eosinophilia suggests presence of blood or air in the pleural fluid, a diagnosis confirmed by spiral CT scan or pulmonary vessels angiography.

Parapneumonic pleurisy developing in parallel with the underlying disease and metapneumonic pleurisy that occur in the final phase of pneumonia or other nonspecific lung diseases associated with bacterial pneumonia, infectious destructive processes in the lungs, and can cause pleural **empyema**.

The clinical picture of aerobic bacterial microflora caused pneumonia complicated by pleurisy manifests by high fever, chest pain, sputum discharge, leukocytosis.

Pneumonia caused by anaerobic pathogens more often associated with breathlessness, weight loss, active leukocytosis, insignificant anemia, the anamnestic information about the probability presence of the factors that can promote to pathogen aspiration.

Approximately about 5% of patients with liver cirrhosis and ascites have the signs of significant right-sided pleural effusion, accompanied by severe breathlessness.

A certain percentage of unexplained etiology pleural effusion may be associated with a viral infection, accompanied by corresponding clinical symptoms.

Chylothorax is the accumulation of fluid in the pleural cavity developing most often from lymphatic obstruction by malignancy or due to the thoracic lymphatic duct rupture injury, which can be the result of trauma or surgery treatment of the mediastinum organs pathology. Expressive dyspnea disturbed the patients, X-ray revealed a large amount of fluid in the pleural

cavity, punctate received at thoracentesis is a milky opalescent fluid with high triglycerides level by biochemical analysis suggests a chylothorax. In the absence of the thoracic duct injury lymphangiography and CT mediastinal lymph nodes scan had to be provided to clarify the possible causes.

Hemothorax diagnosed on the basis of obtained during thoracentesis hemorrhagic fluid with hematocrit levels of more than half of this index value in the peripheral blood. Hemothorax can result from trauma, rupture of blood vessels, malignant tumor and often requires tube thorocostomy.

The presence of pleural effusion may be associated with other conditions. The esophagus rupture or pancreas pathology can be detected by increased amylase in pleural fluid.

Subdiaphragmatic intraabdominal abscess may indicate the presence of high fever, chills, identifying the prevalence of polymorphonuclear cells in pleural fluid in the absence of the lung parenchyma pathology. Benign tumors of the ovaries can cause ascites and pleural effusion due to ovarian hyperstimulation syndrome development. Surgery intervention at the coronary vessels can cause bloody left-sided pleural effusion with large number of eosinophils, which is rather easy to treat.

Complication. Complications of empyema – pleural-bronchial fistula and pleural-skin fistula, chest phlegmon, purulent pericarditis, sepsis.

The most likely serious complication of thoracentesis - hypotension results in collapse, hypoxemia, bleeding, infection, air embolism, pneumothorax.

Treatment. Dry pleurisy should be treated in depends on disease etiology.

The treatment of transudative effusions are usually connected with treating the underlying disorder. The management of the patients with exudative pleurisy depends on the underlying etiology of the pleural effusion. But it is necessary to drain large pleural effusions rather exudative or transudative in the case of severe respiratory symptoms causing.

Medications includes antibiotics in the case of bacterial infection, anti-inflammatory drugs, symptomatic treatment such as cough medication.

The treatment of parapneumonic and metapneumonic pleurisy conducted antibiotics considering the sensitivity of microorganisms. Monotherapy conducted using carbapenems (imipenem 0.5 every 6 hours or 1.0 meropenem every 8 hours) inhibitorprotected penicillins (ticarcillin). Combination therapy includes parenteral (IV) administration of cephalosporin (cefotaxime 1-2,0 every 8-12 hours or ceftriaxone 1-2,0 every 24 hours or ceftazidime 2,0 every 8 hours) or fluoroquinolones of III-IV generation (levofloxacin 0, 5 every 12-24 hours or gatifloxacin 0.4 every 24 hours) in combination with antiseptics (0.5 metronidazole every 8 hours) or linkosamid (clindamycin 0,45-0,6 every 6-8 hours).

Treatment schemes of exudative tuberculous pleurisy management and tuberculosis are the same.

Treatment of patients with empyema involves pleural drainage (active aspiration, drip antiseptics, fibrinolytic agents), antibacterial (mainly empirical with the use of broad-spectrum antibiotics or etiological if it is possible to determine selected microflora sensitivity) infusion (to eliminate volemic and water-electrolyte imbalance, protein compensation), detoxification therapy, surgical treatment if it is necessary (thoracostomy, thoracoplasty) .

Repeated thoracentesis procedure should be recommended if the accumulation of fluid recursing. If it is impossible to remove all liquid during therapeutic thoracentesis thoracostomy combination with instillation of fibrinolytic agents (tissue plasminogen activator 10 mg and deoxyribonuclease 5 mg) should be carried or thoracoscopy with breakdown of adhesions performed. In the case of inefficiency of these procedures pleural decortication should be recommended.

Patients with malignant pleural effusion treated mainly symptomatic. Effusion may indicate the presence of metastasis process, most chemotherapeutic treatment of malignant tumors, accompanied by pleural effusion, are not effective. If the main complaint is shortness of breath,

which decreases during therapeutic thoracentesis, little or permanent catheter draining the pleural cavity imposed with the introduction of sclerosing agents (doxycycline at a dose of 500 mg).

The insertions of conducting intrathoracic pleuroabdominal drainage bypass surgery or the applying a ligature at the thoracic duct with percutaneous transabdominal thoracic duct blocking are a treatment of choice for the patients with chylothorax.

Hemothorax is the indication for pleural cavity drainage. If the bleeding continues and exceeds 200 ml per hour thoracoscopy or/and thoracotomy have to be carried out.

Prognosis. The prognosis of dry pleurisy is often favorable, disease duration about 10-20 days, the resorption of fibrinous pleural layers occurs. At unfavorable course pleural sinus obliteration can develop, interpleural adhesions occur or the transition into the pleural effusion can be observed.

Overall prognosis depends on the etiology of the process, accompanied by pleural effusion. If the presence of pleural effusion associated with tumors, the prognosis is rather unfavorable. Timely identified and adequately treated parapneumonic pleural effusion passes the resorption process without significant consequences, but inadequately treated or untreated pleurisy can lead to constrictive fibrosis.

Prevention. Specific prevention of pleurisy does not exist. Timely etiopathogenetic effective treatment of respiratory system pathologies, early detection and treatment of pathological conditions, which may be accompanied by pleural effusion.

Questions for self-control.

1. What is exudate and its differences from transudate?
2. What is the Alice Damuazo' line; Raufus' and Garlyand' triangles?
3. Auscultation picture in exudative and dry pleurisy.
4. Classification of pleurisy.
5. The etiology of infectious and non-infectious pleurisy.
6. The pathogenetic mechanisms leading to accumulation of fluid in the pleural cavity (inflammatory and non-inflammatory nature).
7. Clinical features of the course, diagnosis and differential diagnosis of exudative pleurisy pneumonia, tuberculosis, systemic connective tissue diseases, cancer, pleural empyema.

Tests:

1. The main mechanisms of accumulation of fluid in the pleural cavity following, except one:
 - A. Increased vascular permeability parietal pleura, prizes dit to increased capillary hydrostatic pressure in the visceral and parietal pleura.
 - B. Permeability Increasing protein pleura, and thereby increase the amount of protein in the pleural cavity.
 - C. Reduced oncotic pressure of blood plasma and vnutrishnoplevral-foot pressure (particularly when atelectasis with bronchogenic lung cancer, sarcoidosis).
 - D. Increased oncotic pressure of blood plasma and intrapleural pressure.**
 - E. Violation of the outflow of pleural fluid through the lymph vessels.

2. The fluid - a liquid inflammatory nature, which is formed by:

- A. Infectious inflammation of the pleura.
- B. Accumulation of tumor cells.
- C. Intrathoracic lymph nodes (neoplastic process, sarcoidosis, tuberculosis) in violation of lymphatic drainage.
- D. All options.**

3. Among the most common causative agents of infectious pleurisy are:

- A. Viruses.

- B. Bacteria.**
- C. Fungi.
- D. Rickettsia.
- E. Amoeba.

4. The patient with pleurisy takes a forced position due to the pain, going on:

- A Healthy side..
- B. The affected side.**
- B. Changes in position.
- D. The situation in the bed is not critical.

5. Pleural puncture is performed on such topographical lines:

- A. Midaxillary.
- B. Anterior axillary in the VII intercostal space.
- C. Posterior axillary in the VIII intercostal space on the upper edge of the lower rib.**
- D. Posterior axillary in the VIII intercostal space at the lower edge of the upper rib.
- E. Posterior axillary in the VIII intercostal space, regardless of the rib edge.

6. Pleural fluid normally has a protein content:

- A 0.5-1 g per 100 ml.
- B. 1.0-1.5 g per 100 ml.
- C. 1.5-2.0 g per 100 ml**
- D. 2.5-3.0 g per 100 ml.
- E. 3.0-3.5 g per 100 ml.

Recommended literature

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Infectious and destructive lung disease

1. Relevance of the topic: The value of bronchiectasis and nonspecific infectious and destructive lung disease due to the high risk of complications, including deaths. The prevalence of bronchiectasis data of the population may not be accurate because the most reliable sign of the disease - locally advanced bronchi - diagnosed only by using special methods of investigation. It should be emphasized that in recent decades throughout the world decreased prevalence of bronchiectasis. This is due to a decrease in the number of childhood infections, cases of tuberculosis infection, as well as the expansion of the diagnostic and therapeutic possibilities, the success of drug treatment of inflammatory lung diseases, the implementation of effective antimicrobial therapy.

2. The main goal: To be able to assess the typical clinical picture of bronchiectasis and nonspecific infectious and destructive lung disease, to determine the tactics of treatment and prophylaxis.

Specific goals - to know:

- Etiology, pathogenesis of bronchiectasis and nonspecific infectious and destructive lung diseases;
- Classification, typical clinical picture of bronchiectasis, acute and chronic lung abscess, gangrene of the lungs; differential diagnosis;
- Principles of treatment, primary and secondary prevention, prognosis.

To be able to:

- select from the data history information indicating the presence of bronchiectasis and nonspecific infectious and destructive lung diseases;
- chart the diagnostic search;
- identify signs of bronchiectasis or nonspecific infectious and destructive lung disease with objective research (inspection, palpation, percussion, auscultation)
- analyze and to interpret the value of data changes instrumental methods of research;
- formulate and justify a preliminary diagnosis;
- describe the chest X-ray;
- make a differential diagnosis with diseases that have similar clinical picture;
- develop a treatment strategy;
- know the principles of treatment, rehabilitation and prevention
- diagnose and provide emergency assistance in cases of pulmonary hemorrhage, spontaneous pneumothorax, acute respiratory failure caused by these diseases;
- assess the patient's prognosis and to propose a plan of prevention;
- apply the deontological communication skills with patients.

3. Basic knowledge, abilities, skills (interdisciplinary integration)

Discipline	Skills
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1. Anatomy	1. Know the topography of the respiratory system
2. Physiology	2. To determine and analyze the functional state and of the gas exchange in the lungs
3. Histology	3. To describe and compare the cytological data
4. Pathological physiology	4. To analyze the state of pulmonary ventilation
5. Propedeutics to therapy	5. To interpret the changes in auscultation and percussion picture
6. Radiology	6. To identify the radiographic changes
7. Pharmacology	7. To know drugs effects, be able to write out the prescription.

4. Tasks for independent work.

4.1. The list of key terms, parameters, characteristics which the student needs to learn while preparing for the class:

time	definition
1. Bronchiectasis	- Preseption of the disease, which is based on localized chronic suppurative process irreversibly advanced and functionally inferior bronchus.
2. Lung abscess	- Non-specific inflammation of the lung tissue, accompanied by the occurrence of one or more necrotic cavities.
3. Lung gangrene	- Severe pathological condition characterized by massive necrosis and collapse of the lung tissue with no tendency to clearly delimited.

4.2. Theoretical questions to the lesson:

1. Definition;
2. Etiology, pathogenesis;
3. Classification of,
4. The typical clinical picture;
5. Diagnostic value of instrumental methods of research data changes
6. Differential diagnosis;
7. Principles of differential treatment, the indications for surgical treatment;
8. Complications;
11. Prevention and prognosis.

4.3. Practical tasks that are performed in class:

1. Evaluation of patients (collection of complaints, medical history, physical examination).
2. Analysis of the data and the substantiation of the preliminary diagnosis.
3. Composing of the laboratory and instrumental studies plan to confirm the preliminary diagnosis.
4. Analysis of the results of additional research methods and formulation of the final diagnosis according to the classification.
5. Treatment.
6. Possible complications.
7. Prognosis
8. Prevention

Content of the topic:

Lung abscess

Definition. Lung abscess is a purulent dissolution and necrosis with the formation of a limited cavity in the lung tissue due to infectious destruction.

Etiology. There are no specific pathogens exist. The most common causative agents are: *Staphylococcus aureus*, anaerobic microflora (*Bacteroides flagilis*, *Bacteroides melaninogenicus*, *Fusobacterium nucleatum*, *Peptococcus niger*, *Peptostreptococcus spp.*), Gram-negative aerobic microflora (*Klebsiella pneumonia*, *Escherichia coli*, *Pseudomonas aeruginosa*); with oropharyngeal mucus aspiration - *Fusobacterium necrophorum* and *Peptococcus niger*, *Peptostreptococcus spp.*, gastric content aspiration - *Bacteroides flagilis*, haematogenously-embolic way- *Staphylococcus aureus*.

Epidemiology. The existing information mainly concerns primary pulmonary abscesses. Overall, middle-aged men suffer more often than women. Risk factors are smoking, alcoholism, diabetes, COPD, maxillofacial injuries, hypothermia or presence of predisposition to pathological aspiration.

Pathogenesis. For pathogenesis lung abscesses distinguished at: bronchogenic - aspiration (compared to alcohol, brain injury, cardiospasm, the esophageal achalasia, strictures, diverticula) and inhaled (against the background of inhalation anesthesia, lobar and viral pneumonia complicated by an metapneumonic abscess); nonbronchogenic (haematogenously-embolic, lymphogenous, traumatic, contact, obstructive).

The right lung is most often affected because the right bronchus is shorter and wide.

The primary pulmonary abscess has been arises mainly due to anaerobic bacteria aspiration. Initially, pneumonitis results pulmonary tissue lesions appropriate to gastric acid exposure and at 7-14 days anaerobic bacteria cause necrosis of the parenchyma to form a cavity with subsequent purulent dissolution.

The background of a secondary abscesses, for example, in postobstructive process (foreign body, tumor, etc.), the states accompanied by a decrease host resistance enhancer defenses, including immune system disorders. In the elderly a violation of the bronchi self-cleaning mechanisms that promotes inflammation with subsequent destruction especially in areas of possible presence of atelectasis may be arise. Pulmonary abscess can be a result of septic emboli in tricuspid endocarditis (*Stafilococcus aureus* caused by) or the Lemierre's syndrome, associated with the infection spreads from the pharynx to the neck and carotid capsule containing jugular vein, causing the development of septic thrombophlebitis.

Pathomorphology. The wall of the abscess cavity has a rough surface; pus accumulates in the cavity; inflammatory infiltration is often expressed around the cavity. In acute lung abscess arises the cavity containing pus and detritus, delimited by pyogenic membrane (granulation tissue, granulation bank of cellular infiltration, connective-tissue capsule) from healthy tissue. Microcirculation around the zone of necrosis is saved.

In the case of chronic abscess formation emphysematous alveoli swelling, bronchial deformation with the formation of bronchiectasis, development of pneumosclerosis areas occurs

Classification. Lung abscess may be acute and chronic due to course, primary and secondary, single and multiple due to the number of cavities; bronchogenic (aspirating, inhaling); non-bronchogenic (haematogenously-embolic, lymphogenous, traumatic, contact, obstructive) due to pathogenesis.

Clinical symptoms. Clinical course depends on the period of *acute abscess*. Abscess formation period lasts on average about 7-10 days. It is accompanied by serious condition, hectic fever (up to 39-40 C⁰), chills and profuse sweating, dry cough with chest pain, severe signs of intoxication (headache, weakness, adynamy), general pallor of the skin with local hyperemia on the affected side and forced posture, dyspnea, lags of the affected part of the chest in breathing act, pain on palpation in the region of localization of abscess, blunting percussion sound over the damage focus, impaired vesicular, sometimes bronchial breathing, tachycardia, decreased blood pressure. In the case of deep located abscess, the percussion and auscultative signs is not detected.

The acute abscess burst period begins with the discharge of a large amount of pus вскрытие абсцесса through the respiratory tract. Complete elimination of cavity may take a few (6-8) weeks, sometimes months.

Sputum with an unpleasant odor discharged by "full mouth", upon standing it forms three layers - mucous foamy, serous and purulent with detritus. The general state improved sharply, normal temperature, intoxication signs are reduced, at percussion over the abscess localization light tympanic sound is defined, at auscultation - bronchial (amphoric) breathing with sonorous moist medium and coarse bubbling rales.

If the abscess cavity is well drained, the walls of the cavity are reduced eventually and recovery occurs.

Sometimes residual cavity may be formed or a transition into a **chronic abscess** should be taken place.

Chronic abscesses are formed at the duration not less than 2 months and are characterized by decreasing of acute symptoms subsiding faster while maintaining the cavity with the signs of pneumosclerosis development. Clinically, there comes a relative improvement of the general state with decreasing of intoxication signs, but ongoing cough with an unpleasant odor purulent sputum discharge, sometimes streaked with blood; in the case of sputum discharge delay fever occurs; breathlessness, excessive sweating disturbed; "drumstick"-like deformation of the distal fingers phalanx of the hands, "watch glass"-like nails deformation; dullness of percussion sound over the lesion area, the moist rales occurrence, sometimes the signs of "amphoric" breathing; in CBC - leukocytosis, anemia, ESR accelerated; X-ray - fluid level remains in the abscess cavity surrounded by inflammatory infiltration.

Differential diagnosis. Unlike to **infiltrative tuberculosis** in a phase of disintegration lung abscess is characterized by rapid development, possible connection with pneumonia, severe clinical symptoms, discharge of large amounts of phlegm with an unpleasant odor, significant blood count abnormalities, biochemical parameters, non-specific immunological reactivity, preferably cavities location in the lungs lower lobes, *Mycobacterium tuberculosis* absence in sputum.

Peripheral lung cancer has gradual undistinguished beginning, no connection with pneumonia, subfebrile fever accompanied by the chest pain, expectorated sputum often with blood, cytological atypical cells are present.

Bronchiectasis is accompanied by severe intoxication, shortness of breath, cough with purulent sputum, fever but less pronounced, characteristic radiographic changes (bronchiectasis) defined.

Lung cyst often occurs at a young age, accompanied by subfebrile fever, there are no signs of intoxication, dyspnea, purulent sputum.

Occasionally there is a necessity to conduct a differential diagnosis with aspergilloma, parasitic cavities (*Echinococcus*).

Diagnosis. At the period of abscess formation in the **general blood analysis** significant leukocytosis with leukogram left shift, accelerated ESR observed; in the **biochemical study** - hyperalpha-2 and gamma globulinemia, hyperfibrinogenemia; in **urinalysis** moderate proteinuria is detected.

Radiological the infiltrative eclipse in respective areas defined. After acute abscess burst an isolated cavity filled with fluid, over which the gas bubble determined, have been detected radiologically. **Computed tomography** allows better and earlier to detect the signs of cavitation, even sometimes provide information about the probable cause of the abscess, such as malignant formations, to distinguish peripheral abscess from pleural infection, which is very important for treatment strategy determine. In particular, the detected empyema requires immediate drainage.

It is necessary to conduct **determination of pathogenic organisms in sputum** by noninvasive as possible or, if it is necessary, by invasive (transtracheal aspiration) method.

Bronchoscopy with bronchoalveolar lavage or percutaneous abscess puncture under CT control can be quite high risk of spreading infection, but early detection of the pathogen that caused the secondary abscess development, especially in immunocompromised persons, is crucial for the purpose of effective treatment.

Complication. Pyopneumothorax – abscess burst into the pleural cavity, empyema, pulmonary hemorrhage, subcutaneous and mediastinal emphysema, metastatic brain abscess, sepsis.

Treatment. Etiological antibiotic therapy is prescribed after bacteriological study. Initially empirical antibiotic therapy assigned considering to the ways of infection. Treatment includes measures aimed at reducing the purulent resorptive intoxications, to improve microcirculation, providing drainage through the bronchi or thoracentesis procedure with abscess cavity drainage, correction of immune reactivity.

Oral anaerobes can produce beta-lactamase, so the drug of choice in the patients with primary abscess is clindamycin 600 mg IV every 8 hours until clinical improvement (disappearance of fever), then 300 mg PO every 6 hours. Another scheme involves a combination of intravenous ampicillin / sulbactam 1.5-3 g every 8-6 hours to stabilize the situation, and then change the combination to amoxicillin / clavulanic acid 1.2 g PO every 8 hours. Therapy should continue until the full abscess purification and cavity regression (from 3-4 weeks to about 14 weeks). Metronidazole (500 mg 3-4 times a day) is used only in combination with other antibacterial agents because it is ineffective against microaerophilic streptococci, which often are the part of a primary pulmonary abscesses mixed flora. The combination of antibiotic therapy with antifungal drugs and probiotics is compulsory.

Detoxification therapy involves the infusion of saline solution (0,9% NaCl -1000-3000 ml / day), 5% glucose solution 400-800 ml / day; correction of microcirculation -heparin 20000-40000 IU / day; dysproteinemia correction - human albumin 100-500 ml / day / drip IV; oxygen therapy; immunosubstitutive therapy - native or fresh frozen plasma 100-200 ml / drip IV. Indications for surgical treatment: acute abscess complications (pulmonary bleeding, pyopneumothorax, empyema), suspected tumor.

Prognosis. Mortality rate in the patients with the primary abscess is about 2%, in the patients with the secondary abscess is much higher - sometimes up to 75%. Unfavorable prognostic factors include age more than 60 years, presence of aerobic bacteria, sepsis, disease duration more than 8 weeks, cavity size more than 6 cm.

Prevention. Great attention should be paid to oral hygiene, patients at risk of aspiration recommend lifting the head end of the bed during rest, great attention should be paid to the patients that require special sensitivity to infections (recipients after organ transplantation, patients with significantly compromised immune system).

Pulmonary gangrene

Definition. Pulmonary gangrene (Gangrene of the lungs) is the infectious lung destruction, characterized by a large suppurative necrotic inflammation without well-marked boundaries with multiple decay cavities formation, differs by considerably severe course, developed in the individuals with deeply impaired immunity.

Etiology. Pathogens are microorganisms associations with obligatory presence of anaerobic microorganisms (*Bacteroides fragilis*, *Bacteroides melaninogenicus*, *Fusobacterium nucleatum*, *Peptococcus niger*, *Peptostreptococcus spp.*). Gangrene of the lungs can occur only against the background of previous certain pathological changes of the lungs, this process is always secondary. Pathogenic bacteria multiply very well in necrotic masses, but even this issue is the cause or consequence of lung tissue necrosis. Lung gangrene can develop due to lobar pneumonia, aspiration pneumonia, pulmonary artery thrombosis, in the case of rupture of the bronchus, carcinogenic or ulcerative lesions of the esophagus, compression due to tumors or aneurysms of the thoracic aorta, septic states.

Pathogenesis. In gangrene the purulent character inflammation become ichorous due to the rapid progression applies to fully lung with the next necrosis of the entire organ. Affected lung tissue becomes dark brownish-green, sometimes black with patches of softening lung tissue with the formation of multiple irregularly shaped cavities filled with stinking abominable greenish liquid. The pleura involve to the pathological process putrefactive empyema develops. The process has an undulating course with a gradual progression.

Clinical symptoms. The patient complaints of cough, high fever - up to 40°C, chest pain. After some time (10-15 days) the purulent fusion and sloughing through the respiratory tract causes discharge of putrefactive sputum in amount about 1-1.5 liters per day. There are pronounced signs of respiratory failure and symptoms of severe intoxication. The vascular lesions accompanied by hemoptysis, pulmonary hemorrhage, sometimes significant at about 70-75% of the patients. There is always a fever; the patient loses strength, weakens, becomes anemic, chills and profuse night sweats continue and gets the signs of sepsis.

At physical examination grayish, dry skin observed, , cyanosis of the lips and nail phalanges, in the early stages over the affected area the dull percussion sound is defined that changes in the next by the signs of the cavities formation. Painful enlarged liver is determined by palpation of the abdomen.

Diagnosis. In the **general blood analysis** leukocytosis with leukogram left shift, the significant acceleration of ESR (60-70 mm / h), anemia observe. Changes in **urinalysis** indicate the toxic kidney damage. Discharged **sputum** upon standing is divided into three layers: the upper - a dense muddy yellowish-brown foam, middle - clean watery fluid, lower - pus, blood, lung tissue sequesters. Microscopy reveals putrefactive bacteria, puss, elastic tissue, fat crystals, granular material.

Radiographic signs in the form of a large shade before the burst of purulent necrotic masses later can form one large cavity with the lung tissue sequesters . After the burst of purulent necrotic mass into the airways in the massive dark shadow multiple small irregularly shaped light focuses with the liquid level in some of them are reveals.

Endoscopic signs of diffuse purulent bronchitis of high degree inflammation with a significant number of purulent secretions detected by **fiber-optic bronchoscopy**. For early diagnosis of putrefy cavities **the chest computed tomography** is appointed.

Differential diagnosis. Diagnosis of lung gangrene is put mainly on the basis of clinical and radiological manifestations. The main radiographic gangrene of the lungs differences are the inflammatory infiltration of lung tissue and massive dark shadow without clear boundaries and not take one lobe, often all over the lung. No trend towards restrictions but disseminated process distribution and rapid accession of signs of pleural effusion and pyopneumotorax is observed.

Complications. The course of gangrene of the lungs may be complicated by hemoptysis, pulmonary hemorrhage, the development of putrefactive empyema, sepsis.

Treatment. Treatment usually antiseptic, the patients must be treated in the ICU with thoracic surgeon involving. Empirical antibiotic combination therapy includes intravenous fluoroquinolone III-IV generation (levofloxacin 0.5 g every 12-24 hours or moxifloxacin 0.4 g every 24 hours, or gatifloxacin 0.4 g every 24 hours) in combination with carbapenem (meropenem 1 g every 8 hours or imipenem 0.5 g every 4 hours) or cephalosporins III-IV generation (cefepime 2 g every 12 hours or cefpirome 2 g every 12 hours). Etiological therapy is conducted after the pathogen bacteriological determination.

Prognosis. The course is not necessarily fatal but always very hard. Recovery is usually possible in the patients of young and middle age without comorbidity states and the previous tuberculosis history. However, gangrene of the lungs can lead to sepsis, multiple organ failure and death. Resection of all gangrenosum tissue is indicated and is just one rescue operation.

Prevention. Reducing the likely impact of all risk factors is very suitable for prevention of pulmonary gangrene. The special attention should be given to improving the protective function of the upper respiratory tract, oral hygiene to minimize the possibility of pathological aspiration in the patients at particularly risk group.

Bronchiectasis

Definition. **Bronchiectasis** is an irreversible dilated bronchi deformation of focal or diffuse nature, classically distributed at cylindrical or tubular (the more common form) and varicose or sacculus.

Bronchiectatic disease is a purulent inflammation of the irreversibly altered bronchi with peribronchial infiltrative and sclerotic changes in violation of the drainage function and the development of atelectasis, cirrhosis, emphysema.

Etiology. Bronchiectasis is polyetiological disease. Bronchiectasis may arise from infectious and noninfectious causes. Focal bronchiectasis may be due to airway obstruction outside (compression peri bronchial lymph nodes or parenchymal tumor) or inside (tumor, foreign body, scar changes stenotic airways etc.). Diffuse bronchiectasis arising against systemic or infectious diseases.

The main etiological factors: genetically caused pathological changes of the bronchial tree (Kartagener's syndrome, cystic fibrosis); congenital anomalies (Mounier-Kuhn syndrome - tracheobronchomegaly, Williams-Campbell syndrome); infectious-inflammatory respiratory diseases in childhood (pertussis, measles); nonspecific chronic respiratory disease; tuberculosis; bacterial lungs destruction; chronic diffuse lung damage due to collagenoses, sarcoidosis, idiopathic pulmonary fibrosis; foreign bodies, tumors, etc. the tracheobronchial tree obstruction; thermal, and chemical damage.

The main etiological agents: *nontuberculous mycobacteria (NTM)*, *Micobacterium avium-intracellulare (MAC)*, *Haemophilus influenzae* and *P. aeruginosa*.

Epidemiology. The prevalence of bronchiectasis according to various studies ranged from 0.45 to 1.2%. Detected in children and young patients bronchiectasis is associated with frequent respiratory infections, bronchitis, pneumonia, whooping cough (pertussis), measles. In childhood, the difference is not found but in adults men suffer in 1,5-3 times more often than women. Overall incidence increases with age.

Pathogenesis. The main pathogenetic links of bronchiectasis are: the initiation of inflammation in the airways; violation of the bronchi drainage function; "motionless cilia" syndrome; the presence of pulmonary atelectasis, fibrotic changes in the lung parenchyma, poor circulation leads to the development of pulmonary hypertension.

Infection process in the abnormally dilated bronchi develops due to drainage function violation, delayed evacuation of infected bronchial secretions on the background of functional disorders of local and general immunity. Purulent inflammatory process causes profound mucosal and muscular layers damage, the degeneration of bronchi cartilage with subsequent development of pathological process. Over the time, there are significant violations of respiratory function by restrictive or mixed type, the symptoms of chronic intoxication, peribronchial pneumosclerosis, emphysema, the signs of respiratory and heart failure are appears.

Pathomorphology. Pathomorphological changes revealed at bronchiectasis include the signs of chronic inflammation, diffuse inflammatory destruction, mucus hypersecretion, replacement of bronchi columnar epithelium per cubic squamous, degeneration of the bronchial muscular layer and cartilage tissue, the alveolar and interstitial lung parenchyma destruction, fibrosis.

Classification. According to the course of the disease - mild, moderate, severe degree of severity; to localization - unilateral, bilateral, segmental, lobar, focal, diffuse; to the bronchiectasis form - cylindrical or tubular (the more common) and varicose or sacculum and mixed; to the phase - exacerbation and remission. Severity of bronchiectasis is determined by the severity of clinical symptoms, the exacerbations frequency and severity, the presence of complications.

Clinical symptoms. The most common clinical manifestation is the continuous productive cough with discharge of thick mucopurulent or purulent sputum with some regularity - in the morning and in the evening in specific drainage position, sputum volume during exacerbations is about 300 ml / day.

Subfebrile fever and high fever, loss of appetite to anorexia, weight loss, malaise, severe weakness, excessive sweating mainly at night are the signs of intoxication syndrome.

The most common another syndromes are the pain (pleural and muscle) syndrome, bronchial obstruction syndrome, respiratory failure, hemoptysis and signs of other complications.

During the physical examination skin pallor, increased skin moisture, reduce of its turgor reveals, the deformation of the distal phalanges as "drumsticks", the nails - as a "watch glass", muscle atrophy due to prolonged intoxication, asymmetric deformation of the chest, vocal fremitus increasing (pneumofibrosis) or decreasing (emphysema) are detected. Dullness of percussion sound is observed in the pulmonary atelectatic changes, in emphysematous – "bandbox" sound; auscultation determined the constant mixed moist and crepitating rales, and dry whistling wheezing, decreased after the phlegm discharge.

Diagnosis. In *general blood analysis* changes characterized chronic infectious inflammation according to severity is revealed leukocytosis, accelerated erythrocyte sedimentation rate, increased acute-phase reactants (C-reactive protein) and anemia. *Analysis of the sputum, bronchial secretions, bronchial lavage* can detect the pathogen and determine its *sensitivity to antibiotic* therapy.

The characteristic *radiographic* signs are strengthening and deformation of lung pattern, reducing of the affected segments and lobes of the lungs volume, a symptom of lung root "amputation", «tram tracks» availability, emphysematous changes, pleural fibrosis signs.

Computed tomography is the main modern method of bronchiectasis instrumental diagnosis to determine the form of bronchiectasis, localization, process extension. The localization of pathological process in the upper lobes is more often the characteristic of *cystic fibrosis* or *post-radiation fibrosis*. Bronchiectasis mainly affecting the lower lobes typically detected chronic recurrent aspiration. Bronchiectasis due to infection with nontuberculous mycobacteria more often with *Micobacterium avium-intracellulare complex (MAC)* usually affects middle lung fields.

Differential diagnosis. The same clinical signs can be detected in the presence of a history of *tuberculosis* or *lung abscess*, atypical process localization against the background of specific residual changes support bronchiectasis; *cystic fibrosis* can be exclude on the basis of the results of sweat glands secret chloride content test with detection of exogenous pancreatic function violations; *cystic lung hypoplasia* has less severe clinical symptoms accompanied by abnormal branching of bronchi and congenital malformations of other organs, Kartagener's syndrome characterized by the congenital bronchiectasis, perirhinal sinus pathology, heart inversion or dextraposition of internal organs.

Complication. The condition can be complicated by hemoptysis, pulmonary hemorrhage, bronchial obstructive syndrome, pleural empyema, spontaneous pneumothorax, abscesses, gangrene, lung cirrhosis, chronic pulmonary heart, sepsis, amyloidosis, gastric and duodenal ulcers.

Treatment. Treatment of bronchiectasis is aimed at controlling the activity of infection, bronchial hygiene and to minimize the risk of re-infection.

Bronchial sanitation is in the main focus in treatment of bronchiectasis. There are passive (physiotherapy - postural drainage, vibration massage, breathing exercises, mucolytics intake) and active (cleaning and bronchial lavage followed by administration of medicines) methods of rehabilitation should be applied. Postural drainage is made in view localization bronchiectasis at least 2 times a day. Drainage efficiency reinforced by the appointment of mucolytics: non-enzyme of direct action - acetylcysteine 600 mg / day for 4-6 months; indirect action- Bromhexine (mucolytic) 16 mg 3-4 times a day from 4 days to 4 weeks depending on the course; Carbocystein (mucoregulator) 750 mg 3 times daily for clinical effect, then 375 mg 4 times daily, duration of treatment no more than 8-10 days; Ambroxol (stimulant of surfactant) 75 mg daily after meals with plenty of warm liquids for 1-4 weeks.

Antibiotic etiological therapy is prescribed only after bacteriological study. Empirical therapy by protected aminopenicillins, cephalosporins of II-III generation, respiratory fluoroquinolones should be combine in different ways (intravenously, endobronchial, inhalation) administration of drugs to get adequate effect. Medical schemes include macrolides in combination with rifampin and ethambutol.

Drugs prescribed at exacerbation period for at least 7-10 days, the duration of medication by one drug should not exceed 14 days.

The bronchodilators (beta-2-agonists, short acting M-cholinolytics), detoxification drugs, immunomodulators should be prescribed at exacerbation also. Purpose systemic glucocorticoids may be important only in certain etiology such as association with allergic bronchopulmonary aspergilosis (ABPA), improvement in this group of patients can be observed after prolonged treatment by antifungal itraconazole. In some cases, surgical treatment is necessary. Indications for surgical treatment are the lack of conservative treatment effect during 2-3 years and presence of complications (pulmonary hemorrhage, abscess, atelectasis, poor drainage of pneumothorax, aspergiloma).

Prognosis. The prognosis can vary widely depending on the cause of the disease, frequency and severity of exacerbation and pathogen specificity.

Prevention. Primary prevention is the timely treatment and prophylaxis of acute infectious respiratory diseases in childhood. Resumption in immunodeficiency states (gamma globulin at medical disposal), vaccination of the patients with a predisposition to chronic respiratory diseases (influenza and pneumococcal vaccine) can reduce the risk of re-infection, to prevent exacerbation.

In order to prevent exacerbation the treatment may include the appointment of oral antibiotics (ciprofloxacin) daily 1 - 2 weeks each month; change them according to microflora sensitivity panel to prevent the development of resistance; appoint macrolides 1-3 times a week, using their anti-inflammatory effects and ability to inhibit gram negative forms; inhalation of aerosol antibiotics (tobramycin inhalation solution) over a month every other month to reduce the level of infection by microbial pathogens; intermittent antibiotics IV generation prescription at the severe course and pathogen resistance.

To prevent the progression of bronchial obstruction syndrome, in people who smoke - a complete rejection of habit.

Respiratory failure

Definition. Respiratory failure (RF) is a condition in which the respiratory system is unable to provide normal gas exchange, resulting in developing hypoxemia and / or hypercapnia.

According to E.Campbell RF is a condition when at quiescent state the oxygen partial pressure in arterial blood (PaO_2) <60 mm Hg and / or partial pressure of carbon dioxide (PaCO_2) > 49 mm Hg.

Etiology. Respiratory failure syndrome may occur at the functional disorders of the respiratory system any component (CNS regulation, peripheral innervation, respiratory muscles, chest, airways, lung parenchyma, alveoli). Various pharmacological effects, structural and metabolic disorders of the central nervous system may lead to the inhibition of the respiration nervous regulation, which can cause acute or chronic pulmonary hypoventilation with subsequent hypercapnia. It may be caused by brain tumor or vascular anomaly, an overdose of drugs and sedatives, metabolic disorders (myxedema, chronic metabolic alkalosis).

Concomitant hypoxemia and hypercapnia may occur on the basis of the peripheral nervous system disorders, the respiratory muscles damage, chest lesions (muscular dystrophy, myasthenia gravis, expressed kyphosis and scoliosis, morbid obesity, Guillain-Barre s-m, amyotrophic lateral sclerosis), making it impossible to maintain normal ventilation level according to the carbon dioxide production .

Severe bronchial obstruction is the common cause of acute and chronic hypercapnia (acute epiglottitis inflammation, trachea tumors, lower respiratory tract lesions - COPD, asthma, cystic fibrosis).

Hypercapnia can complicate the clinical course of hypoxemic respiratory failure resulting from pathological conditions involving a total alveolar ventilation volume decrease (cardiogenic pulmonary edema and noncardiogenic, aspiration pneumonia, severe pulmonary hemorrhage).

Common causes of respiratory failure

Type I (hypoxemic)	Type II (hypercapnic)
COPD Pneumonia Pulmonary edema Pulmonary fibrosis Asthma Pneumothorax Pulmonary embolism Pulmonary arterial hypertension Pneumoconiosis Granulomatous lung diseases Cyanotic congenital heart disease Bronchiectasis Acute respiratory distress syndrome Fat embolism syndrome Kyphoscoliosis Obesity	COPD Severe asthma Drug overdose Poisonings Myasthenia gravis Polyneuropathy Poliomyelitis Primary muscle disorders Porphyria Cervical cordotomy Head and cervical cord injury Primary alveolar hypoventilation Obesity-hypoventilation syndrome Pulmonary edema ARDS Myxedema Tetanus

Epidemiology. Respiratory failure is more syndrome than self-contained clinical nosological form therefore the spread of this one and over all frequency are not well known.

Respiratory failure is one of the most common causes for admission of the patients to the intensive care unit (ICU).

Mortality in acute RF reaches 40-49% and depends on the nature of the underlying disease, severity of lung dysfunction, probable systemic complications.

Mortality among the patients with acute hypoxemic respiratory failure, which is the main clinical manifestation of acute respiratory distress syndrome (ARDS) was always quite high so as 50-70%, but there has been decreased to about 30% due to the revision of treatment tactics of these patients.

Pathogenesis. The pathophysiological bases of respiratory failure occurrence and development is directly related to the process of gas exchange in the lungs, which is determined by the level of alveolar ventilation intensity, gases (O_2 and CO_2) diffusion through the alveolar-capillary membrane, pulmonary circulation state, ventilation-perfusion ratio also.

Ventilative ((hypercapnic) respiratory failure form usually accompanied by a total alveolar ventilatory capacity and respiratory minute volume decreasing, reducing the elimination of CO_2 to the development of hypercapnia ($PaCO_2 > 45$ mm Hg) followed by hypoxemia, compensated or decompensated respiratory acidosis development.

The basic mechanisms of ventilative respiratory failure are disorder of breathing process central regulation, peripheral nerve damage, respiratory muscles or chest sketch injury and other pathological states accompanied by ventilator capacity decreasing .

Parenchymal (hypoxemic) form of respiratory failure developed due to significant violations of blood oxygenation process in the lungs with a primary decreasing of $PaO_2 < 60$ mm Hg in the arterial blood.

The basic mechanisms of hypoxemia are ventilative-perfusion processes abnormality with alveolar shunt formation or alveolar dead space enlarged. The alveolar-capillary membrane functional surface area reduces and disorders of the gases diffusion process increased.

Classification. Respiratory failure may be classified on the basis of the external breathing function pathophysiological disorders. According to the preferred type of breathing mechanics breach there are *obstructive* and *restrictive* types distinguished. According to the severity of ventilative-perfusion disorders with a predominance of certain changes in the blood gas composition respiratory failure can be differentiated at *ventilative (hypercapnic)*, *parenchymal (hypoxemic)*, *mixed* forms.

Type	The mechanism of development	Clinical cause
obstructive	Arterial hypoxemia due to severe	COPD, chronic obstructive

	violations of ventilative-perfusion ratio, accompanied by significant and irregular disorder of blood oxygenation in lungs, with formation of perfusion form of respiratory failure with decreased $\text{PaO}_2 < 60 \text{ mm Hg}$.	bronchitis, pneumonia, asthma.
restrictive	Arterial hypercapnia due to lower volume of alveolar ventilation, reduction of CO_2 output, followed by hypoxia with ventilative form of respiratory failure formation ($\text{PaCO}_2 > 45 \text{ mm Hg}$. In.)	Severe kyphoscoliosis; massive pleural effusion, pneumothorax; lobar, total pneumonia, interstitial lung disease, pulmonary edema; atelectasis, lung resection, etc ..

According to the clinical course respiratory failure can be **acute** and **chronic** depending on the blood gas composition rate of change and compensatory abilities to eliminate these violations.

Clinical symptoms. Acute respiratory failure develops over a few minutes to hours and is characterized by life-threatening derangements in arterial blood gases and acid-base balance.

Chronic respiratory failure occurs on a background of progressive hypoxemia and hypercapnia increase according to progression of the underlying disease and can continue for many years.

The distinction between acute and chronic hypoxemic respiratory failure cannot be made only on the basis of arterial blood gases amount.

The clinical signs of chronic hypoxemia such as breathlessness, diffuse cyanosis, strengthening of respiratory muscles and participation of ancillary muscles in breathing, tachycardia, secondary polycythemia, cor pulmonale suggest lingering respiratory disorder.

Dyspnea can be inspiratory (more often at ventilative RF as restrictive type), expiratory (usually suggests obstructive type of RF) and mixed (obstructive and restrictive disorders combination).

Cyanosis (central, diffuse) related to violation of blood oxygenation and increased content of reduced hemoglobin and is an objective clinical sign of hypoxemia.

In acute so as in chronic form of respiratory failure the ancillary muscles participation in breathing act took place.

Tachycardia as a result of compensatory blood circulation intensification observed in any type and stage of respiratory failure.

Secondary polycythemia is caused by compensatory hypoxic stimulation of bone marrow and accompanied by a deterioration of blood rheological properties.

There are three stages of chronic respiratory failure:

I st - dyspnea occurrence as appropriate the realization of physical activity exceeded daily one;

II st - the appearance of dyspnea and other signs of respiratory failure during performing usual normal daily physical activity loads;

III st- the signs of respiratory failure occurs even at quiescent state.

Differential diagnosis. In the patients with the signs of respiratory failure for timely emergency care, determination for referral or transfer the patient to the intensive care unit (ICU) necessity should be quickly decided with the possibility of critical condition developing. According to information from Harrison's Principles of Internal Medicine (2015) the following types of respiratory failure requiring emergency care distinguished :

Type I- hypoxemic acute respiratory failure in consequence of decreased alveolar ventilation in the patients with pulmonary edema, pneumonia or alveolar hemorrhage. Pulmonary edema can be conditioned by increased pressure at microcirculative level that occurs in the patients with heart failure, or intravascular overflow as in the case of ARDS («low-pressure pulmonary edema»). This type of respiratory failure may occur in clinical conditions such as sepsis, aspiration pneumonia, repeated blood transfusions, pancreatitis.

Type II- alveolar hypoventilation is caused by and associated with the inability to effectively carbon dioxide CO₂ removing (hypercapnic respiratory failure) conditioned by inhibition of breathing act neural regulation due to central nervous system (CNS) affection, violation of neuromuscular function with increasing load at the respiratory system. These disorders accompanied by conditions associated with brain injuries, drugs and sedatives overdose, sleep apnea, severe hypothyroidism.

Type III of respiratory failure associated with the development of atelectasis, which often occur during surgery treatment (perioperative respiratory failure). The collapse of the dependent lung lobes may occur after general anesthesia, accompanied by a decrease of functional residual lung capacity. Frequent changes in body position, physiotherapy, medication control of abdominal pain and in the postoperative scar area. Positive-pressure ventilation can also be used to reverse the development of atelectasis.

Type IV respiratory failure resulting from hypoperfusion respiratory muscles in patients with shock as a result of respiratory distress associated with pulmonary edema (cardiogenic shock), lactic acidosis and anemia. Intubation and mechanical ventilation allows to redistribute cardiac output to vital organs while the shock is treated.

Diagnosis. Complete blood count (CBC) can detect anemia as the cause of tissue hypoxia. Found out polycythemia can be result of chronic hypoxemic respiratory failure. **Biochemical parameters** detected changes (including those that indicate the functional state of the liver, kidney, etc.) may be helpfull in the diagnostic process of etiology identification, in prevention of the development of possible complications in other organs and systems.

Chest radiography is essential in the determination of respiratory failure because it often reveals the cause. But sometimes distinguishing between cardigenic and noncardigenic edema is rather difficult. The signs of cardiomegaly, vascular redistribution, peribronchial cuffing, pleural effusions, two-sided lung infiltration suggests hydrostatic edema.

Examinations with using of modern research methods aimed at identifying the specific causes, occurrence mechanisms, severity assessment, concomitant functional and organic disorders and others. Evaluation of lung function using spirometry, spirometry, pneumotachometry, peakflowmetry, diffusion lungs capacity tests and others allows to diagnose the disorders of respiratory function, to give an objective assessment of the respiratory failure severity to provide differential diagnosis of obstructive and restrictive disorders, to substantiate pathogenetic therapy and to evaluate the effectiveness of treatment.

The main indicator of the traditional **spirometry** is vital capacity (VC), forced vital capacity (FVC) test allows to determine the parameters of pulmonary ventilation, characterizing the degree of intrapulmonary airways obstruction. Forced expiratory volume after 1 second (FEV₁) decreases both in the obstructive and restrictive disorders. Tiffno index (the ratio of FEV₁ to FVC) is significantly reduced in bronchial obstruction syndrome in consequence of exhalation deceleration. In restrictive disorders Tifno index is almost unchanged due to the nearly equal decrease in both indicators.

Computed spirometry today is the most promising method giving the possibility to conduct a quantitative assessment of pulmonary ventilation ratio "flow-volume" violations with the help of modern computer systems. In the case of bronchial airway disorder the curve "flow-volume" that shaped like loop is deformed in expiratory part, left shift of the expiratory part due to the increased lung volume marked. In restrictive disorders the curve "flow-volume" looks like a small copy of the normal curve shifted to the right due to the general decrease in lung volume.

Gas analytical methods for determining the structure of total vital capacity (TVC) can more reliably diagnose pulmonary ventilation mechanisms in restrictive disorders. The main criterion for the presence of restrictive disorders is a significant reduction in TVC. When combined with bronchial obstruction TVC structure changes significantly - increased RV / TVC (> 35%) and FRC / TVC (> 50%). The analysis of TVC allows to differentiate all types of ventilation violations as restrictive and obstructive and mixed.

The level of gas exchange is determined by the **ventilation-perfusion ratio** (N-0,8-1,0), one of the most important characteristics of respiratory function. In the case of decreased expiratory reserve volume (ERW) perfusion leads to reducing of arterial blood oxygenation – hypoxemia. ERW increasing is observed in the excessive ventilation of the areas with significant reduction in perfusion that leads to hypercapnia.

Diffusion lung capacity tests with gas composition in exhaled air determination (N - D_LCO_2 18 ml / min / mmHg / m²; DLO_2 - 22,1418 ml / min / mmHg / m² ($D_LCO \times 1,23$) allows to reveal decrease due to reducing the surface area of the alveolar-capillary interaction in emphysema or in vascular lesions in the lung capillary bed in vasculitis, or as a result pulmonary artery small branches thromboemboli, or diffuse lung parenchyma lesions with pneumonia, pulmonary edema, diffuse fibrosis, cystic fibrosis, alveolitis etc.

Determination of the blood gas measurement carried PaO_2 (N - 80-95mm Hg) and $PaCO_2$ ((N - 35-45mm Hg)), determination of oxygen saturation (hemoglobin oxygen saturation).

PaO_2 values <80 mm Hg is considered as the initial manifestation of arterial hypoxemia with the development of mild compensated respiratory failure. Reducing index <60mm Hg occurs with moderate and severe decompensated respiratory failure. $PaCO_2$ value of > 50 mm Hg (hypercapnia associated with clinics of significant ventilative or mixed respiratory failure. Clinically ventilative respiratory failure accompanied by hypercapnia and increasing hypoxemia. The initial stages of parenchymal RF accompanied by a decrease in PaO_2 (hypoxemia), compensatory dyspnea with alveolar hyperventilation that leads first to hypocapnia than with the progression of changes to hypercapnia $PaCO_2$ with values > 45-50 mm Hg. So the clinical manifestation of parenchymal RF accompanied by progressive hypoxemia combined with hypercapnia.

Violation of **acid-base balance** is determined by measuring blood pH, buffer bases containing (BB), standard bicarbonate (SB), the value of bases deficit or excess (DE). For the diagnosis of respiratory or nonrespiratory acidosis and alkalosis sometimes it is enough to define the blood pH (N - 7,37-7,43), $PaCO_2$ (N - 35-45mm Hg) , BE (N from -2.5 to + 2,5 mmol / L) and SB (21-25 mmol / L). Compensated respiratory alkalosis is caused by increased $PaCO_2$, for compensated metabolic acidosis the changes of BE are primary but $PaCO_2$ deviation is secondary.

In order to realize the monitoring of peripheral tissue supply of oxygen according to saturation of hemoglobin with oxygen in clinical practice most common is used the non-invasive **pulsoxymetry** method (in N saturation of hemoglobin with oxygen > 90%). Index decreases in hypoxemia and decline of PaO_2 <60 mm Hg.

Complication. In severe respiratory failure on a background of significant changes in gas composition of blood, acid-base disbalance, disorders of pulmonary hemodynamics serious complications in other organs and systems can be arise. Severe **systemic complications** (cardiac, vascular, renal, gastrointestinal, neurological, etc.) with impaired function due to severe hypoxia of organs and tissues, are more common in acute respiratory failure. The clinical picture of **acute respiratory failure** characterized by a rapid increase of the symptoms involving vital organs to the pathological process. There are 3 pathogenic stages: 1 - symptoms of compensatory circulatory and respiratory systems activation; 2 - the appearance of clinical and laboratory signs of hypoxemia-hypercapnia; 3 - signs of decompensation of respiratory function, rapid multiorgan system failure progression.

The risk of unfavorable dramatic prognosis significantly increases on the basis of multiorgan system failure.

Chronic respiratory failure can be complicated by the development of **pulmonary arterial hypertension**. Hypoxic pulmonary vasoconstriction due to chronic alveolar hypoxia manifests with Euler-Lil'yestrand's reflex, and bronchial passability disorder, endothelial dysfunction, anatomical changes in pulmonary vessels associated with hypercapnia. Chronic respiratory failure can be complicated by formation of chronic pulmonary heart. **Chronic pulmonary heart (Cor pulmonale)** is a serious heart condition in which there is enlargement and failure of the

right ventricle resulting from lung disease (structural and functional changes of right heart - myocardial hypertrophy, expansion cavities, diastolic and systolic dysfunction of the right ventricle, the relative tricuspid valve insufficiency, increased central venous pressure, congestive venous disorders in systemic circulation). Chronic pulmonary heart may be compensated (in the absence of heart failure) and decompensated (if any signs of heart failure presence).

Treatment. Tactics of treatment will depend on the causes, characteristics of pathophysiological processes that caused and contributed to the development of respiratory failure.

Treatment of the patients with ARF should be conducted in a specialized department or in the intensive care unit and should include the removal of the main factors of respiratory failure, ensuring the airway with maintaining adequate levels of ventilation, correction of hypoxemia and tissue hypoxia, acid-base dysbalance, hemodynamics, precautions to prevent complications.

Most patients with chronic respiratory insufficiency can be treated at home using long-term oxygen therapy or ventilation support with complex treatment of the underlying disease, which was the cause of respiratory failure.

The treatment tactics depends on the nature and severity of the underlying disease, type of the respiratory failure, blood gas composition, state of the acid-base balance, presence of comorbid conditions, age of the patient.

Oxygentherapy is an essential part of a comprehensive treatment of respiratory failure with the purpose of reverse or prevent tissue hypoxia. Indications for oxygen therapy are the following clinical and laboratory data: $\text{PaO}_2 < 60$ mm Hg st.ta / or $\text{SaO}_2 < 90\%$, tachypnea, cyanosis, tahy- or bradycardia, arterial hypo- or hypertension, reduced exercise tolerance, possible impaired consciousness, metabolic acidosis phenomenon and others. Oxygen therapy can be inhaled, hyperbaric, intravenously, or in the form of extracorporeal membrane oxygenation (ECMO), or artificial oxygen carriers using, prescription of anti-hypoxic agents.

Different inhaled systems of oxygen or oxygen-air mixture delivery into the respiratory tract can be used. In the patients with acute COPD the nasal cannula or Venturi mask appropriate to use allowing to create a mixture of oxygen with FiO_2 24-40%; in trauma, pneumonia - simple full-face mask (FiO_2 to 35-50%); in ARDS - masks with consumed sac.

Patency of airways resumption at the patients with respiratory failure of any etiology (parenchymatic or ventilative) is one of the most important conditions for the effective treatment. Hydration and warming of the air, percussive or vibrating massage of the chest, postural drainage combined with **bronchodilators** and **expectorative agents** can have a positive effect. The best is the inhaled route of administration using ultrasonic nebulizers that create aerosol particles up to 1.5 nm with increased penetrating power. At the ARF anticholinergic drugs used aminophylline / theophylline or beta-2-agonists as bronchodilator. In severe bronchial obstruction combine treatment with inhalation of beta-2-agonists and parenteral or PO administration of other bronchodilators expedient. Euphilline initially introduced with the aim of saturation IV slowly at account of 6 mg / kg (in 10 ml of 0.9% NaCl solution), then IV drip in a maintenance dose of 0.5 mg / kg / h. For the patients with concomitant liver disorder and with signs of chronic heart failure the dose reduced to 0.1-0.2 mg / kg / h. Expectorative agents parenterally such as ambroxol in the dose of 10-30 mg / kg can be administered, if necessary - prednisone in a daily dose of 0.5-0.6 mg / kg or hydrocortisone 2.5 mg / kg every 6 hours. Acetylcysteine can reduce the viscosity of sputum. In the case of ineffective airways clearing by bronchodilators and expectorative agents, if it is necessary **the tracheobronchial catheterization** can be used that allows evacuate sputum.

In order to remove phlegm, sanitation airway mucosa of trachea to segmental bronchi **fibrobronchoscopy (FBS)** method uses.

If it is necessary to prolonged constant aspiration of tracheobronchial secretions but the inability to perform or contraindications to endotracheal intubation presence, the transmitting transcutaneous trachea and bronchi catheterization (**microtraheostomy**) is recommended.

If no effect on achievement provide adequate airway **endotracheal intubation** and **mechanical ventilation (MV)** applied. Increasing of PaO₂ and PaCO₂ reduction is the main task of mechanical ventilation.

The **artificial lung ventilation (ALV)** can be carried out by invasive methods such as endotracheal or through tracheostoma and non-invasive methods using the nasal or full-face masks. **Noninvasive positive-pressure ventilation** - method to create a positive pressure in airways in different phases of the respiratory cycle, facilitates breathing and improves blood oxygenation by increasing the pressure gradient between the diffuse and gaseous exchange zones. **Noninvasive positive-pressure ventilation** requires compulsory monitoring of heartbeat rate (HR), blood pressure (BP), ECG, oxygen saturation of hemoglobin, blood gas composition. However, it is a simple and convenient method of non-invasive respiratory support without complications that may arise in endotracheal intubation or tracheotomy. Noninvasive positive-pressure ventilation should be considered in patients with mild or moderate but acute respiratory failure.

Ventilation with positive end-expiratory pressure due to a slight positive pressure prevents the shrinkage of the alveoli that improves oxygenation and reduces hypoxemia. This mode is used to treat patients with parenchymal acute respiratory failure or with the signs of bronchial obstruction.

Invasive ventilation is performed for the patients with ARF, accompanied by severe dyspnea, agitation, confusion of consciousness or coma, progressive cyanosis, hemodynamic instability, hypotension, tachy- or bradycardia, active engagement of subsidiary muscles supporting the act of breathing; TLC half reduction, hemoglobin oxygen saturation <80%, PaO₂ <55 mm Hg. Art., PaCO₂ > 53 mm Hg, pH <7.3. Invasive ventilation is more effective in patients with ventilative form of acute respiratory failure.

Possible complications of mechanical ventilation are spontaneous pneumothorax, hypovolemia, the occurrence of respiratory alkalosis and related disorders of the central mechanisms of respiratory regulation, hemodynamics instability, electrolyte and gas exchange disorders; aspiration infectious complications and others.

Clinical criteria for mechanical ventilation cessation is the general condition of the patient stabilization, independent breath with RR <20-22 / min, SaO₂ > 90%, stabilization of normal blood gas composition. In order to maintain **hemodynamic disorder** which manifested by hypovolemia patients prescribed infusion of physiological saline or low dextrans that not only replenish blood volume, but also optimize the microcirculation by improving rheological blood properties.

Prognosis. The mortality in the patients with respiratory failure associated with the etiology of pathological condition that led to its development. Patient's age is of great importance, more likely to have a favorable prognosis are the patients younger than 60 years. The mortality rate for ARDS reaches 40-45%. In the majority of patients surviving after ARDS manifest some impairment of lung function over a time after recovery. The higher mortality observed among patients with hypercapnic respiratory failure due to the presence of chronic respiratory disease and concomitant diseases of the heart, liver, kidney and neurological disorders. In severe exacerbations of COPD mortality rate can reach 30%, but in general mortality in patients with COPD and acute respiratory failure recently dropped almost 10%. The life prognosis of patients with chronic respiratory failure significantly improved by the possibility of long-term oxygen therapy at home (more than 15 hours a day with FiO₂ 24-28%).

Prevention. Timely detection and adequate treatment of the pathological conditions that can lead to the respiratory failure development; prevention and effective treatment of the exacerbations of the respiratory system chronic diseases. Prevention of the complications

Materials for self-control

1. A man of 40 years old, complains of paroxysmal cough with yellowish-brown color expectoration, the pain in his right side, associated with deep breathing, sweating. Sick 6 days after exposure. He took an aspirin. Objectively: T - 39.6 ° C, RR - 26 / min, Ps - 110 / min, BP - 110/70 mm Hg. In the lower lobes of the lungs - sounorous fine bubbling wet rales. X-ray: the lower part of the lung - a massive inhomogeneous infiltration with areas of enlightenment, sinus differentiated. What complication of the disease most likely to develop in patients?

A. Fibrinous pleurisy

B. Abscess formation

S. Empyema

D. Spontaneous pneumothorax

E. Pulmonary atelectasis

2. The student of 17 years, in childhood is often sick with respiratory diseases. Between the episodes of ARI persisted cough with phlegm continue. Once noticed blood in the sputum examined at a TB were encouraged to observe. Above the lungs, especially at right, variegated wheezing auscultated. Mucopurulent expectoration by 50 ml per day. X-ray – harsh lung pattern and cell image (honeycomb-like) on the right lower lobe. What is the most likely diagnosis?

A. Chronic lung abscess

B. Chronic bronchitis

C. Pulmonary fibrosis metapnevmonichny

D. Metatuberkulezny fibrosis

E. Bronchiectasis

3. Male of 38 years old fell ill two weeks ago, cough, weakness, fever up to 38.0 ° C. Condition deteriorated acute at the end of the first week, when there was a chill, pouring sweats, in the evening the temperature up to 39.0 ° C. Over the 2 days before admission the patient allocate a large amount of foul-smelling sputum with blood through coughing, after which the patient's condition improved. Ps - 80 / min, the RR -.20 / min, T⁰-. 37.6°C. What changes are possible at the chest cavity radiograph?

A. The shift of the mediastinum towards homogeneous shade

B. Smooth rounded shadow in the lung field

C. The presence of the cavity with the horizontal liquid level

D. Shade in the lower section of the oblique upper boundary

E. Eclipse lobe

4. A man 54 years old complains of chest pain, increased shortness of breath, cough with blood streaks. In anamnesis: prolonged cough with purulent sputum up to 200 ml per day, more in the morning, periodic body temperature increase up to 37.8 ° C, sweating, chills. Smokes with 14 years. Objective: body weight is reduced, the skin with an earthy shade, swollen face, fingers in the form of "drumsticks", in the lungs at the background of pulmonary and boxed sounds - dullness, dry and sonorous medium and coarse bubbling rales and wheezing. In the blood: leukocytosis, ESR moderately accelerated. What is the most likely cause of pulmonary hemorrhage in a patient?

A. Tuberculosis

B. Bronchiectasis

C. Chronic bronchitis

D. Lung abscess

E. Lung Cancer

5. A woman aged 58, was admitted to the hospital complaining of shortness of breath and palpitations. Objectively: the state is severe, restless, excited, noisy breathing with the

participation of accessory muscles, recurrent seizures, diffuse cyanosis. In the lungs - scattered dry rales, breath is deeply weakened in the lower lobes. Ps -. 100 / min, 3 extrasystolic beats / min, liver near the edge of the costal arch, there is no swelling, BP -. 140/100 mm Hg, PaO₂ - 45 mm Hg., pH -7.3. What syndrome is most likely leading at the patient?

A. Respiratory failure

- B. Hypertension
- S. Tachycardial
- D. Arrhythmia
- E. Heart Failure

6. The patient of 50 years, was admitted to the hospital for 9 days after onset of disease with complaints of fever up to 38.5 ° C, severe weakness, pain in the right shoulder blade during breathing, a dry cough. RR -28 / min. Ps - 100 / min, signs of intoxication.. In the area of the right shoulder blade dullness, bronchial breathing, single finely wheezing and crackles. After three days, cough arose with the release of 200 mL of purulent sputum, after which the body temperature decreased. At the level of the blade angle is detected on the background of the infiltration of the lungs rounded enlightenment with the horizontal level of the liquid. Diagnosis?

A Lung cyst

B. Acute lung abscess

- C. Lung Cancer with the decay
- D. Bronchiectasis
- E. Pleural cavity limited empyema

7. The patient was diagnosed with pneumonia. Despite treatment, there was a hectic fever, and then sputum by "full mouth." What disease should be suspected?

A. Lung abscess

- B. Bronchiectasis
- C. Chronic bronchitis
- D. Pulmonary tuberculosis
- E. Staphylococcal pneumonia

Recommended literature

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