The diseases of respiratory organs

• ACUTE BACTERIAL INFECTIONS OF THE LUNGS
  • Acute and chronic bronchitis,
  • pneumonia,
  • destructive processes (abscess and gangrene),
  • bronchial asthma,
  • chronic non-specific pulmonary diseases and cancer of lungs

ACUTE BACTERIAL INFECTIONS OF THE LUNGS

• Occur when normal lung or systemic protective mechanisms are impaired.
  • Pulmonary protective mechanisms include
    • nasal,
    • tracheobronchial,
    • and alveolar mechanisms to filter,
    • neutralize, and clear inhaled organisms and particles.
  • Important factors interfering with normal lung defenses are
    • Decreased cough reflex leading to aspiration (seen in coma, anesthesia, drug effects).
    • Injury to mucociliary apparatus (as with cigarette or other smoke / gaseous inhalations).
    • Decreased phagocytic / bactericidal function of the alveolar macrophage (as a result of alcohol, tobacco, oxygen toxicity).
    • Edema/congestion
    • Accumulation of secretions.

• Pathogenic organisms gain access to the lung through
  • the airways,
  • through the bloodstream,
  • by traumatic implantation,
  • or by direct spread across the diaphragm probably through the lymphatics.
• The most common route is the airways.

Pneumonia

• Pneumonia is acute inflammation of the respiratory tract with deposition of intraalveolar exudates.
• It is a disease, which unites the large group of various after etiology, pathogenesis and morphological description of inflammations of respiratory department of lungs.
• There are three ways of penetration of exciter of pneumonia in lungs –
  • bronchogenic,
  • hematogenic and
  • lymphogenic.
• The first of them has a leading value.

Clinical-morphological classification

• Lobar pneumonia.
• Bronchopneumonia.
• Interstitial pneumonia.

Etiologic classification of pneumonia

Bacterial pneumonia.
Viral and mycoplasmal pneumonia.
Other types of pneumonias:
- Pneumocystis carinii pneumonia.
- Legionella pneumonia.
- Aspiration pneumonia.
- Hypostatic pneumonia.
- Lipid pneumonia.

Lobar pneumonia

- Synonyms: crupous, lobular, fibrinous, pleurapneumonia.
- Croupous pneumonia is infectious-allergic infection and involves a lobe of lung.
- Most lobar pneumonias are caused by pneumococci and Klebsiella pneumonia which enter the lungs via the airways.
- The pneumococcus continues to be responsible for 30% to 80% or more of community-acquired pneumonias.

- Illness often arises up for persons
  - with alcoholism,
  - avitaminosis,
  - cardiac insufficiency,
  - chronic overstrain.

Morphological stage of croupous pneumonia by (K.Rokitansky)

- 1 - stage of congestion (from 12 h to 3 days),
- 2-stage of red hepatization (1-3 days),
- 3-stage of grey hepatization (2-6 days),
- 4-stage of completion.

Pathomorphology of croupous pneumonia

- Pneumonia begins with the small hotbed of inflammation in the back or back-lateral departments of lungs round the colonies of pneumococcus.
- Inflammation spreads a contact way and quickly enough takes one or a few pulmonary particles.
- In the stage of congestion a lung is megascopic in a volume, tissue of it filling out and sanguineous.
- In the stage red hepatization exudate is enriched on a fibrin and red corpuscles. Lungs after closeness remind a liver, on a cut – crimson. The color of phlegm is ferruginous.
- stage of grey hepatization On a 4-6th day composition of exudate changes – red corpuscles disappear, but the number of neutrophils which phagocyte pneumococcus grows. A surface of lungs is on the cut of grey color (stage of grey grained detritus it is possible to find tailings of fibrins hepatization). In the period of convalescence exudate resolves.
- stage of congestion
  - tissue of it filling out and sanguineous.

Complications of croupous pneumonia
are divided into lung and extralung.

**Intolung**
- Carnification belongs to the first,
- empyema of pleura,
- abscess formation, and
- gangrene.

**Extralung**
- Empyema of pleura,
- abscess formation, and
- gangrene.

**Carnification** belongs to the first,
- abscess formation,
- gangrene.

**Carnification abscess formation**
- gangrene.

**Complications of croupous pneumonia**
- Pneumococcus inflammatory processes in different organs:
  - lymphadenitis,
  - meningitis,
  - peritonitis,
  - arthritis, etc.

**Focus pneumonia (bronchopneumonia)**
- Unite different originally inflammations of lungs the general line of which is localization of primary process in bronchial tubes. From here inflammation passes to pulmonary tissue and can be limited acinus, by a particle, segment or particle.
- Focus pneumonia is met more frequent, than croupous.
- As children and old persons have an independent disease for years.
- Focus pneumonia is complicated by sharp respiratory and viral diseases (flu, measles).

**Bronchopneumonia**
- Bronchopneumonia
- Bronchopneumonia

- can arise up
- at insufficiency of circulation of blood,
- especially on a background the stagnant phenomena in lungs (stagnant pneumonia),
- at the protracted confinement to bed mode for heavy and weakened patients (hypostatic pneumonia),
- in a postoperation period.

**Morphogenesis of bronchopneumonia**
- In most cases the reason of bronchopneumonia is the aerogenic infection,
- but hematogenic is not eliminated and lymphogenic ways of infecting.
- A process begins with bronchiole and passes to alveolar motions.
- To the bronchitis can join peribronchitis. From peribronchial tissue a process passes to the nearby alveolar ways (peribronchial pneumonia).
- Inflammation of alveolar tissue quite often is preceded the slump of alveolar ways. It can be consequence of clench from outside or corking of bronchial tube with exudates and next suction of air from alveolar ways which lost connection with respiratory ways.

**Pathomorphology of bronchopneumonia**
- Exudate at bronchopneumonia consists of serous liquid with the admixture of leucocytes, falling off cells of alveolar epithelium, red corpuscles, at times to the fibrin. That is why serous, festering, desquamation, hemorrhagic and fibrinous pneumonia is distinguished.
• Macroscopically separate inflammatory focuses which correspond to the staggered bronchial tubes or particles appear in lungs. They burst above the surface of cut, have a yellow grey, grey or red color, dense by touch, sink in water. A turbid liquid which does not contain the blisters of air flows down during squeezing of them. From bronchiole mucus-festering exudate is pressed out.

**bronchopneumonia**

**Intermediate pneumonia**

• belongs to the atypical forms. Interstitial (intermediate) pneumonia spreads mainly on intermediate tissue
• It met at viral infections, croupous pneumonia.
• A process begins with a bronchitis with next distribution on lymphatic ways (lymphangitis) or vasculitis (system red lupus). Productive inflammation prevails at times (measles).
• More frequent is festering lymphangitis.
• Distinguished peribronchial, interlobular and interalveolar pneumonia.
• Macroscopically rather yellow ribbons which mark off particles one from one induration evidently. Sometimes at festering inflammation the intervals of sequestrum and particles become separated.
• Such pneumonia assists development of interstitial emphysema. Complication are abscess formation, empyema, mediastinitis.

**Intermediate pneumonia**

**Pneumonia in children**

• has some features:
• a) an inflammatory process develops mainly in the respirator departments of lungs;
• b) infecting takes place antenatal intrauterine or through aspiration of amniotic waters;
• c) hyaline membranes appear as a result of high permeability of blood vessels;
• d) infection is more frequent than for adults, spreads outside lungs – on kidneys, liver, cerebrum.

**Bronchitis**

• is distinguished for a sharp and chronic bronchitis (bronchitis acuta, bronchitis chronica).
• Among the etiologic factors of sharp inflammation of bronchial tubes of primary value is given to viruses and bacteria which cause respirator diseases.
• From physical factors it follows to select the pathogenic action of
  • dry or cold air,
  • dust,
• from chemical is breathing in tobacco smoke,
• steams of chlorine, oxides of nitrogen and etc.
• The inherited insolvency of barrier mechanisms of mucus, insufficiency of cellular and humoral (IGA) protective factors of local value, assists development of bronchitis. In reply to the pathogenic influencing of gland and goblet cells of mucus bronchial tubes producing of mucus increases.
• It results in peeling of ciliary prismatic epithelium, baring of mucus and penetration of infection through the membrane of bronchial tube.

**Acute bronchitis**

• can be independent nosology unit or second sign of row of other illnesses (croupous pneumonia, uremia and others like that). In mucus bronchial tubes almost all forms of catarrhal inflammation are developed – serous, festering, fibrinous, fibrinous-hemorrhagic, mucus. Destruction of mucus is sometimes possible with development of ulcers. In such cases it is talked about a destructively ulcerous bronchitis.
• Inflammation begins from a mucus membrane (endobronchitis), then passes to the muscular layer (endomesobronchitis) and in a terminal phase takes all of layers (panbronchitis).
• Existing of sharp bronchitis can be complicated with bronchopneumonia or peribronchial by intermediate pneumonia.
• Bronchopneumonia mostly is the result of aspiration of the infected mucus in the respirator department of lungs. Peribronchial intermediate pneumonia arises up as a result of transition of inflammation not only on peribronchial but also on interstitial tissue.
• Serous and mucus catarrh is quickly completed by convalescence.
• Festering, fibrinous and fibrinous-hemorrhagic catarrh, and also an ulcerous-destructive bronchitis have the protracted motion and often pass to the chronic form or pneumonia.

**Acute bronchitis**

*Chronic inflammation* of bronchial tubes

• is shown up in such forms:
  • a) chronic mucus or festering catarrh with atrophy of mucus, by the cystous regeneration of glands and metaplasia of prismatic epithelium in multi-layered flat;
  • b) chronic productive inflammation is with formation of polyposis from granulation tissue (polyposis chronic bronchitis);
  • c) deformation of bronchial tube at ripening of granulation tissue, excrescence of connecting tissue in a muscular layer, sclerosis and atrophy of mucus (deforming chronic bronchitis).

**A chronic bronchitis**

• with the protracted motion, except for **sclerotic changes**, is accompanied by **dystrophy** elastic, muscular and cartilaginous frameworks.
• That is why during a cough, when intrabronchial pressure grows sharply, in the areas of the least resistance the membrane of bronchial tube is changed broadens and bursts.
• So saccate bronchioectasis appear.
• At diffuse expansion of bronchial tube they have a cylinder form.

**A chronic bronchitis**

• A chronic bronchitis is always accompanied violation of drainage function of bronchial tubes of, which causes the delay of their maintenance in lower departments, closing of road clearance of bronchiole and development of bronchiolung complications:
  • obstructive emphysema,
  • chronic pneumonia,
  • pneumofibrosis.

**A chronic bronchitis**

**Bronchoeptasis (bronchektasia)**

is born and acquired expansions of bronchial tubes as cylinders or sack.

Born bronchioeptasis arise up in connection with violation of forming of bronchial tree. They are marked the chaotic location of structures of membrane of bronchial tubes.

Sometimes bronchiole are closed blindly in parenchyma of lungs, cysts appear. In such cases it is talked about a cystous lung. Bronchioeptasis is acquired related to the sharp bronchitis, pneumonia, and collapse of lungs.

**Pathomorphology of bronchioeptasis**

• At bronchioeptasis there are the phenomena
  • of chronic inflammation in the membrane of bronchial tubes,
  • metaplasia of prismatic epithelium in multi-layered flat,
  • dystrophic changes from the side of elastic fibres, cartilaginous tissue and leyomicyte,
  • sclerosis.

**bronchioeptasis**

**Pathomorphology of bronchioeptasis**

• In the cavities of bronchioeptasis saved mucus and festering exudate.
• On this basis there are abscesses,
• perifocal festering pneumonia,
• perifocal fibrous,
• obstructive emphysema.

Pathomorphology of bronchioectasis
• A sclerosis develops in vessels that at presence of plural bronchioectasis and emphysema results in development of hypertension in the small circle of circulation of blood and hypertrophy of right ventricle of heart.
• The signs of hypoxia appear with next violation of trophism of tissues.
• A very typical sign is a bulge of nail phalanges of fingers of hands and feet as “drumsticks”.

Complications of bronchioectasis

• pulmonary heart,
• general amyloidosis,
• hypoxic signs,
• sclerosis, etc.

Emphysema of lungs

• is the pathological state of pulmonary tissue, which is characterized by the promoted maintenance in it air.
Distinguish:
• vesicular,
• chronic diffuse obstructive,
• chronic focus,
• vicarious,
• primary panacinaric,
• senile and
• interstitial emphysemas

• Development of vesicular emphysema is related to
• the chronic bronchitis,
• broncholitis and by their consequences –
• plural bronchoectasis.
• It is found out, that there is a deficit of inhibitors of protease at these diseases – to elastase, collagenase.
Insufficiency of important whey inhibitor,1-antitrypsin can be genetically conditioned. Activation of elastase and collagenase causes destruction of interalveolar partitions with confluence of alveolar ways in greater cavities.

Chronic diffuse obstructive emphysema

(emphysema pulmonum obstruictum diffusum chronicum)
• arises up at a chronic diffuse bronchitis. Development of it is taken to the valvular mechanism.
• It happens because of mucus clot which appears in shallow of bronchial tubes and bronchiole, at inhalation skips air in alveolar ways, but does not allow him to go out during exhalation.
• Air is saved in acinus, which are broaden as a result of insufficiency of elastic and collagenase fibres.
• At overwhelming expansion of respirator bronchiol and acinus talk about centeracinus emphysema, and in the case of expansion of large bronchial tubes and acinus – about panatsinamu emphysema.

Pathomorphology of Chronic diffuse obstructive emphysema

• Stretching of membranes of acinus results in thinning of interalveolar partitions, expansion of interalveolar por and formation of vesicular blisters.
The capillary net of partitions empties.
Thus, there is the considerable diminishing of area of interchange of gases and a vent function of lungs is violated.

Complication of **Chronic diffuse obstructive emphysema**

- Devastation of capillary net of alveolar ways together with the sclerosis of interalveolar capillaries conduces to development **hypertension of small circle of circulation of blood** and **hypertrophy of right ventricle of heart** (*pulmonary heart*).

**Chronic focus emphysema**

(emysema pulmonum focale chronicum)
- Arises up as a result of expansion of acinuses and respirator bronchiole round the old healths of tubercular inflammation or postatack scars.
- At confluence of a few bubbles it is talked about bullous emphysema.
- Bubbles, which are located under pleura, can break through in a pleura cavity and cause spontaneous pneumothorax.
- This type of emphysema is not accompanied by hypertension of small circle of circulation of blood, as a capillary river-bed is damaged on the limited area of lungs.

Vicarious emphysema

(emysema pulmonum vicarum s. compensatorium) is also named compensate.
- It arises up after the amputation of part lungs or one of lungs.
- This type of emphysema is accompanied by compensate hypertrophy and hyperplasia of structures of lungs, which are remained. Reason of primary (idiopathic) emphysema is unknown.
- For it such signs are characteristic,
  - as atrophy of membranes of alveolar ways,
  - reduction of capillary membrane, and
  - hypertension of small circle of circulation of blood.
- Development of senile emphysema, more precisely are emphysemas for old men, related to age-old involution of lungs.

**Interstitial emphysema**

(emysema pulmonum interstetiale)
- Characterized by penetration of air in intermediate tissue. Reason of such phenomenon is a break of alveolar ways at strong coughing motions.
- Through the cellular tissue of root lungs air gets to intercellular spaces of mediastinum (*pneumomediastinum*), hypodermic cellular tissue of neck, thorax, chairman (*hypodermic emphysema*).
- At pressure on the areas exaggerated air of skin to hear a characteristic crunch (crepitation).

**Bronchial asthma**

Is a chronic disease of allergic nature, which is characterized by attacks of expiration shortness of breath. Two main forms of bronchial asthma are selected:
- atopic and
- infectiously allergic.

*An atopic form Bronchial asthma*

Arises up at operating on the respiratory tracts of allergens of uninfectious origin.
In the half of cases illness is predefined a room dust in the complement of which high-allergic carbohydrates - products of disintegration of cellulose enter from a cotton plant. In addition, in a room dust the special type of tick with which link the origin of bronchial asthma in child's age is found.

From other allergens such, as vegetable pollen, epidermis and wool of animals, medications (acetylsalicylic acid, morphine), domestic chemicals have a most value (detergents, varnishes).

The Infectiously allergic form of bronchial asthma develops for patients with broncho-pulmonary pathology, caused infectious agents – viruses, bacteria, mushrooms.

**Pathogenesis** of both forms of bronchial asthma is similar.

- Immunological,
- pathochemical and
- patophysiological stages are selected.

**Atopic form**

- the immunological stage is characterized by hyperproduction and piling up of IgE.
- These antibodies are adsorbed on the cells of bronchiole and at the repeated hit of antigen in respiratory tracts cooperate with it after the mechanism of anaphylaxis.
- The reaction of immediate type is formed; the attack of shortness of breath arises up in a few minutes after the action of antigen.

At infectiously allergic bronchial asthma

- the immunological stage is opened out after the mechanism of hypersensitiveness of slow type,
- where a leading role is played by not antibodies, but sensibilised lymphocytes.
- The shortness of breath appears through 12-36 hours after a contact with an allergen.
- During the pathochemical stage under the act of complexes an antigen-antibody active substances are released-histamine acetylcholine, prostaglandin, leukotriene.
- They violate the function of cells -targets, stopped up in the membranes of bronchiole, – leyomiocytes, goblet and other cells.

**Pathomorphology of bronchial asthma**

- It shows up in bronchospasm,
- hypersecretions of mucus and
- edema of mucus bronchiole.
- Eventually vent possibilities them limited strongly. Exhalation especially suffers, when due to additional tension of respiratory muscles high intrapulmonar pressure is created.
- Bronchiole stick together, and exhalation is bothered or in general becomes impossible.
- Violation of breathing for patients with bronchial asthma shows up as the repeated attacks of shortness of breath.

During an attack of bronchial asthma

- there is infiltration of membranes of bronchiole by
  - eozinophiles,
  - neutrophiles,
  - labrocytes,
  - and T-lymphocytes.
- There is an edema of mucus and submucose,
- obturation of bronchiole by mucus in which eozinophiles appear and an epithelium peeling.
- In pulmonary tissue sharp obstructive emphysema develops with focus of atelectasis.
- Respiratory insufficiency which can lead to death of patient during an attack comes as a result.

**Chronic signs of bronchial asthma**

- the phenomena of diffuse chronic bronchitis,
• bulge belong and
• hyalinosis of basal membrane of bronchiole,
• sclerosis of intraalveolar partitions,
• chronic obstructive emphysema of lungs,
• hypertension of small circle of circulation of blood,
• hypertrophy of right ventricle of heart.

**Interstitial illnesses of lungs**

• are characterized by primary inflammatory process in intraalveolar connecting tissue (pneumonitis),
• they are also named by fibrotic alveolitis. They end with development of diffuse pneumofibrosis.

**Interstitial illnesses of lungs**

• Distinguish three nosology forms of fibrous alveolitis:
  1) idiopathic fibrous alveolitis;
  2) exogenous allergic alveolitis (lung „farmer”, „poultry farmer”, „cattle-breeder”, „textile worker”, „pharmaceutist”;
  3) toxic fibrous alveolitis.

**Reasons** **Interstitial illnesses of lungs** :

• 1) viral, bacterial, mycosis infection;
• 2) dust with the antigens of animal and vegetable origin;
• 3) medical preparations:, immunosuppressors, antitumor antibiotics, antidiabetic preparations and others like that

**Interstitial illnesses of lungs**

• In pathogenesis a basic role is played by the immunocomplex damages of capillaries between alveolar partitions and stroma of lungs with next cellular immune cytolsis.

**Interstitial illnesses of lungs**

• A pathological anatomy is presented by three stages:
  1) diffuse or granulematose alveolitis with infiltration neutrophiles, lymphocytes, plasmatic cells;
  2) disorganization of alveolar structures and pneumofibrosis;
  3) forming of cellular lungs with development alveolar-capillary block, panatcynaric emphysema, bronkioloectasia, hypertension in the small circle of circulation of blood, hypertrophy of right ventricle.

• A syndrome of Khammen-Rich is a sharp form of fibrous alveolitis, that meets at the system diseases of connecting tissue, viral active hepatitis.

**Pneumofibrosis**

– chronic process in lungs, which develops after the previous diseases of pulmonary tissue or interstitial

Charactzerized by excrescence of connecting tissue,
• deep alteration of microcirculations,
• development of hypertension in the small circle of circulation of blood with the next
• hypertrophy of right ventricle and
• forming of pulmonary heart,
• hypoxia of pulmonary tissue,
• its alteration and
• deformation

**Pneumofibrosis**
The cancer of lungs

• occupies the first place among malignant tumors for men and the second – for women.
• A death rate because of it is 26 %.
• The cancer of bronchial tubes arises up mainly for smokers (90 %).
• An important role belongs also to the carcinogenic substances which penetrate with blood and lymph

The cancer of lungs

• To the precancer states take
  • a chronic bronchitis,
  • bronchiolectasis,
• and to the precancer changes
  • hyperplasia,
  • displasia and
  • metaplasia of epithelium.

the cancer of lungs

• develops from the epithelium of bronchial tubes (bronkhogenic, central cancer),
• rarely – from the epithelium of bronchiole and alveolar epithelium (pneumogenic, peripheral cancer).

classification of cancer of lungs

• division after localization,
• character of growth,
• macroscopic form and
• microscopic kind.

classification of cancer of lungs

• According to localization selected:
  • 1) a periapical (central) cancer which is developed from an epithelium a barrel lobular and initial part of bronchial tube, grows as a node or polypus of white color and dense consistency;
  • 2) peripheral cancer which is developed from the peripheral department of bronchial tube and his branches, and also from alveolar epithelium, exophitaly grows for a long time, often is developed in the area of scar;
  • 3) the mixed (massive) cancer shows by itself soft tissue of white color, which can occupy all of fate or whole lung.

classification of cancer of lungs

• On character of growth
  • endophitic (endobronchial) and
  • exophitic (exobronchial and peribronchial) cancers are distinguished.
  • On macroscopic form a cancer is
    • plague-like,
    • polypus,
    • endobronchial diffuse,
    • ramified and
    • nodal ramified cancer.
  • On microscopic structure
    • flatcellular (epidermoid) cancer,
    • undifferentiated,
    • anaplastic cancer (finecellular, largecellular, oastmealcellular),
    • golden-flatcellular cancer,
    • of bronchial glands – adeno-cystous and mucoepidermoid are selected.
cancer of lungs
The cancer of lungs metastases
• by lymphogenic and hematogenic ways.
  • Lymphogenic metastases arise up in peribronchial, bifurcational, neck and other lymphatic nodes,
  • hematogenic – in a cerebrum, bones (mainly vertebrae), and adrenal glands.
• For a central cancer are typical lymphogenic metastases,
• for peripheral – hematogenic.
• First clinical sign of peripheral cancer, which is developed in the area of scar and has small enough sizes
  (microcarcinoma), related to the plural hematogenic metastases.
complication of cancer of lungs
• especially central, is development of atelectasis.
• Pneumonias,
• abscesses,
• bronchioectasis,
• bleeding which mask motion of cancer,
• developed as a result of violation of drainage function.
• Distribution on pleura causes development of serous-hemorrhagic and hemorrhagic pleuritis,
• and also to carcinomatosis of pleura.
• Cachexy during the cancer of lungs is developed later than during the cancer of stomach.
The pleuritis
• is inflammation of pleura more frequent arises up as complication some visceral pathologies.
• Often is met during the diseases of lungs:
  • pneumonias,
  • heart attack,
  • cancer,
  • tuberculosis and etc.,
• at rheumatism and other system diseases of connecting tissue (allergic pleuritis),
• at the diseases of kidneys (pleuritis of uremia).
• On character of inflammations of pleuritis serous, fibrinous, serozno-fibrinous, festering, hemorrhagic are
distinguished.
purulent pleuritis