Diseases of the respiratory system

Acute pneumonia

- A group of acute infectious inflammatory lung diseases of different etiology, pathogenesis and morphological characteristics
- For acute pneumonia is typical:
  - Primary defeat of respiratory departments,
  - Presence of intraalveolar exudate.

On the basis of clinical and morphological features:

- Croupous (lobar) pneumonia,
- Bronchopneumonia (focal),
- Interstitial pneumonia.

Croupous pneumonia

- Croupous pneumonia is an infectious-allergic disease.
- It is an independent nosological form.
- There are the following synonyms, reflecting the morphological features of lung lesions: lobar, fibrinous, pleuropneumonia.
- The causative agent is Streptococcus pneumoniae (pneumococci), rarely - Klebsiella pneumoniae (Friedlander's bacillus).
- Great importance in the pathogenesis has an immediate reaction of hypersensitivity.

Morphology of croupous pneumonia

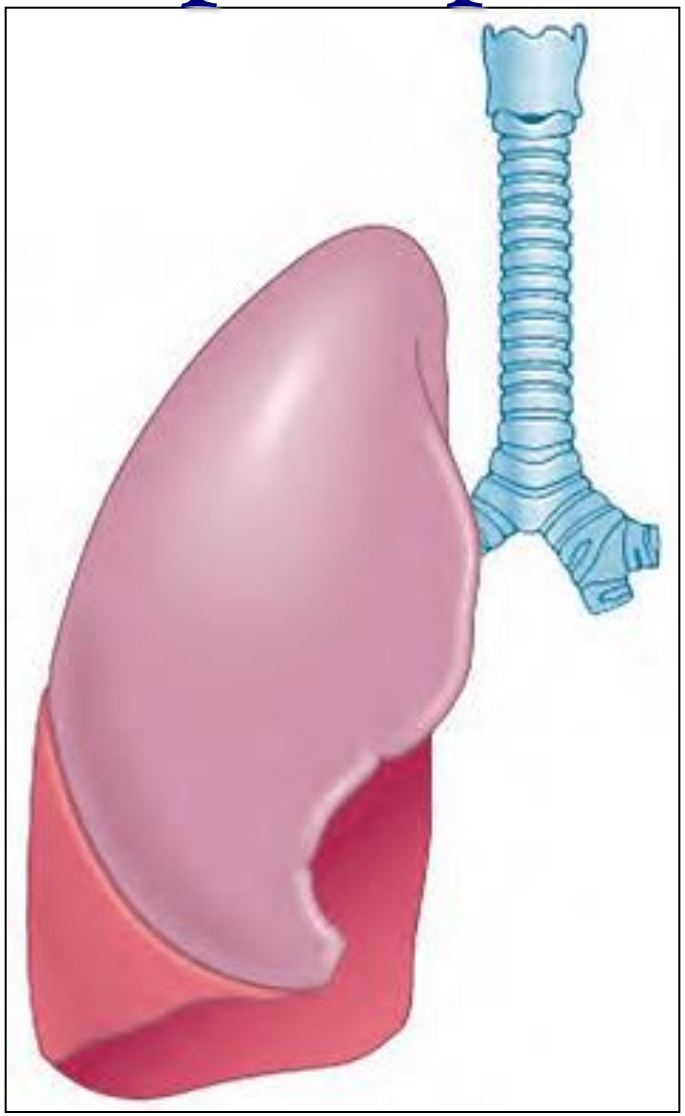
- Characterized by the defeat of the alveoli of the entire lobe, while keeping the bronchi intact.

- It is always accompanied by fibrinous pleuritis (pleuropneumonia).

- In the morphogenesis of croupous pneumonia, the following stages are distinguished:
  Tidal (microbial edema),
  Red hepatization (custody),
  Gray hepatization (custody),
  Resolution.

- This or that stage can fall out, the stages of red and gray hepatization can change places.

Croupous pneumonia



The tide stage

- It develops in the 1st day.
- Morphologically manifested:
  - Hyperemia of capillaries
  - In the alveoli serous exudate containing a large number of microbes.

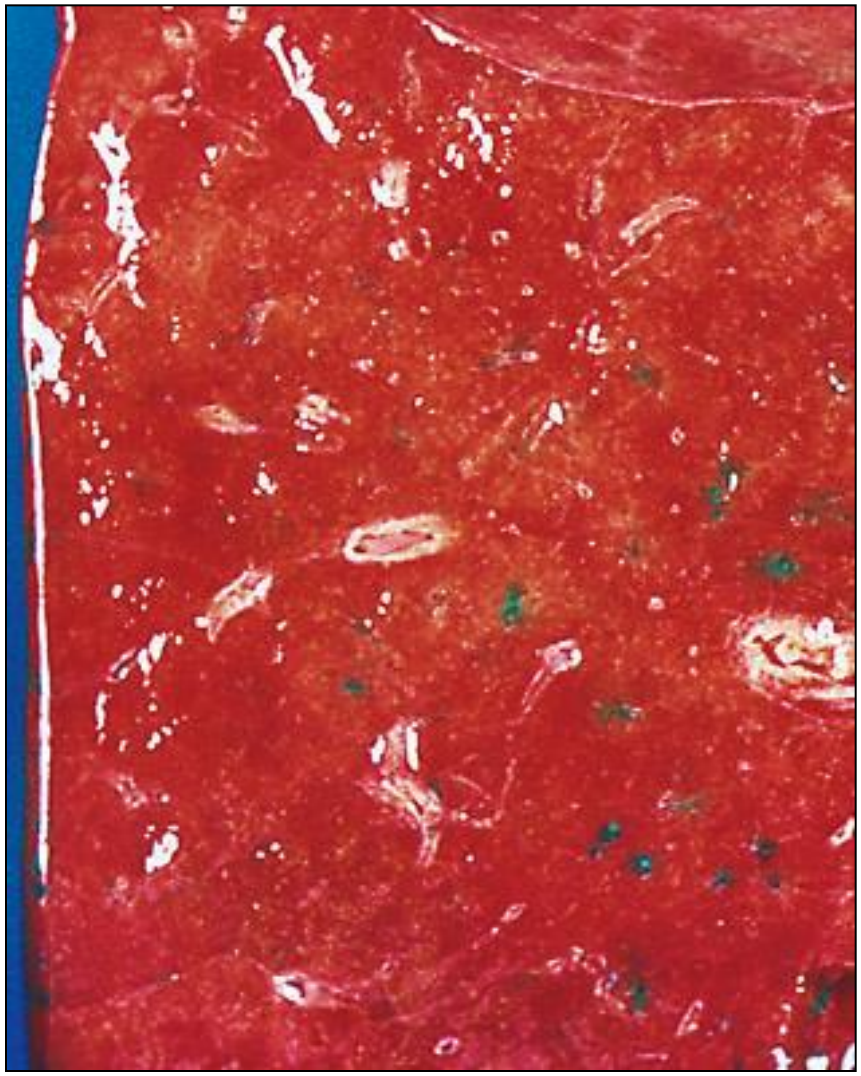
Stage of red hepatization

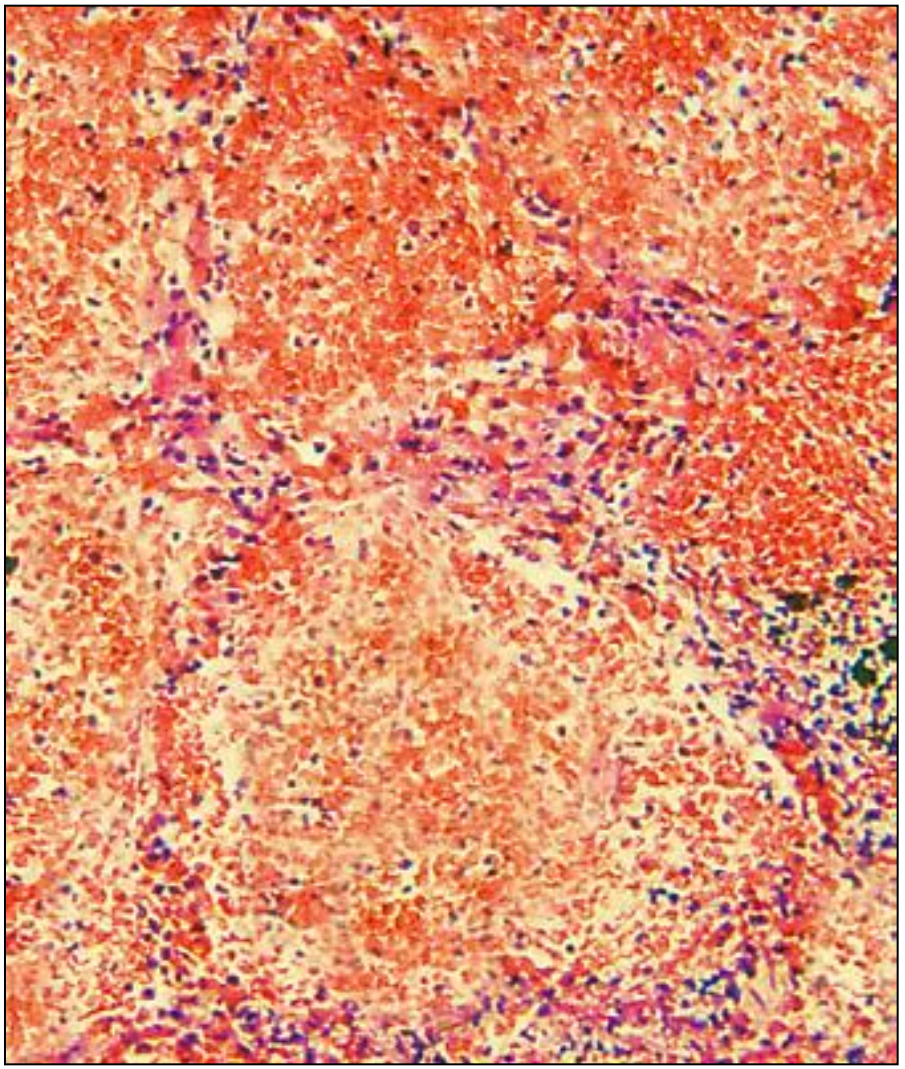
- It develops on the 2nd day.

- Macroscopic picture:
  - The affected portion of the lung is increased
  - Dense consistency (similar to the density of the liver - custody, or hepatization)
  - Red
  - On the pleura, the fibrinous superimposition (fibrinous pleuritis)

- Microscopic picture:
  - The alveoli are filled with exudate consisting of fibrin and erythrocytes
  - Capillaries of the interalveolar septa are dilated and full-blooded.

Stage of red hepatization





Fibrinous pleurisy

Stage of gray hepatization

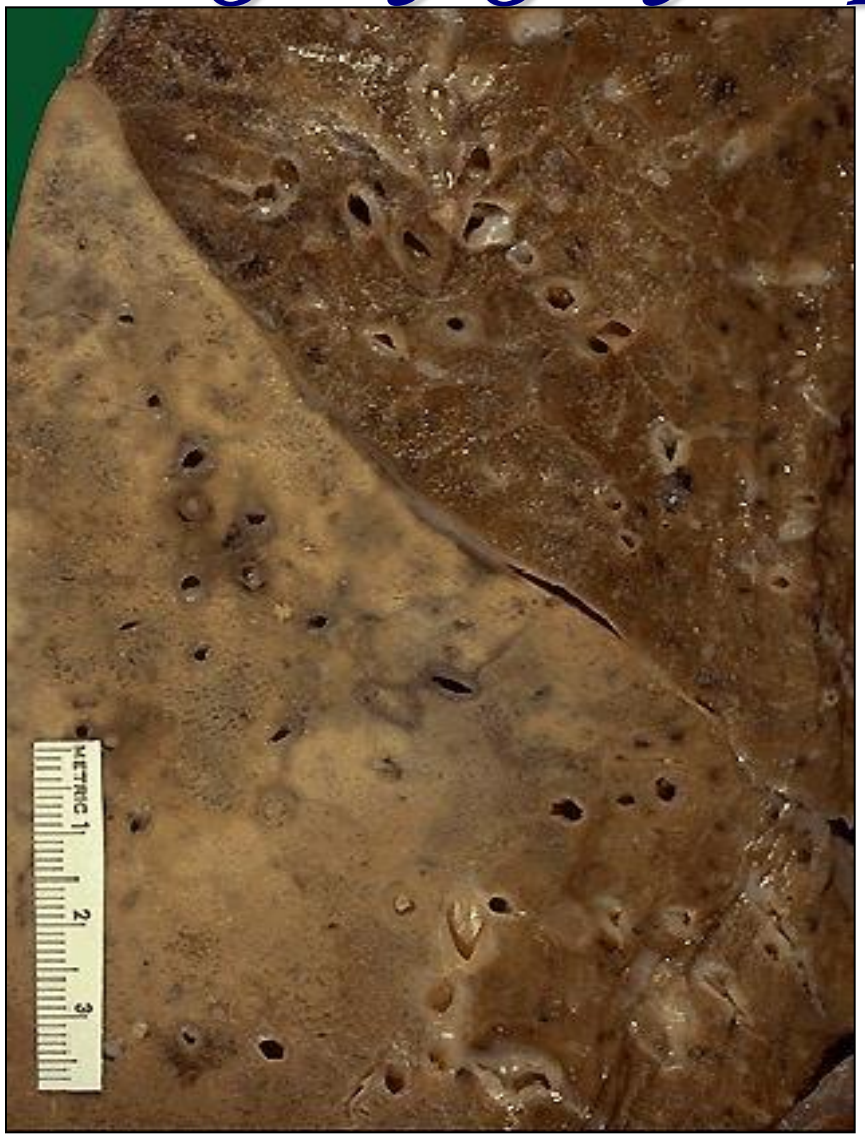
- It develops on the 4th - 6th day.

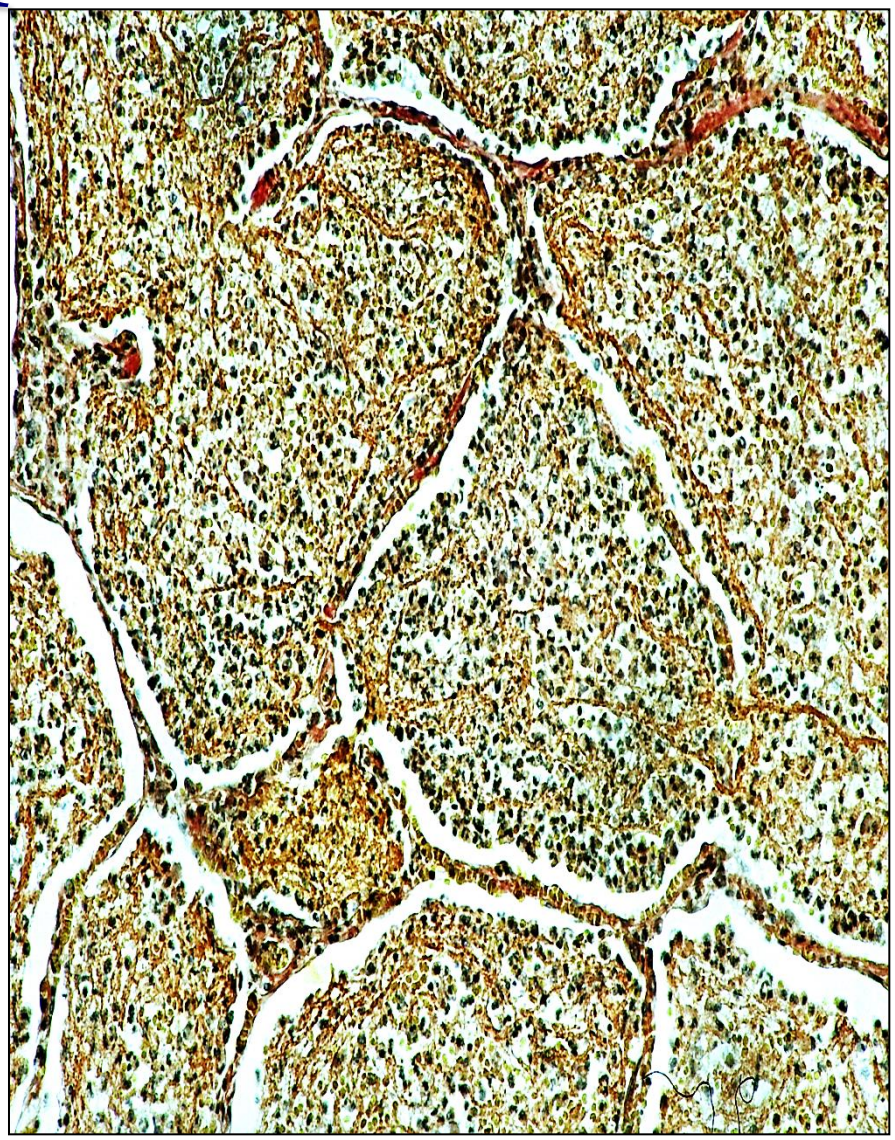
- Macroscopic picture:
  - The affected proportion is increased
  - Dense consistency
  - On a section granular
  - Gray color
  - On the pleura fibrinous overlap

- Microscopic picture:

  - The capillaries are empty
  - In the alveolar exudate - fibrin, leukocytes, macrophages

Stage of gray hepatization





Resolution stage

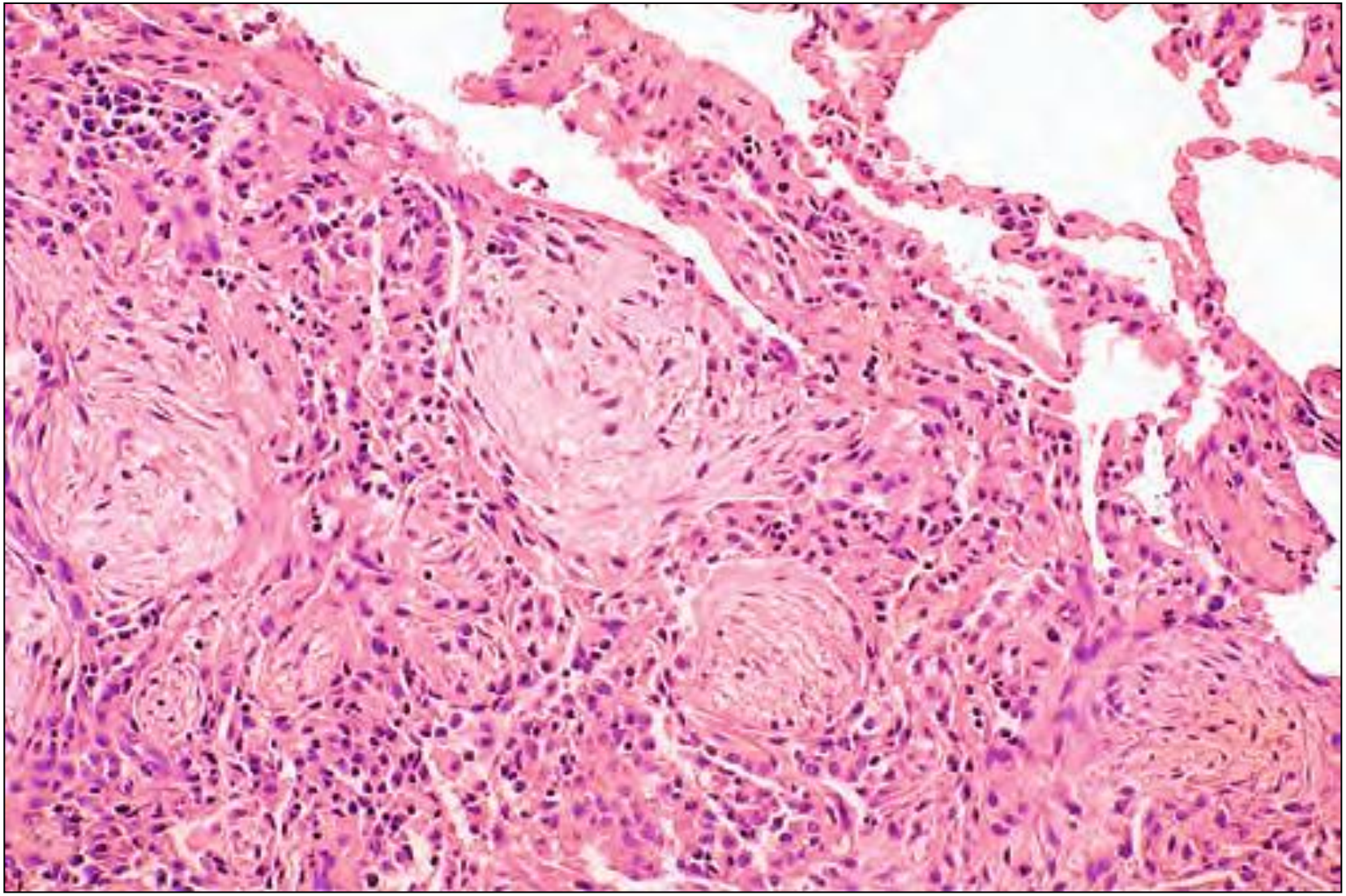
- It develops on the 9th - 11th day.

- Morphologically manifested:
  Melting and resorption of fibrinous exudates by neutrophils and macrophages.

Complications of croupous pneumonia

- Pulmonary complications:
  - Carnification (the organization of exudate in the lumen of the alveoli, the lungs become airless, fleshy).
  - Abscess of the lung (with excessive neutrophil activity).
  - Gangrene (wet).
- Extrapulmonary complications:
  - Occur with lymphogen or hematogenous spread of the infection.
  - Include purulent mediastinitis, pericarditis, peritonitis, purulent arthritis, acute ulcerative endocarditis (more often tricuspid valve), purulent meningitis, cerebral abscess.

Carnification



Causes of death in croupous pneumonia

- The lethality is about 3%.

- Death comes from acute cardiopulmonary insufficiency or purulent complications.

Lobar Fridlender's pneumonia

- More often there is an intrahospital (nosocomial) infection. Sick elderly, newborns and patients with alcoholism.

- Morphologically characteristic are:

  - Necrosis of alveolar septa
  - Formation of abscesses
  - Foci of carnification
  - Pronounced interstitial fibrosis

Lobar Fredlender's pneumonia

Bronchopneumonia

- It constitutes the bulk of acute pneumonia.
- It is polyethiologic. The most common pathogens are bacteria: pneumococci, staphylococci, streptococci, Pseudomonas aeruginosa, etc.
- It can arise as an intrahospital infection in weakened patients, is caused, as a rule, by gram-negative microorganisms (Klebsiella, Pseudomonas aeruginosa and Escherichia coli) and Staphylococcus aureus.
- More often is a complication of other diseases. Bronchopneumonia of newborns and elderly, as well as some etiological variants of bronchopneumonia (eg, legionella) can be considered as independent nosological forms.

Bronchopneumonia

- It often occurs as an autoinfection.

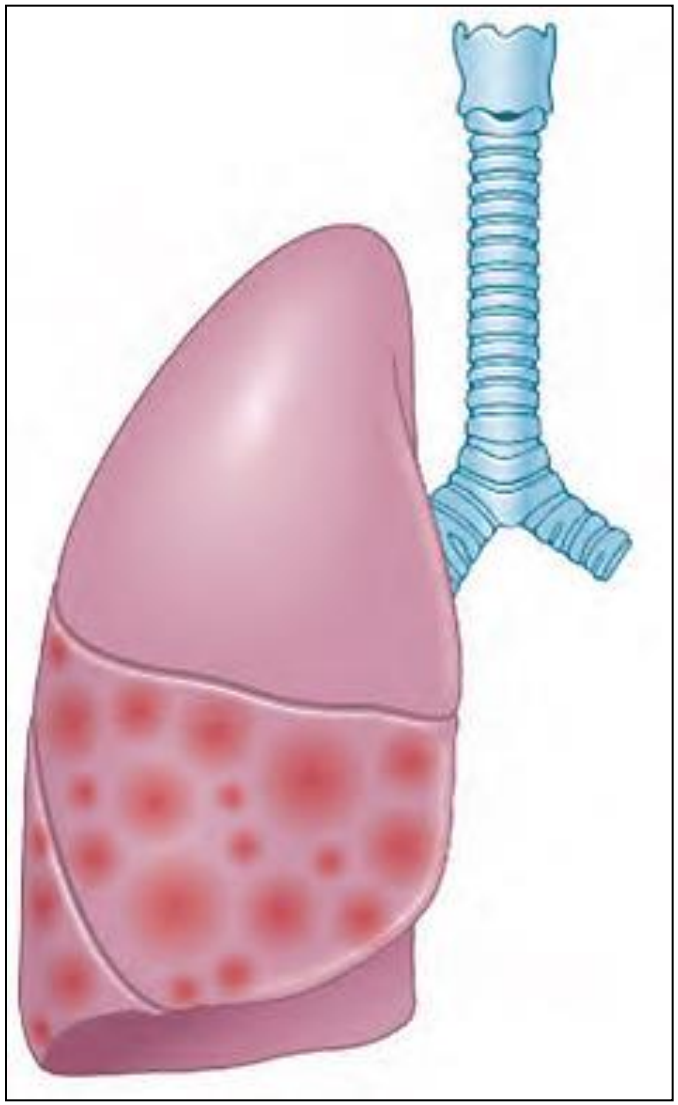
- Depending on the features of pathogenesis, autoinfection bronchopneumonia may be:

  - Aspiration
  - Hypostatic
  - Postoperative
  - Against the background of immunodeficiency

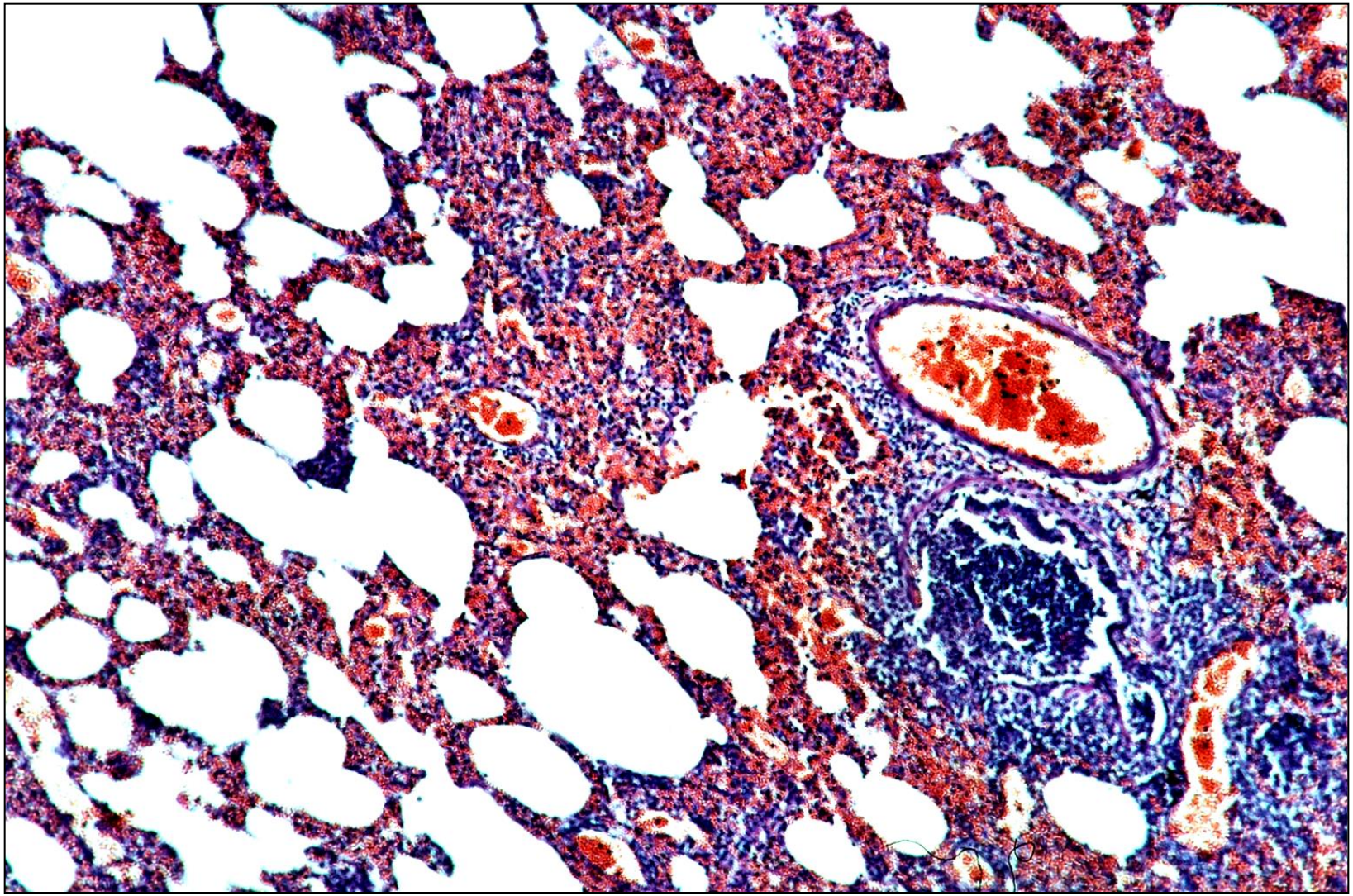
Morphology of bronchopneumonia

- Initially, the bronchi (endo-, panbronchitis) are affected.
- Inflammation to the alveoli is spreading from the bronchus wall by a descending pathway with endobronchitis or peribronchial with panbronchitis or destructive bronchiolitis.
- Exudate can be serous, purulent, hemorrhagic, mixed.
- The prevalence of the process is:
  - Acinous
  - Lobular
  - Drainage lobule
  - Segmented
  - Miliary pneumonia.

Bronchopneumonia



Bronchopneumonia



Complications of bronchopneumonia

- Cargnification.
- Formation of abscesses.
- Pleuritis with potential pleural empyema.

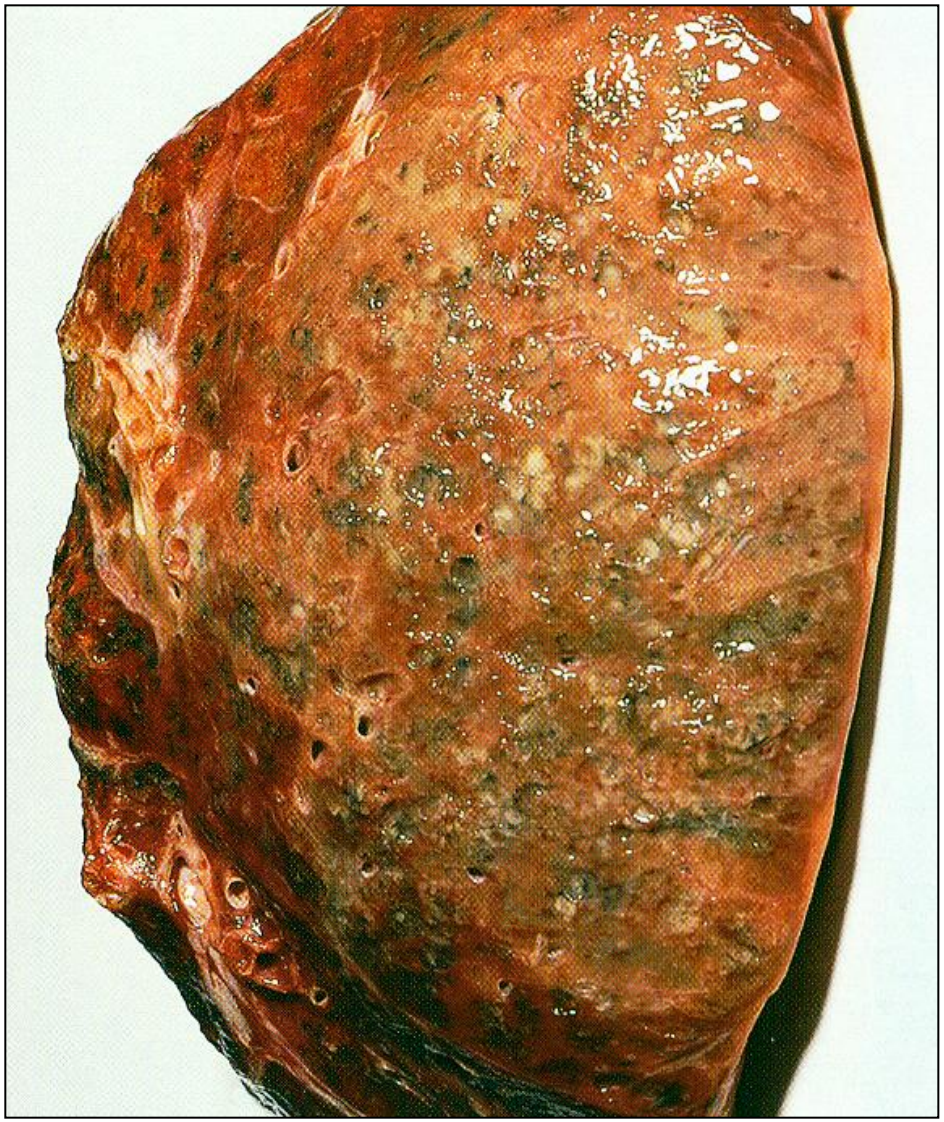
Pneumococcal bronchopneumonia

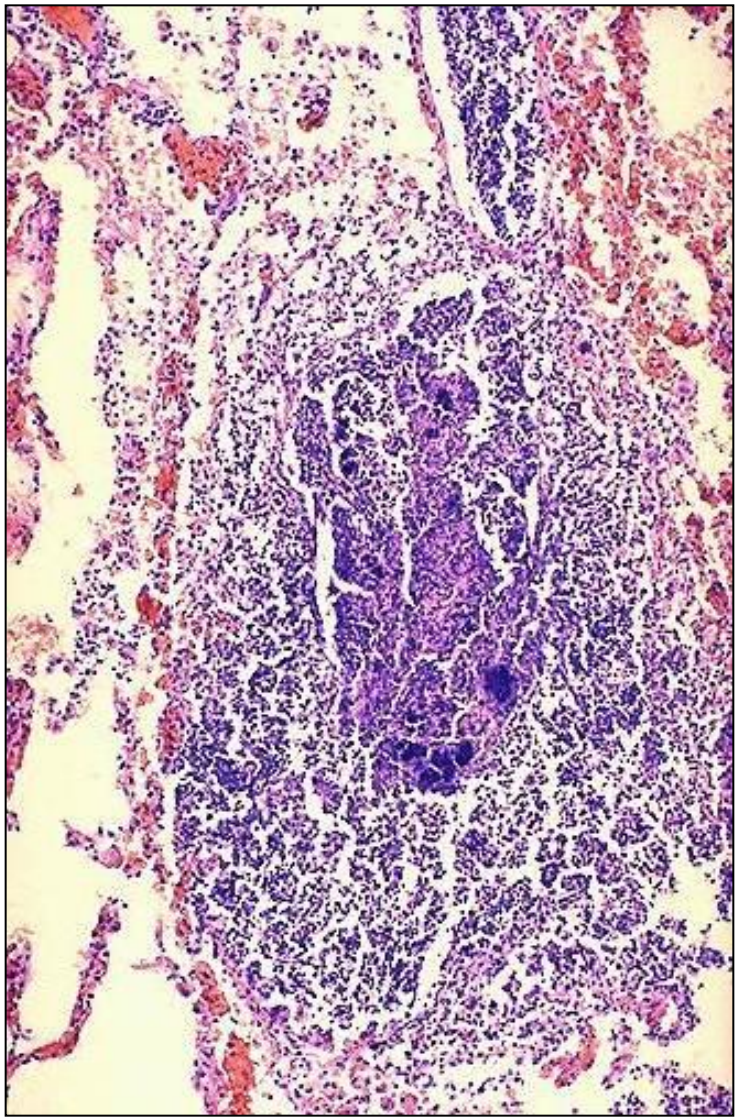
- It is more common in the elderly.
- Weakened recumbent patients, especially with cardiopulmonary pathology, develops as an autoinfection hypostatic pneumonia.
- It is often complicated by pleural empyema.

Staphylococcal bronchopneumonia

- The greatest value in the etiology is Staphylococcus aureus.
- Usually occurs as a complication of respiratory viral infections (influenza, etc.).
- It often develops in addicts with intravenous dribbling of the infection, as well as in weakened elderly patients with chronic pulmonary diseases.
- Characterized by abscess formation, the development of pleural empyema, often serves as a source of septicopyemia.

Staphylococcal bronchopneumonia

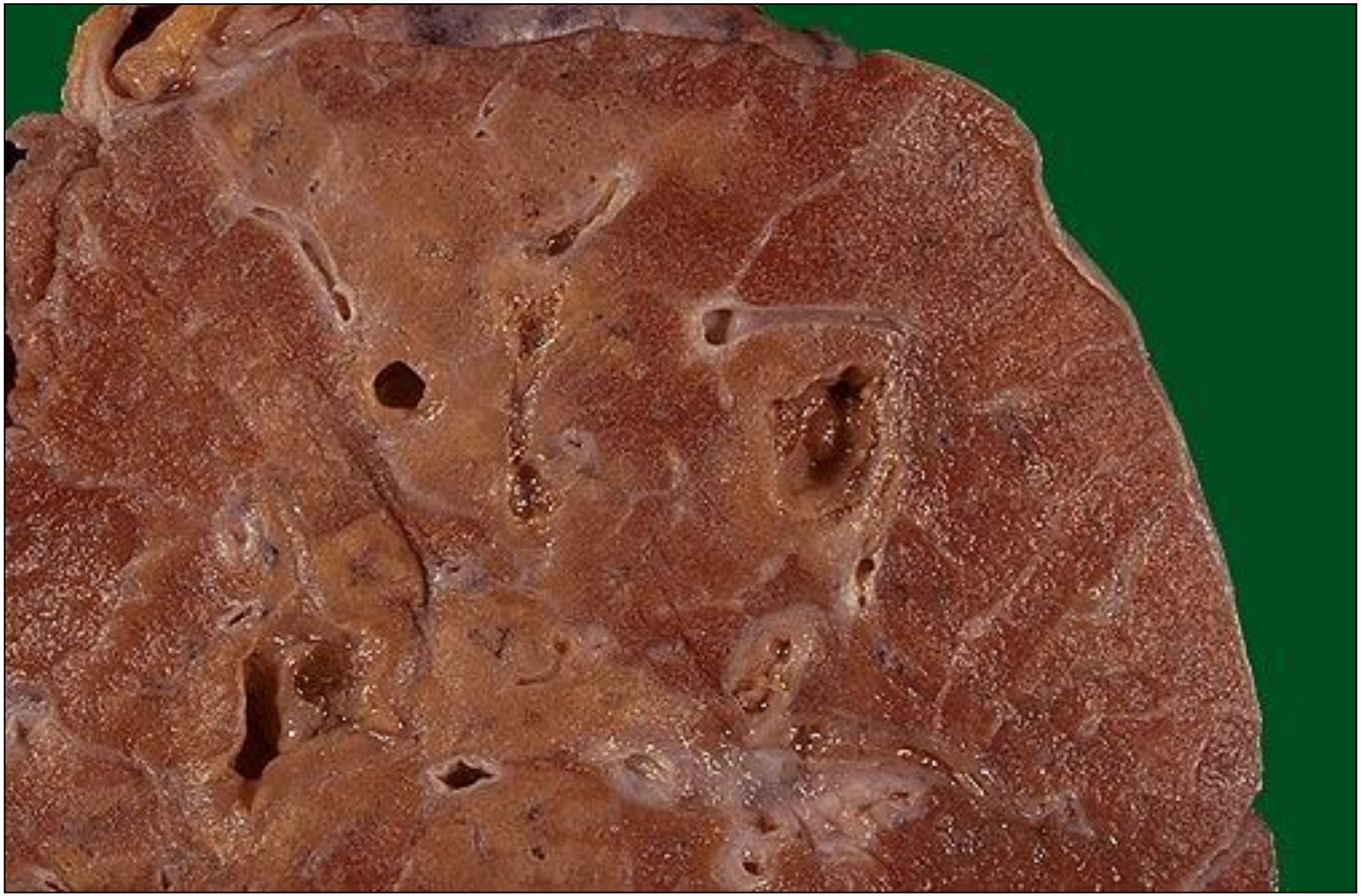




Streptococcal bronchopneumonia

- It is usually a complication of viral infections - influenza and measles.
- Characteristic of the lesion of the lower lobes.
- In a number of cases, acute abscesses and bronchiectasis occur.

Streptococcal bronchopneumonia



Pseudomonas aerobic bronchopneumonia

- One of the most common nosocomial infections.
- Characterized by abscess and pleurisy.
- When hematogenous drift of an infection into the lungs (usually from extensive festering wounds), coagulative necrosis and a hemorrhagic component are characteristic.
- The forecast is poor.

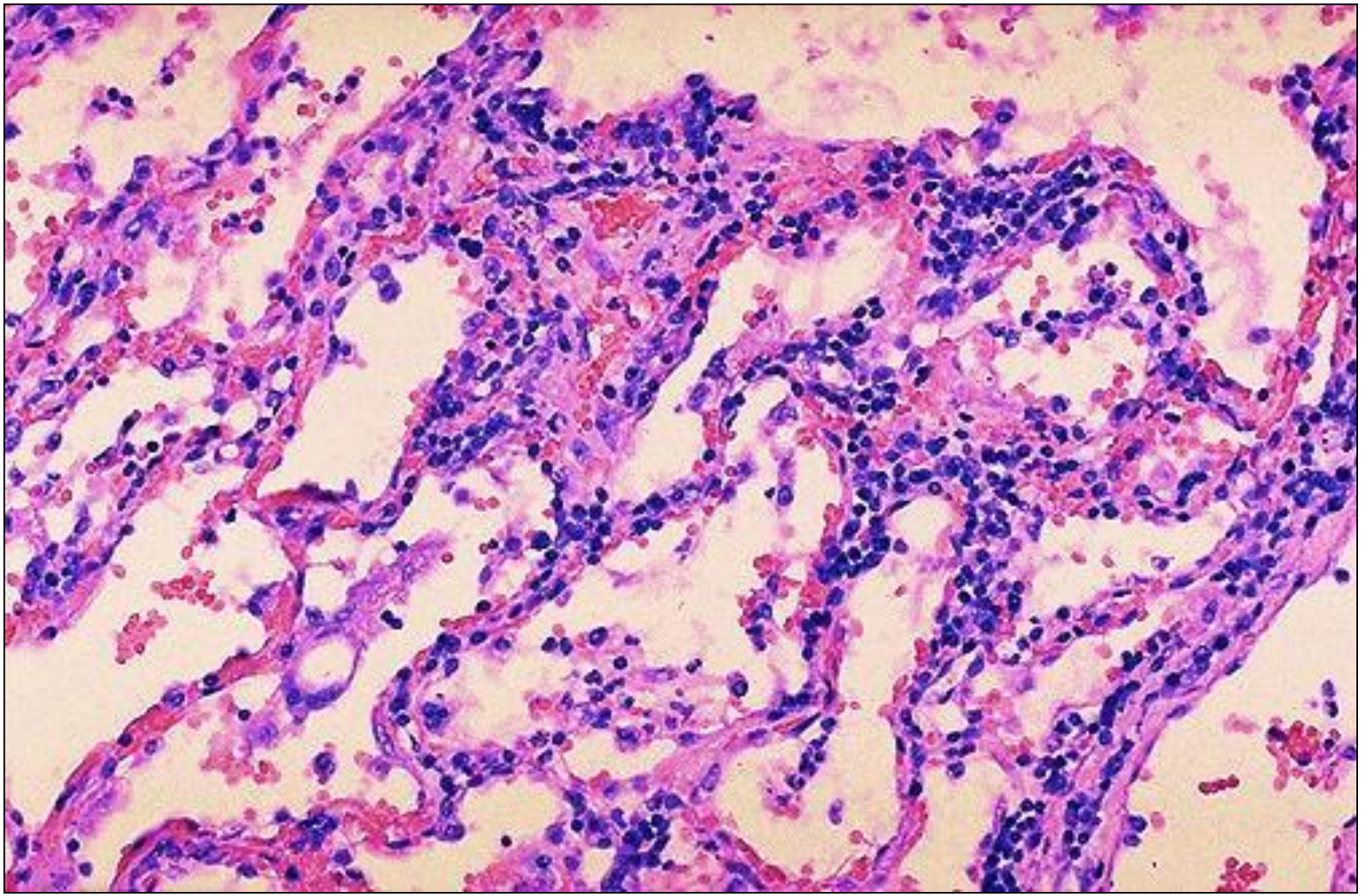
Interstitial pneumonia

- Inflammation develops mainly in the alveolar septa with secondary accumulation of exudate in the lumens of the alveoli.
- Synonyms: alveolitis, pneumonitis.
- The process can be diffuse or limited.
- It is caused by certain pathogens: viruses, fungi, mycoplasmas, chlamydia (ornithosis), rickettsia (Ku-fever-pneumomicticosis), pneumocystis.

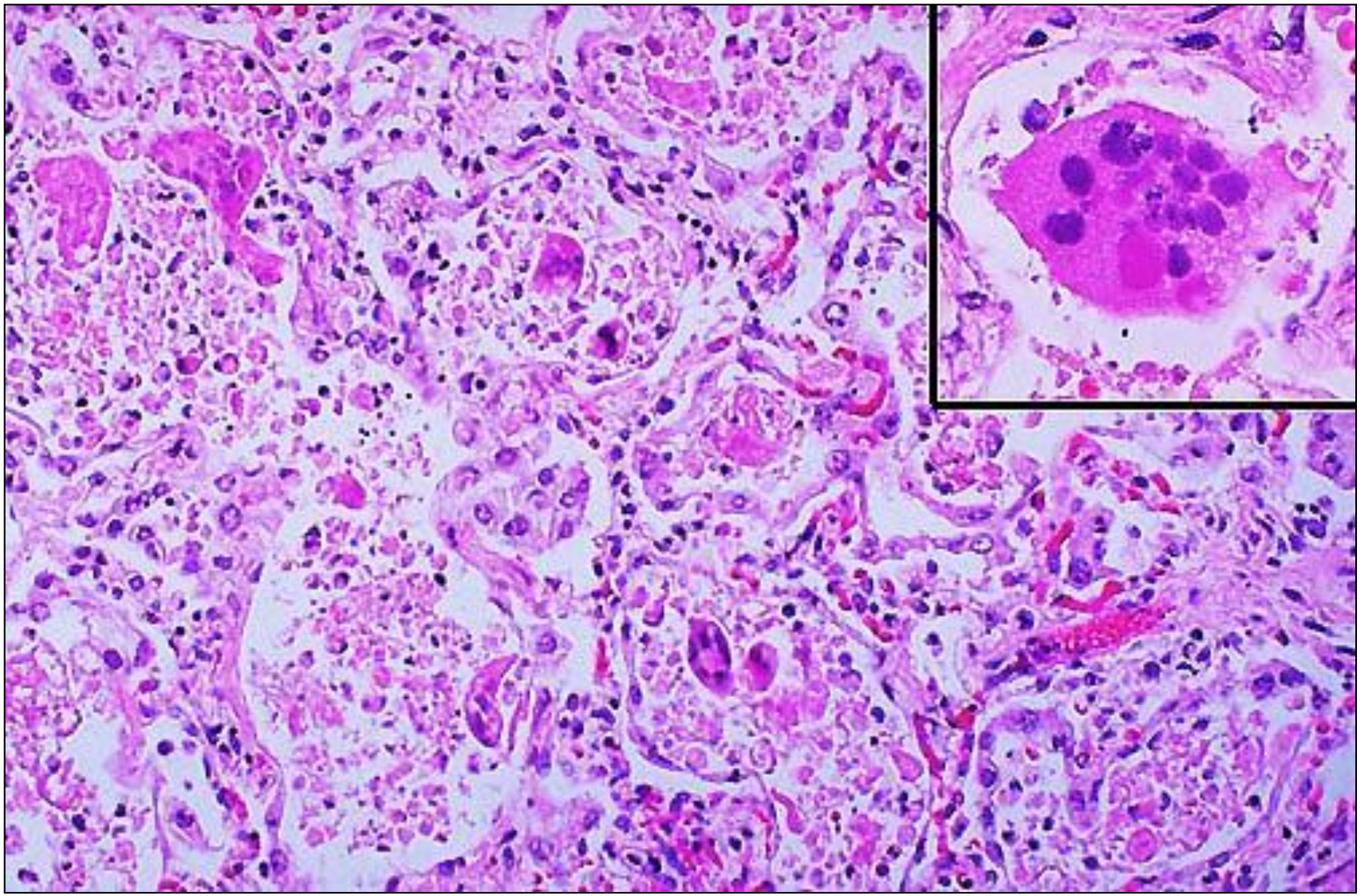
Viral pneumonia

- The most common in childhood.
- More often caused by viruses of influenza, parainfluenza, respiratory syncytial virus, adenovirus.
- Characterized by hyperplasia of the alveolar epithelium with the formation of giant cells, which differ in their appearance in various diseases, squamous metaplasia of the bronchiolar epithelium is possible.
- It is often complicated by a secondary bacterial infection

Pneumonia with influenza



Pneumonia with RS-infection



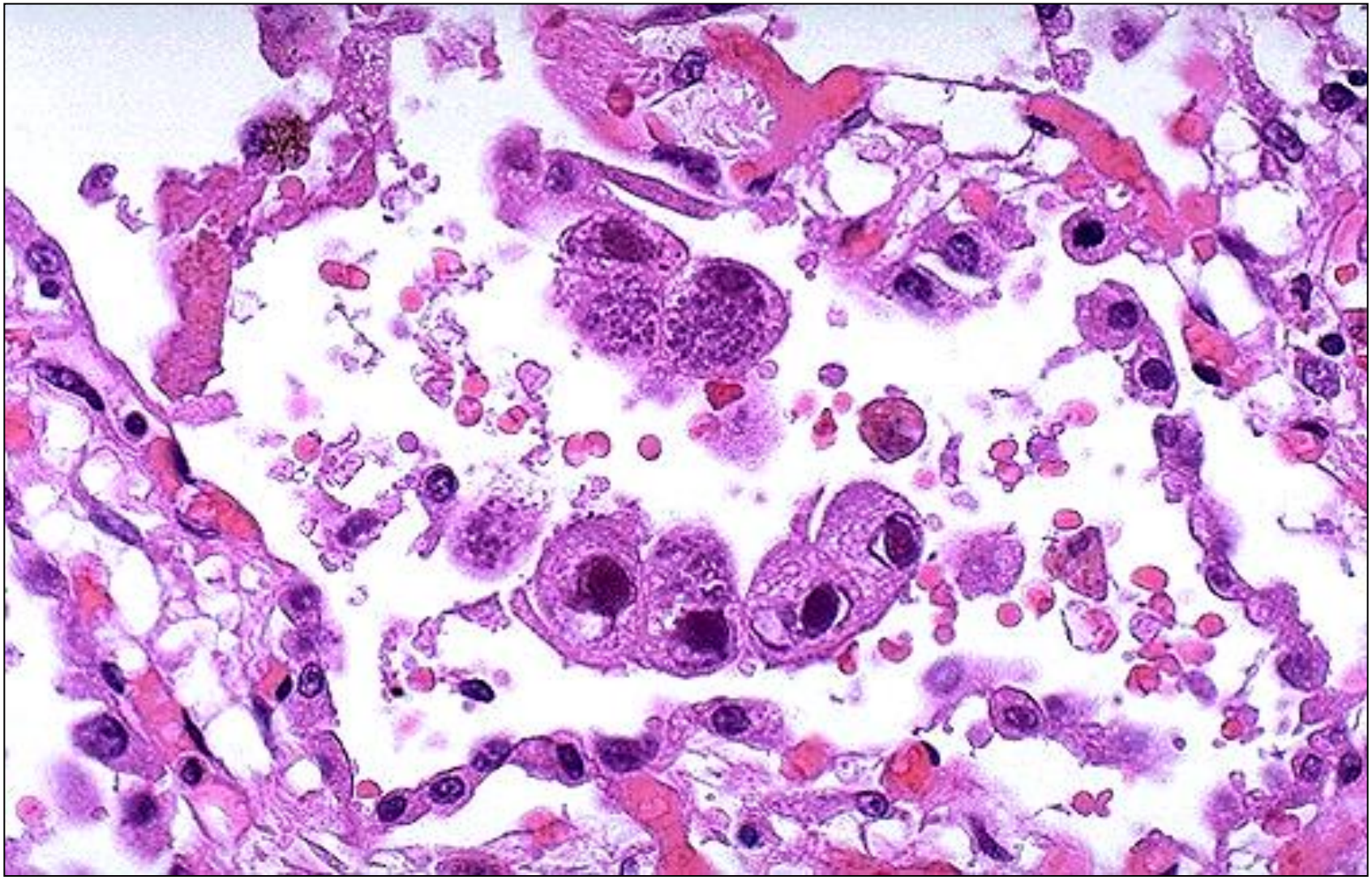
Cytomegalovirus pneumonia

- Cytomegalovirus pneumonia (opportunistic infection) the most frequently encountered in immunodeficiency states of viral pneumonia is.

- Its morphologically characteristic:

  - Mainly mononuclear infiltration of alveolar septa
  - Hyperplasia of the alveolar epithelium
  - The appearance of large cells with characteristic intranuclear inclusions
  - There is serous fluid in the lumens of the alveoli.

Cytomegalovirus pneumonia



Mycoplasmal pneumonia

- It is also known under the name "atypical pneumonia".
- One of the most common forms of non-bacterial pneumonia.
- Usually occurs in children and adolescents.
- The beginning is more imperceptible, erased than with bacterial pneumonia.

Mycoplasmal pneumonia

- Morphologically it is characterized by:
  - Inflammatory lymphoplasmocytic infiltrate of alveolar septa
  - Hyperplasia of the alveolar epithelium
  - Presence of intraalveolar hyaline membranes
  - Exudate in the lumen of the alveoli may be absent

- Often combined with changes characteristic of bronchopneumonia:
  The appearance of leukocytes in the lumen of bronchioles and alveoli.

Pneumocystis pneumonia

- Opportunistic infection, the most characteristic for patients with HIV infection.

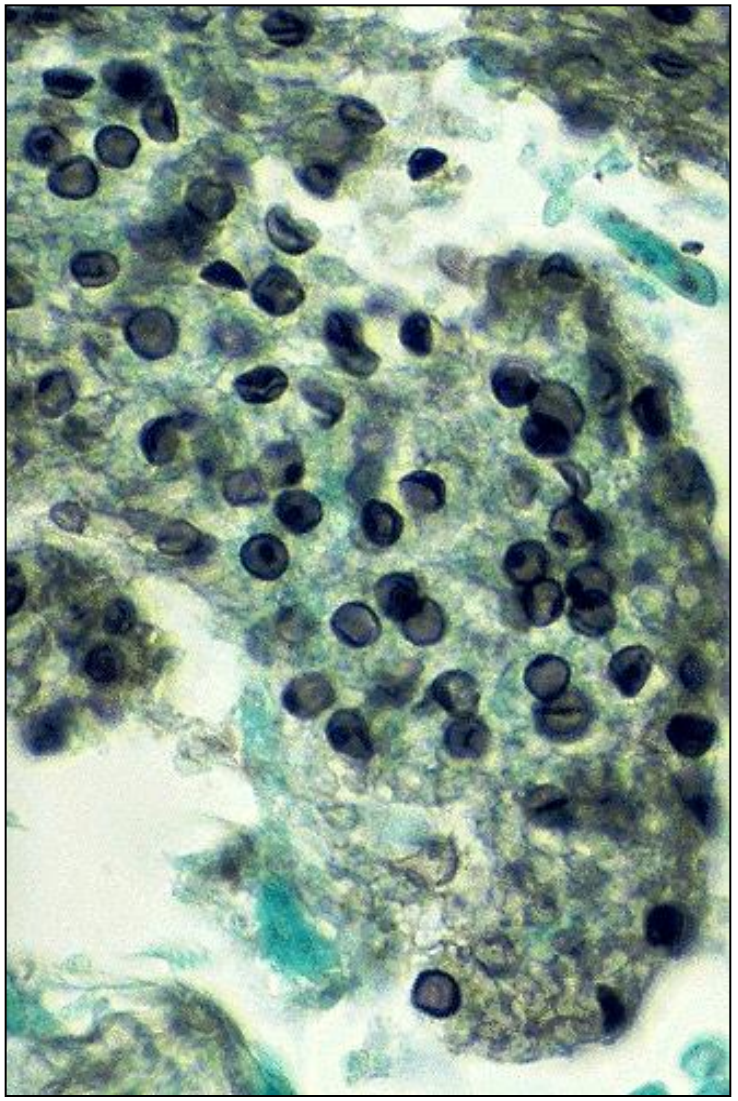
- It occurs in other forms of immunodeficiency.
  Called P. carinii - a conditionally pathogenic microorganism belonging to the simplest (some refer to its fungi).

- In people with impaired cellular immunity can develop due to the previous presence of pneumocysts in the pulmonary foci of latent infection or as a result of fresh infection.

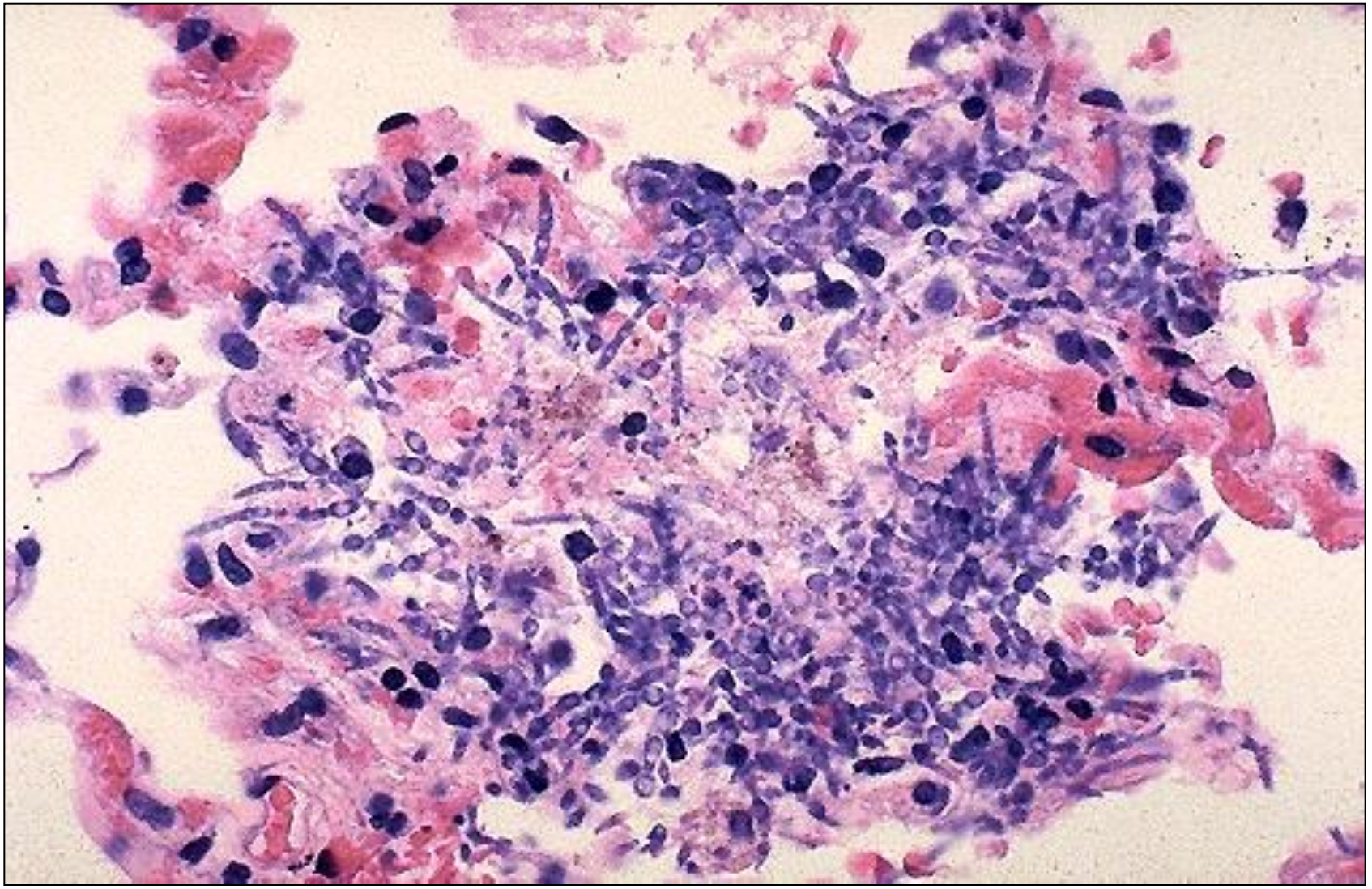
Pneumocystis pneumonia

- Morphologically characteristic are:
  - Desquamation of alveolar epithelial cells
  - Filling of alveoli with a foamy fluid containing pneumocystis
  - Lymphohistiocytic infiltration of alveolar septa with possible destruction of them
  - The fullness of the capillaries
- It can take the form of a mixed infection with the addition of another flora (fungi, cytomegalovirus, cocci, mycobacteria, etc.).

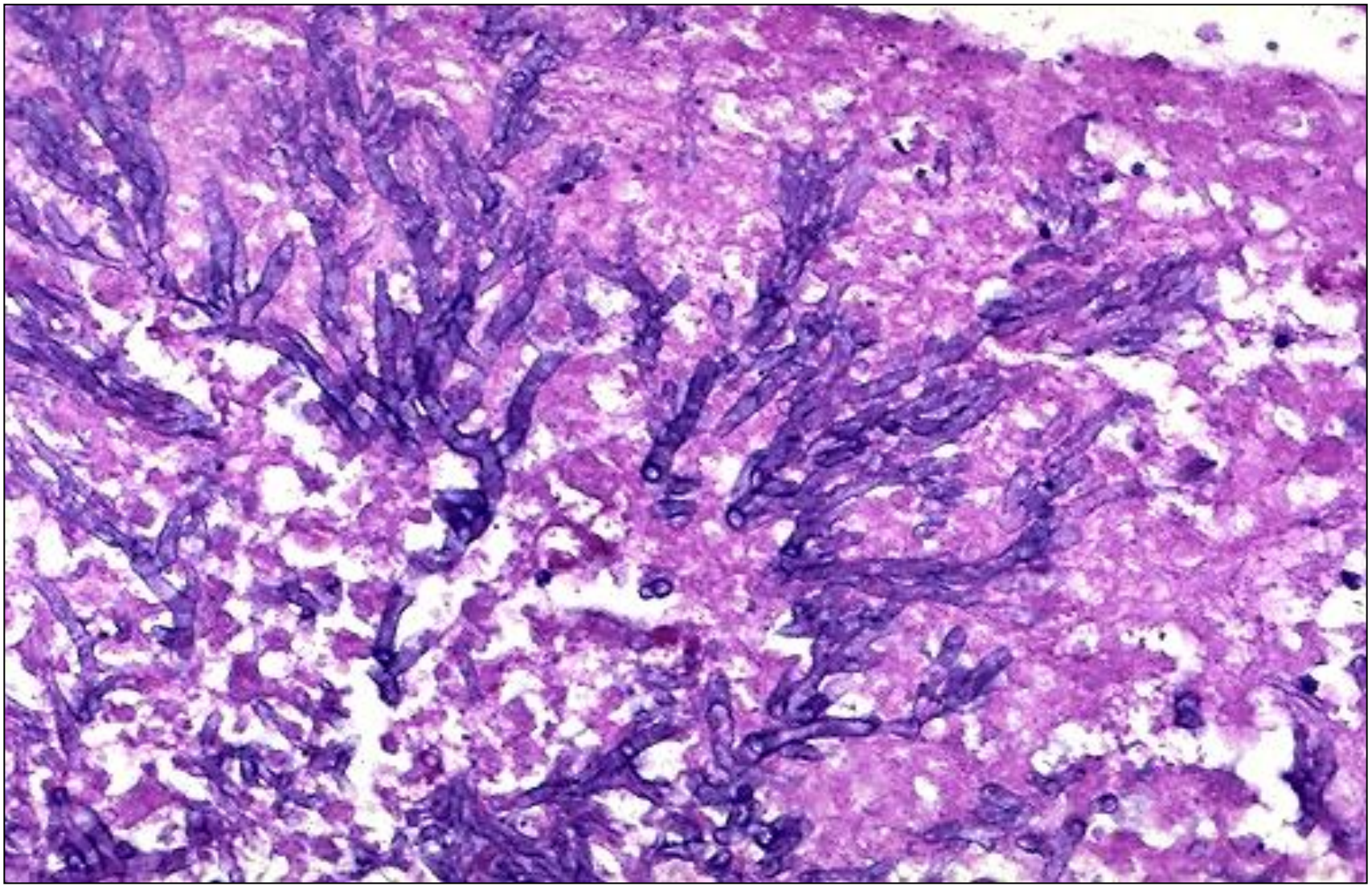
Pneumocystis pneumonia



Candida pneumonia



Aspergillus pneumonia



Chronic nonspecific lung diseases (CNLD)

- Chronic non-specific lung diseases (CNLD) is a group of lung diseases of various etiology, pathogenesis and morphology, characterized by the development of chronic productive cough and difficulty breathing, which are not associated with specific infections, primarily pulmonary tuberculosis.

- For all chronic obstructive pulmonary disease, hypertension of the small circulation and pulmonary heart develops.

Classification of CNLD

- Depending on the morphofunctional features of the damage air-conducting and respiratory sections of the lungs, X-ray diffraction:
  - Obstructive CNLD
  - Restrictive CNLD

- In the late stages of most pulmonary diseases there is a combination of obstructive and restrictive components.

Obstructive CNLD

- At the heart of obstructive lung diseases is a violation of the drainage function of the bronchi with partial or complete obstruction, resulting in increased resistance to air passage.

- Obstructive CNLD include:

  - Chronical bronchitis
  - Bronchoectatic disease
  - Chronic obstructive pulmonary emphysema
  - Bronchial asthma

Restrictive CNLD

- Restrictive lung diseases are characterized by a decrease in the volume of the pulmonary parenchyma with a decrease in the vital capacity of the lungs.

- At the heart of restrictive pulmonary diseases is the development of inflammation and fibrosis in the interstitial respiratory departments, which is accompanied by progressive respiratory failure.

- Restrictive include chronic interstitial lung diseases, represented by various forms of fibrosing alveolitis, or pneumonitis.

Mechanisms of development of CNLD

- **Bronchytogenic mechanism:**
  The basis is a violation of the drainage function of bronchi and bronchial patency.
  Diseases, combined by this mechanism, are chronic obstructive pulmonary diseases.

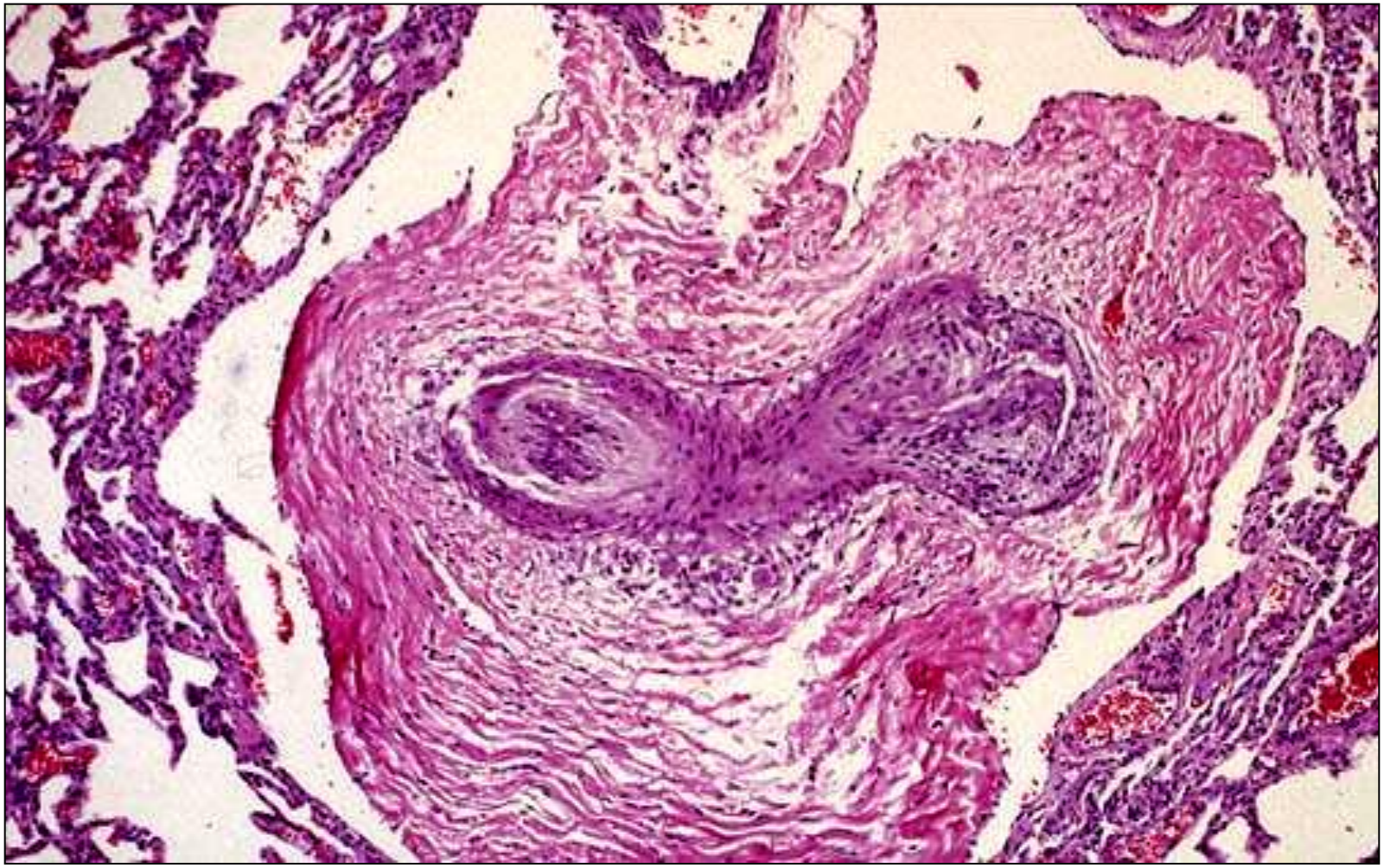
- **Pneumonia mechanism:**
  It is associated with acute pneumonia and its complications (acute abscess, carnification).
  It leads to the development of chronic abscess and chronic pneumonia.

- **Pneumonitogenic mechanism:**
  It is associated with the alveolitis (pneumonitis).
  It determines the development of chronic interstitial lung diseases.

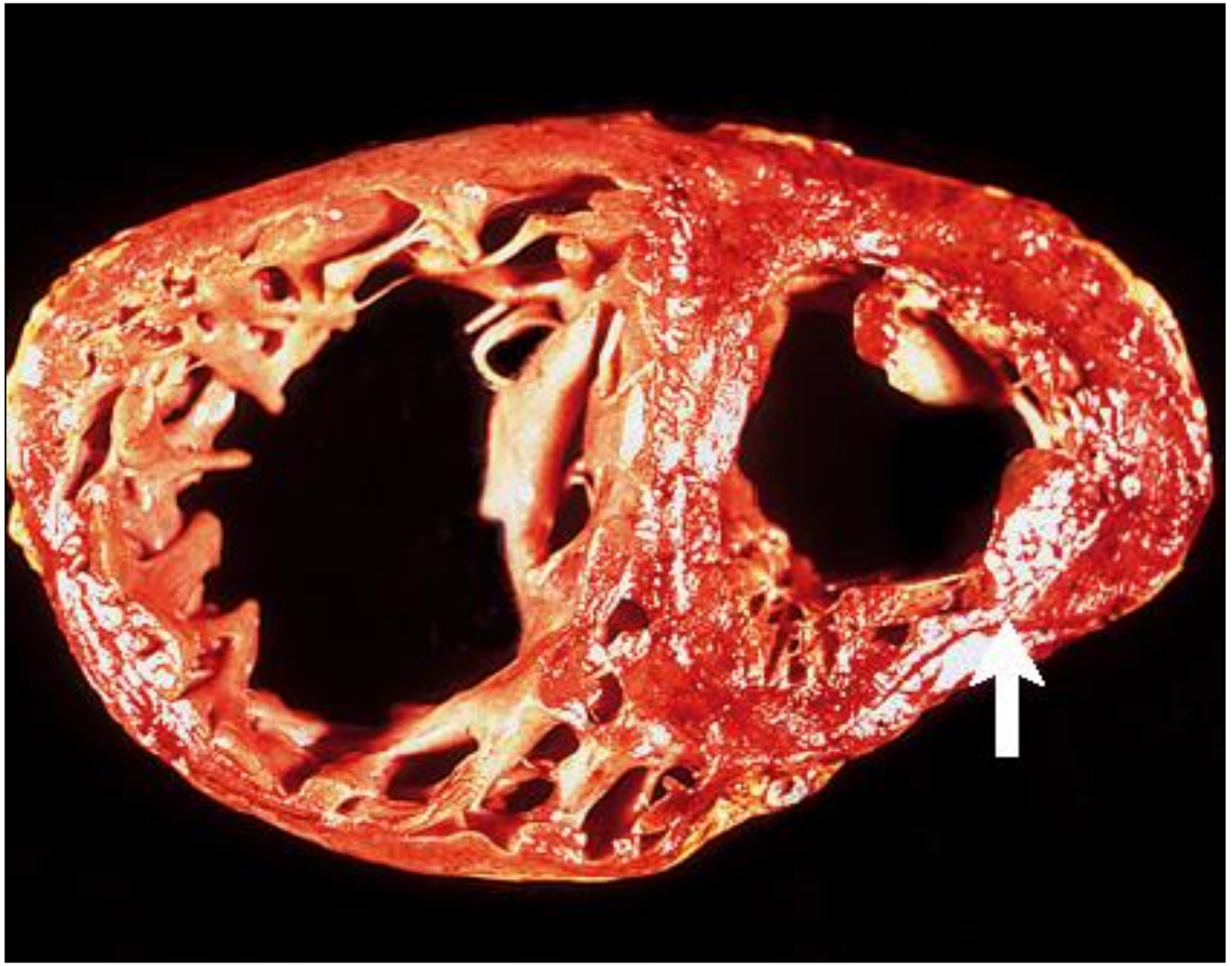
Mechanisms of development of CNLD

- As a result, all three mechanisms for the development of CNLD lead to:
  - Pneumosclerosis
  - Secondary pulmonary hypertension
  - Hypertrophy of the right ventricle of the heart (pulmonary heart)
  - Cardiopulmonary insufficiency.

Sclerosis and reduction of capillaries with pulmonary hypertension



Pulmonary heart



Chronical bronchitis (CB)

- Chronic bronchitis is a disease characterized by excessive production of mucus by the bronchial glands, which leads to the appearance of a productive cough with a duration of at least 3 months (annually) for at least 2 years (WHO criteria).

- Smoking is the most important etiological factor of chronic bronchitis.

Classification of CB

- By prevalence:
  - Local
  - Diffuse
- Depending on the presence of bronchial obstruction:
  - Obstructive
  - Non-obstructive
- By the nature of catarrhal inflammation:
  - Simple catarrhal
  - Mucopurulent

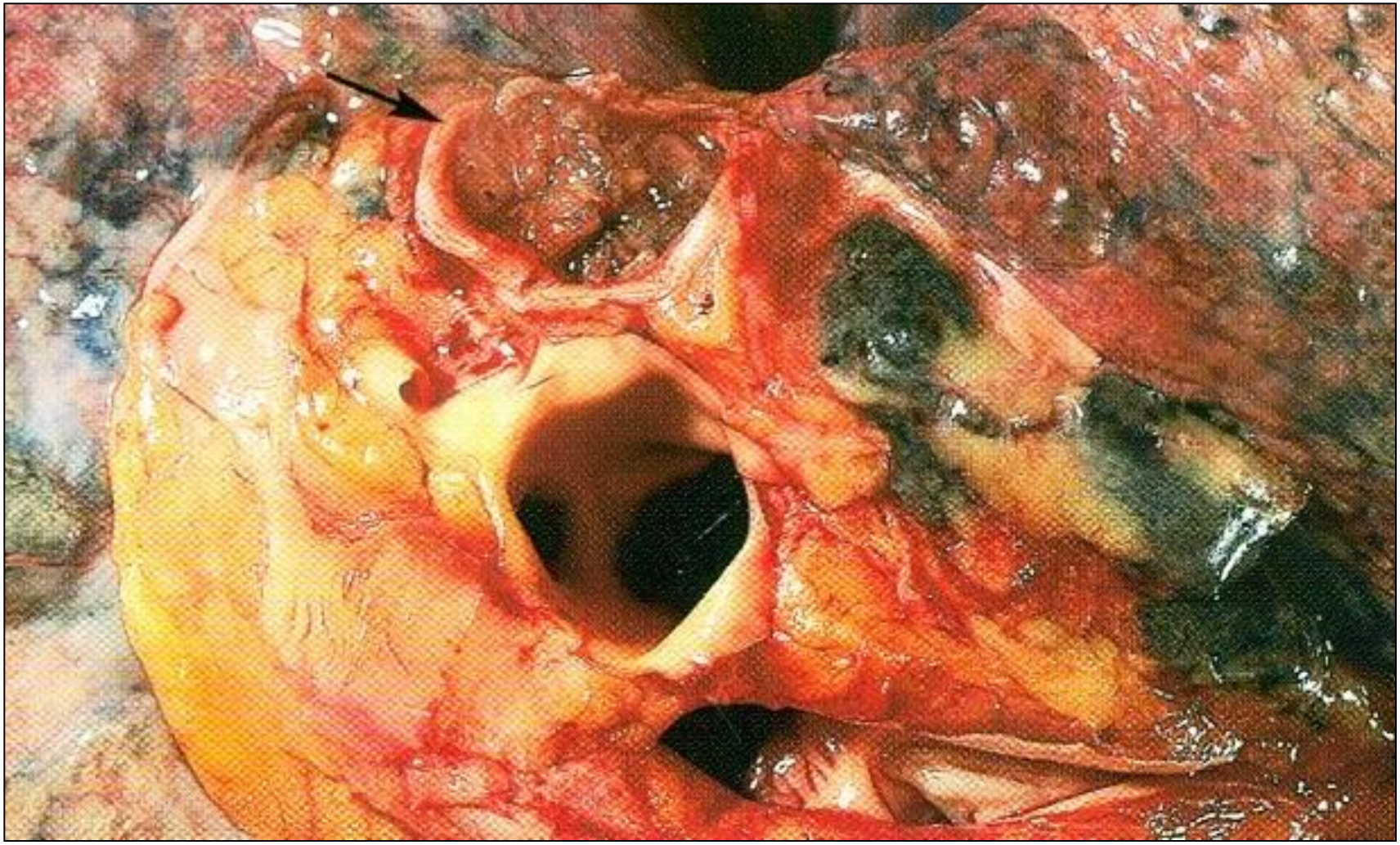
Simple catarrhal CB

- Morphologically it is characterized by:
  - Hyperplasia of the mucous glands in the mucosa and submucosal membranes of the bronchi
  - An increase in the thickness of the submucosal layer of the bronchi
  - Filling the lumens of bronchi with thick viscous mucus.

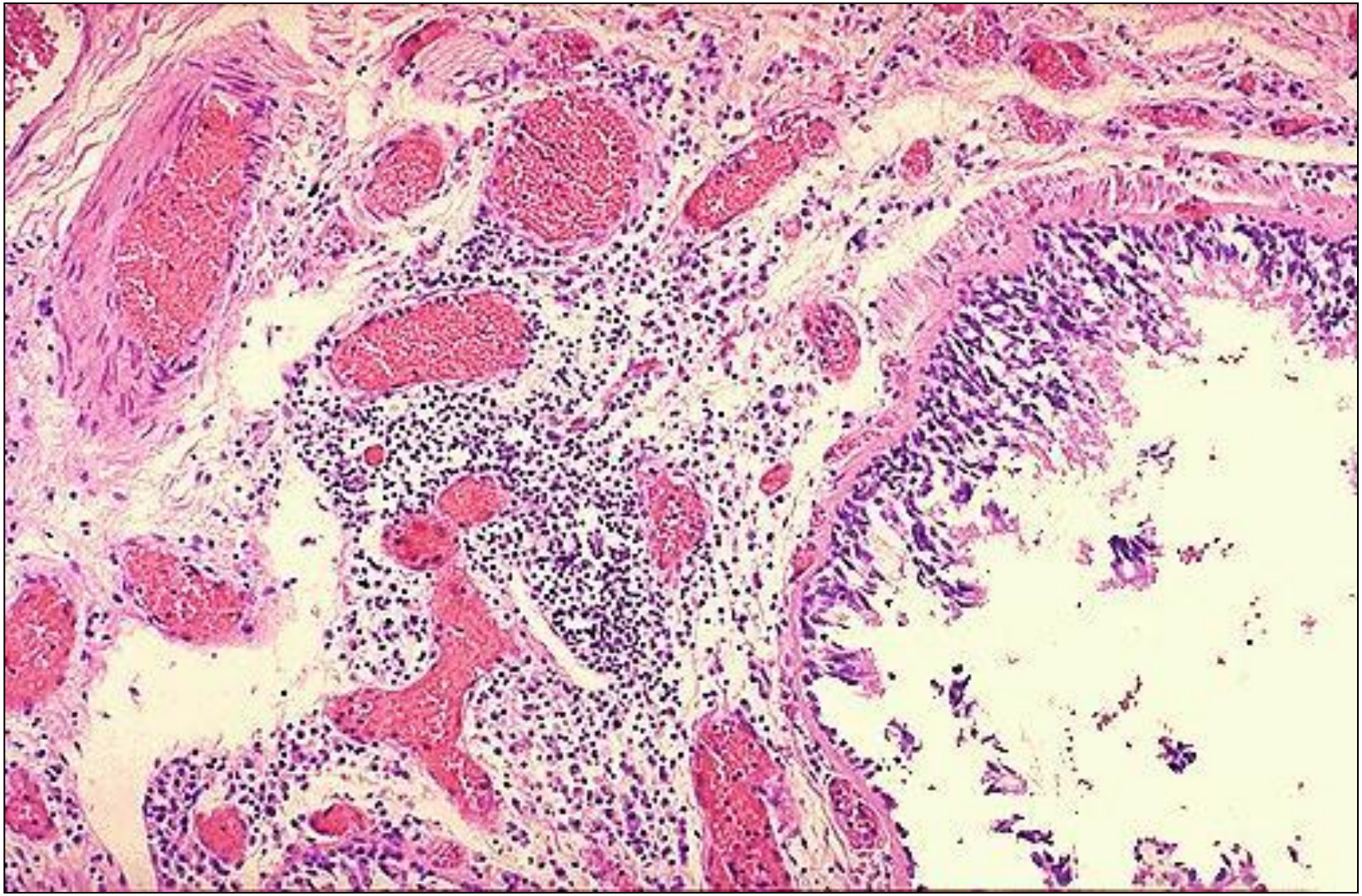
Mucopurulent CB

- Morphologically it is characterized by:
  - A picture of purulent endo- or panbronchitis
  - Focal desquamation of the prismatic epithelium of the bronchi
  - An increase in the number of goblet cells
  - Foci of metaplasia of prismatic epithelium in multilayered flat epithelium
  - Proliferation of coarse fibrous connective tissue around the bronchi
  - In the stage of remission purulent exudate is replaced by mucous.

Mucopurulent CB



Mucopurulent CB



Bronchoectatic disease

- Bronchoectatic disease is characterized by a combination of a characteristic morphological substrate - pronounced bronchiectasis and a certain extrapulmonary symptom complex due to respiratory hypoxia and the development of hypertension in a small circulatory system.

- In patients, the fingers become the shape of drum sticks, the nails are the shape of watch glasses, warm cyanosis, right ventricular hypertrophy and pulmonary heart development are characteristic.

Bronchiectasis

- Bronchoectasis is a persistent pathological enlargement of one or more bronchi containing cartilage plates and mucous glands, with destruction of the elastic and muscular layers of the bronchial wall.

- The development of bronchiectasis and bronchiectasis is often pathogenetically associated with complications of measles and a severe form of influenza due to protrusion of the bronchus wall at the site of the most pronounced inflammatory process, more often with coughing thrusts.

- By origin, bronchiectasis can be congenital and acquired.

- Acquired bronchiectasis, developed against the background of chronic bronchitis, can be considered a morphological substrate of bronchiectasis.

Morphology of bronchiectasis

- Macroscopic picture:

  Saccinated bronchiectasis (at the level of the proximal bronchi, including bronchi of the 4th order);

  Cylindrical bronchiectasis (at the level of bronchi 6 - 10th order).

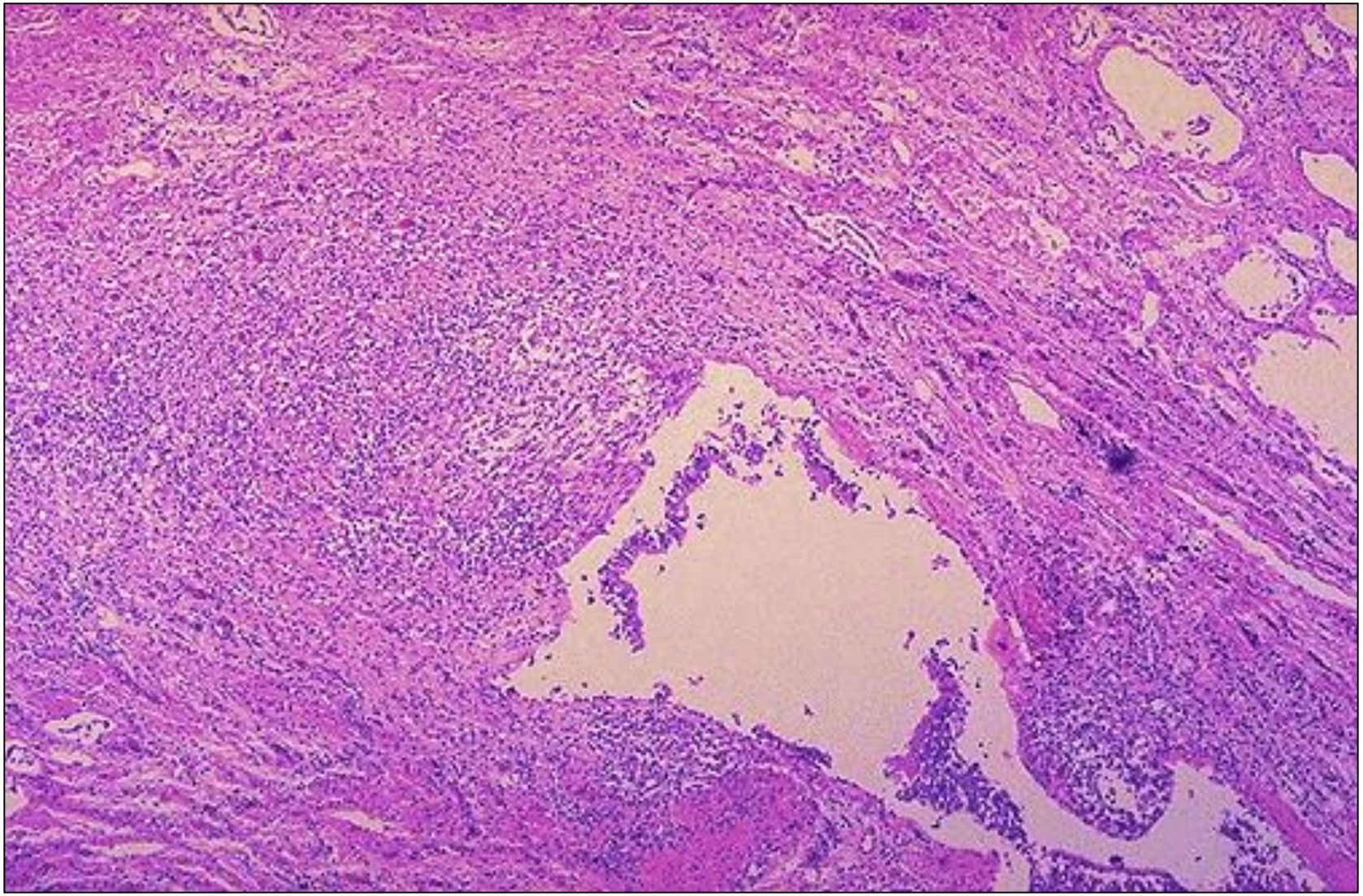
- Microscopic picture:

  In the wall of bronchiectasis, chronic purulent inflammation with destruction and atrophy of structural elements and sclerosis is observed;

  In the adjacent pulmonary tissue - fibrosis fields, foci of obstructive emphysema.

Saccinated bronchiectasis

Bronchiectasis



Complications of bronchiectasis

- Pulmonary bleeding.
- Abscesses of the lung (bronchiectasis abscesses).
- Empyema of the pleura.
- Chronic cardiopulmonary insufficiency.
- Secondary amyloidosis (AA-amyloidosis).

Bronchial asthma (BA)

- Bronchial asthma - a disease which is based on the spasm of the small bronchi and bronchioles, manifested attacks expiratory dyspnea.

- The cause of this spasm are allergic reactions to exogenous allergens: animal and vegetable dust, smoke, various smells, fogs, high humidity.

- Depending on the features of etiopathogenesis, two forms of bronchial asthma are distinguished:
  - Atopic
  - Infectious-allergic

Atopic BA

- Atopic form of bronchial asthma often begins in childhood.
- The pathogenesis is based on the reactions of the immediate reaction of hypersensitivity, which are manifested by a spasm of smooth muscles of small bronchi, increased secretion of their mucous membrane and impaired patency of the bronchi.
- In patients, serum IgE levels usually increase, a positive skin test with the antigen that causes the disease is noted.

Infectious-allergic BA

- At the heart of the infectious allergic form of bronchial asthma is a similar mechanism that develops against the background of acute or chronic diseases of the respiratory system caused by infectious agents.

- However, with this form of bronchial asthma, the level of serum IgE is not increased, and skin tests are always negative.

Morphology of BA

- It is necessary to distinguish acute morphological changes that develop during an attack of bronchial asthma and chronic changes caused by frequent attacks and a prolonged course of the disease.

- Morphological changes in atopic and infectious-allergic forms of the disease, as a rule, are identical.

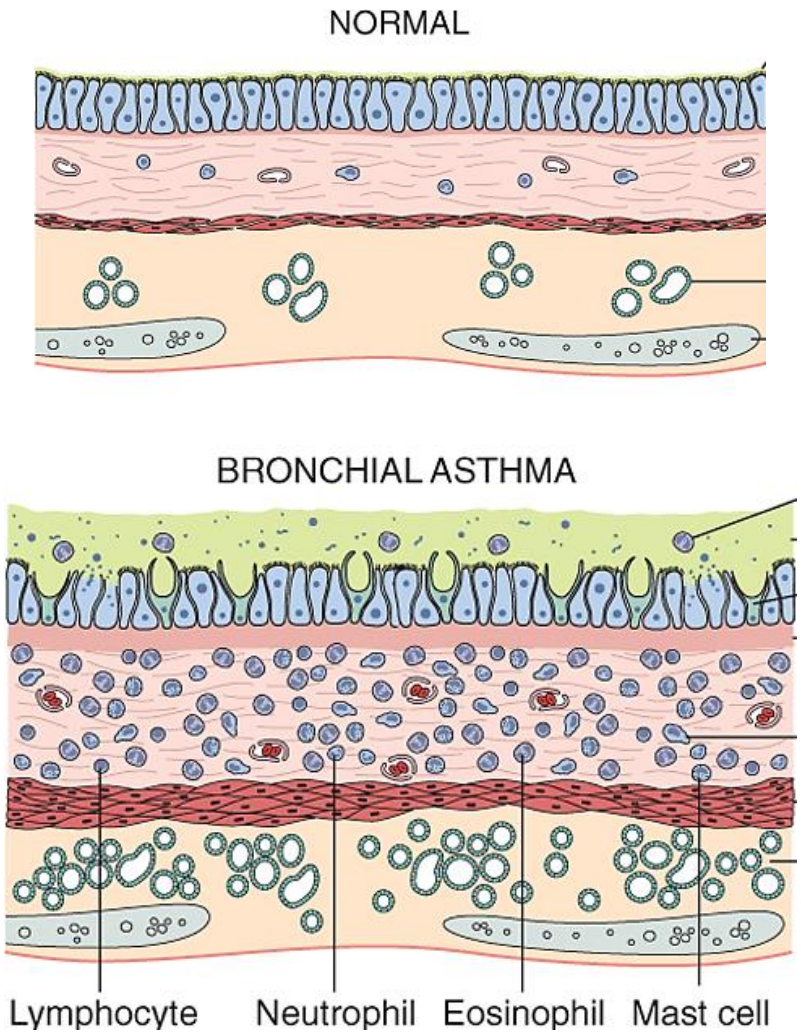
Acute changes in BA

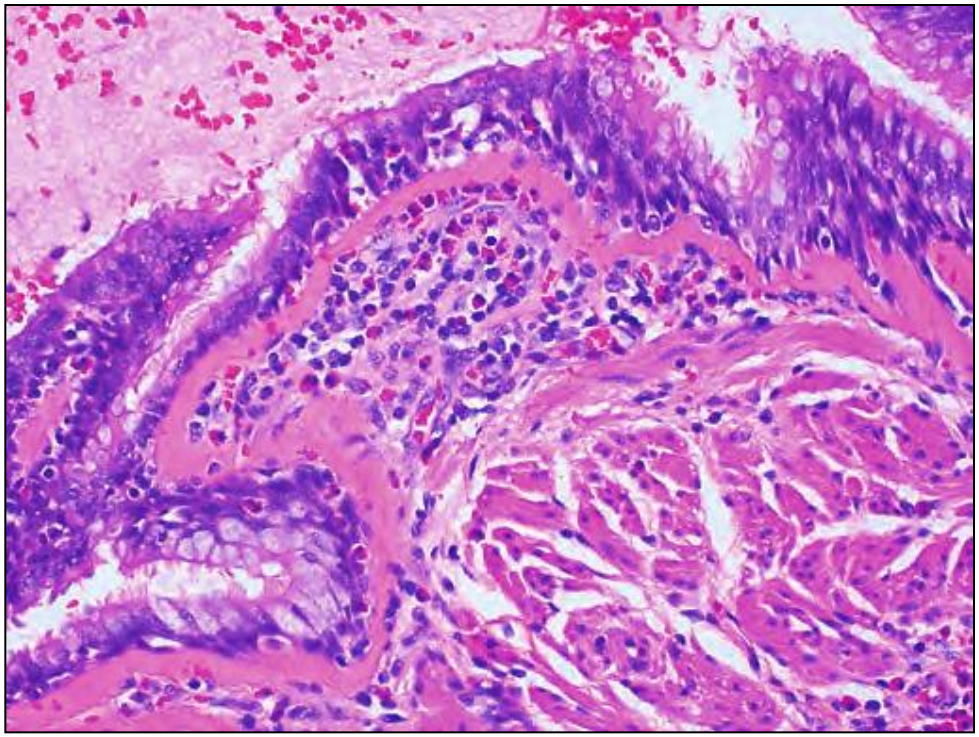
- During an attack of bronchial asthma due to a spasm of smooth muscle fibers of the walls of the bronchi, their lumens take a stellate appearance.
- There is an acute edema of the mucous and submucous layers of the bronchi and their infiltration by the lobrocites, basophils, eosinophils, lymphoid and plasma cells.
- The number of eosinophils in the inflammatory infiltrate varies from 5 to 50%.
- Due to hypersecretion of mucus by goblet cells, bronchial lumens fill with dense mucus, which sometimes takes on a layered appearance.

Acute changes in BA

- In mucus there may be eosinophils and desquamated layers of bronchial epithelium cells, which in cytological smears of patients with bronchial asthma were called Kuršman spirals.
- In addition to eosinophils in mucus, it is possible to find Charcot-Leiden crystals, which are clusters of crystalloids formed by the membrane protein of dead eosinophils.
- Because of the violation of patency of the bronchi, foci of emphysema of pulmonary tissue or atelectasis appear.
- In severe attacks of bronchial asthma, a fatal outcome may occur, due to acute respiratory failure.

Wall of bronchus with BA





A cast of bronchial tubes from mucus in BA

Chronic changes in BA

- With a long course of the disease with frequent attacks, chronic inflammation develops in the bronchial walls, and chronic obstructive pulmonary emphysema is gradually formed.

- In the inflammatory process, bronchi and peribronchial parts of the lung tissue can be involved, followed by the development of bronchiectasis and pneumosclerosis.

Pulmonary emphysema

- Emphysema of the lungs is a syndrome characterized by persistent expansion of airway spaces distal to terminal bronchioles.

- There are different types of emphysema:

  - Perifocal
  - Vicarious
  - Senile
  - Idiopathic
  - Interim
  - Chronic obstructive

Chronic obstructive pulmonary emphysema (COPE)

- Chronic obstructive pulmonary emphysema is a disease caused by the formation of chronic airway obstruction due to chronic bronchitis and bronchiolitis (chronic obstructive pulmonary disease - COPD).

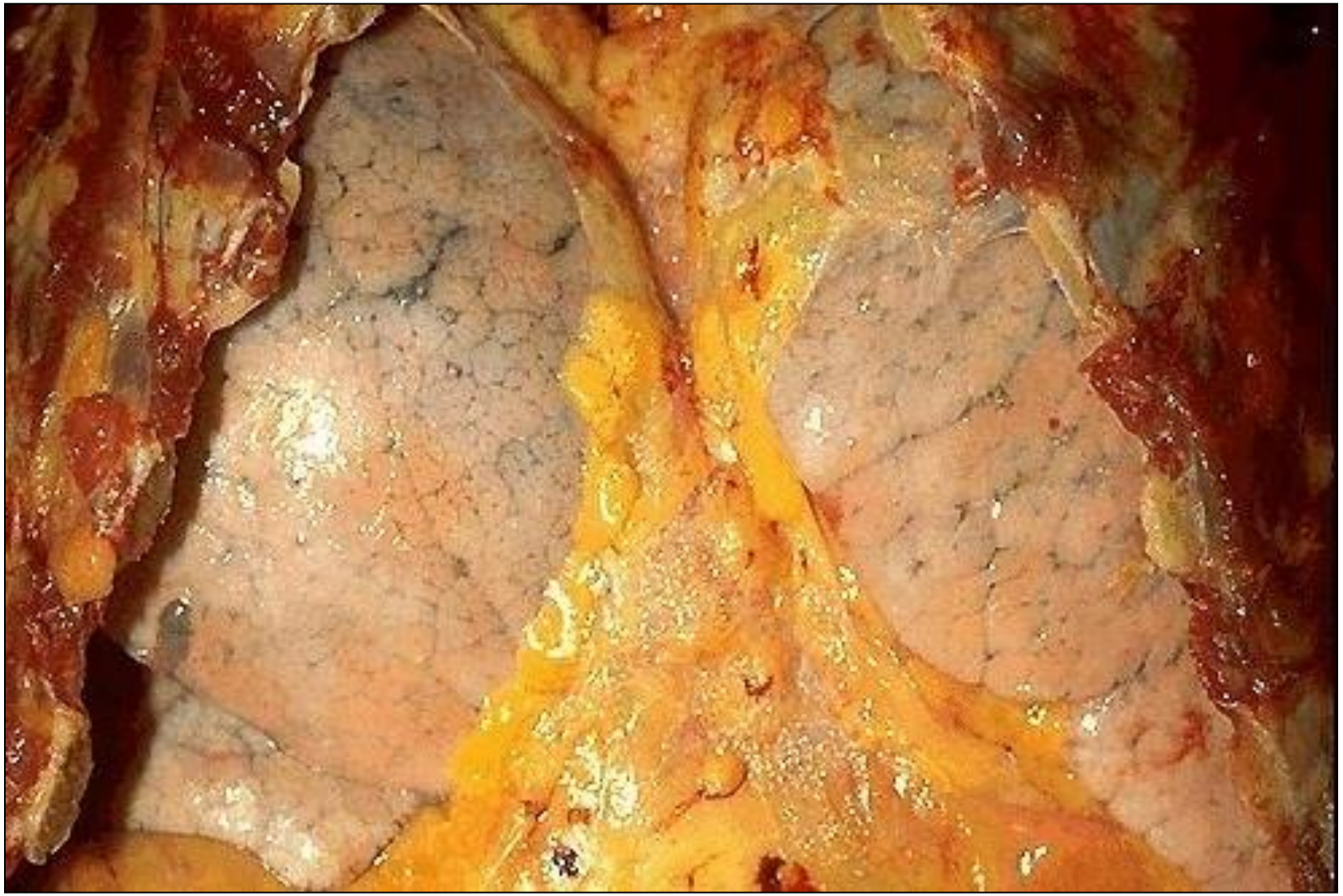
- The disease is associated with the destruction of the elastic and collagenous skeletons of the lung due to the action of leukocyte proteases (elastase, collagenase) in inflammation.

- The crucial pathogenetic link is the genetically determined deficiency of the serum protease inhibitor-alpha-1-antitrypsin.

Morphology of COPE

- Macroscopic:
  - Both lungs increased in size
  - Light gray color
  - Difficult to cut
  - When cut
  - lung tissue crunches (crepitus)
  - When pressing - does not straighten

Pulmonary emphysema

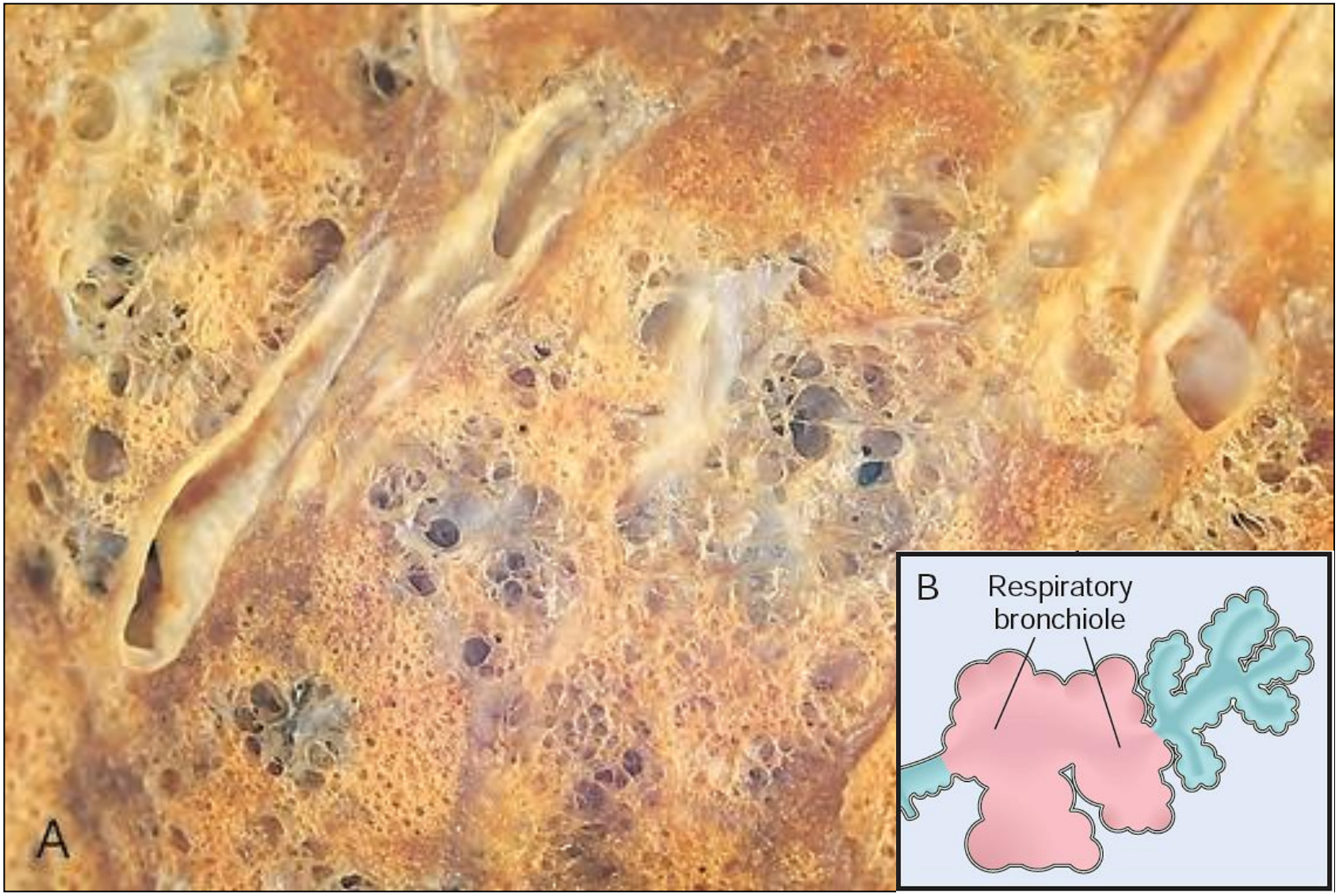


Pulmonary emphysema

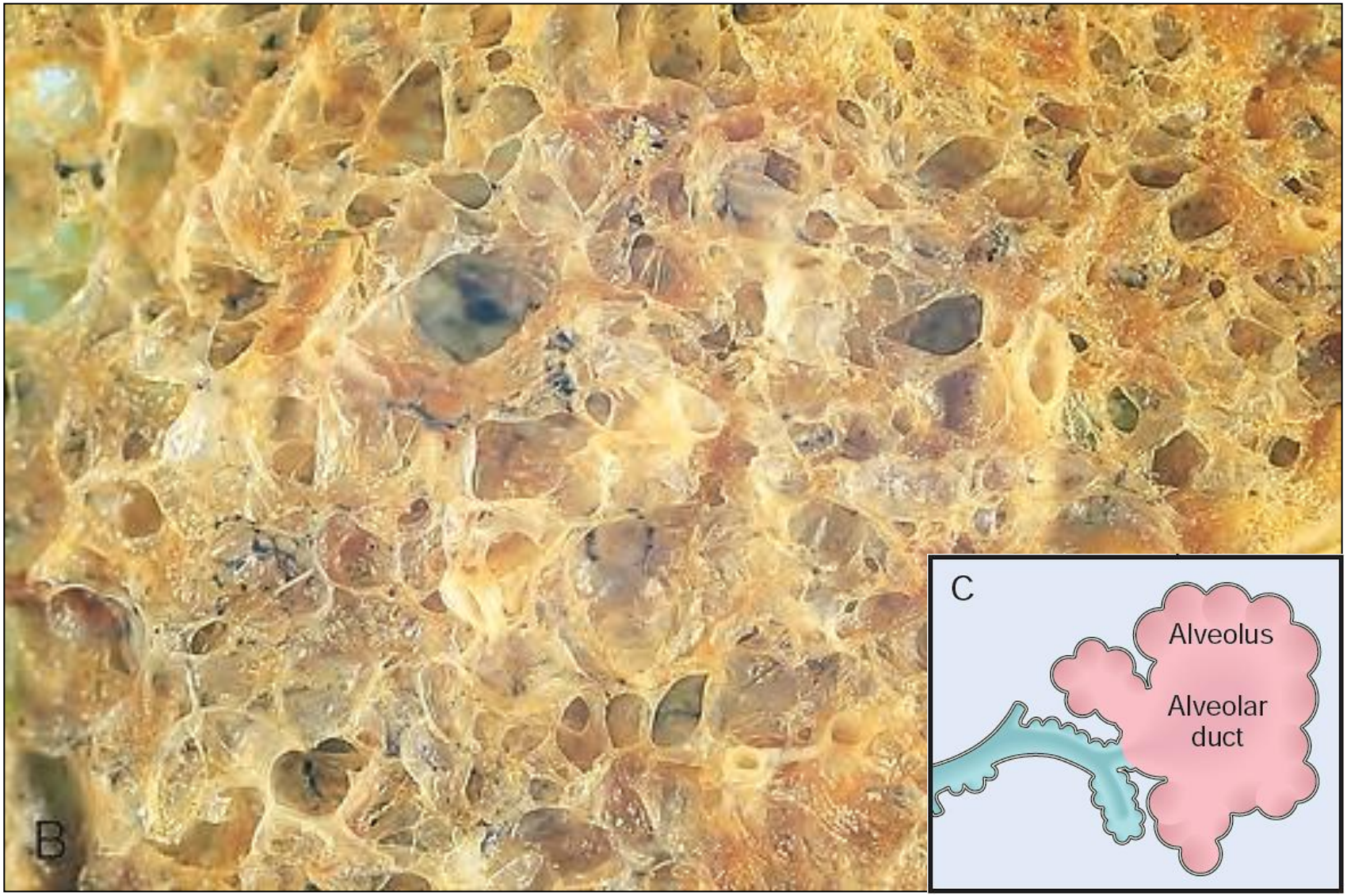
Morphology of COPE

- The morphology is distinguished by the following species of COPE :
- Centroacinar: the bronchioles and proximal sections of the acinus are enlarged.
- Panacinar: the whole acinus is subjected to expansion.
- Paraseptal: dilated distal sections of the acinus extend (often occurs in the subpleural sections).
- Irregular: the acinuses widen unevenly.

Centroacinar emphysema



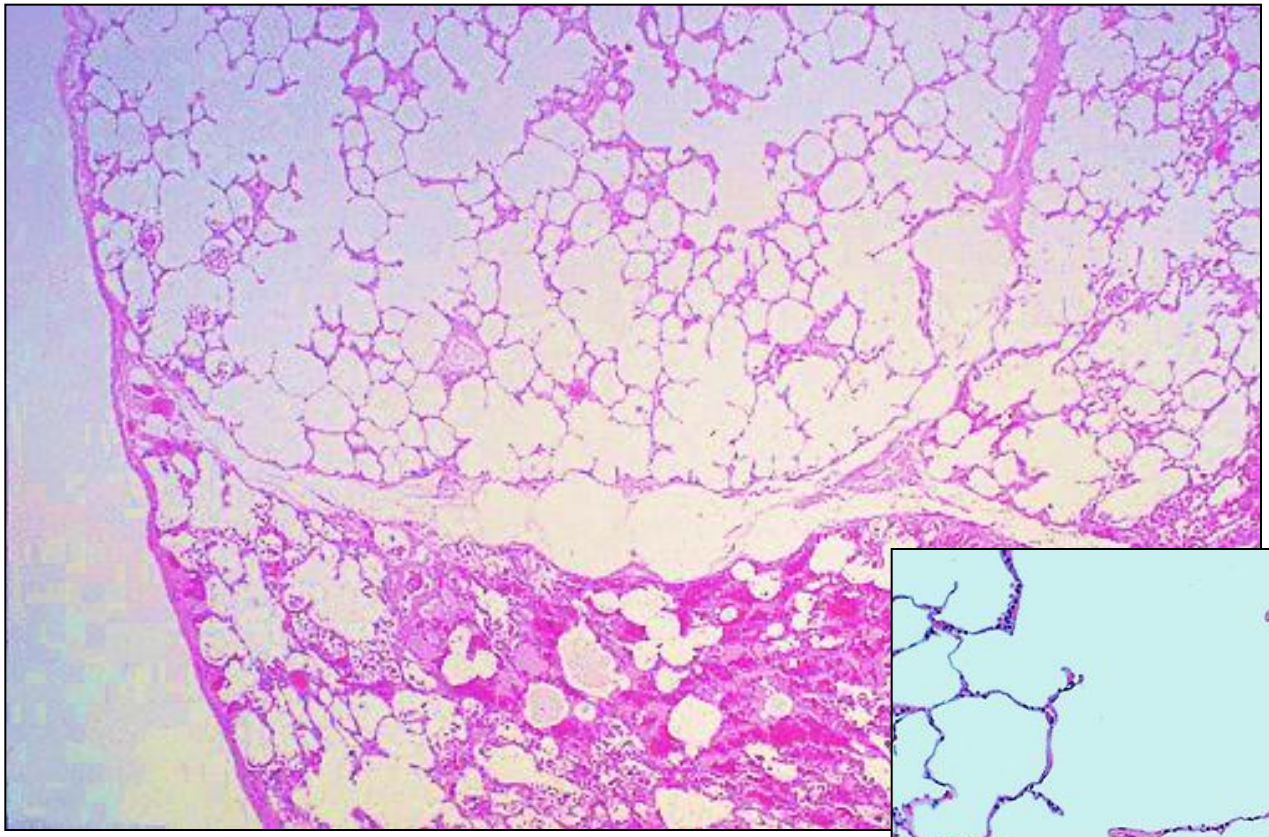
Panacinar emphysema

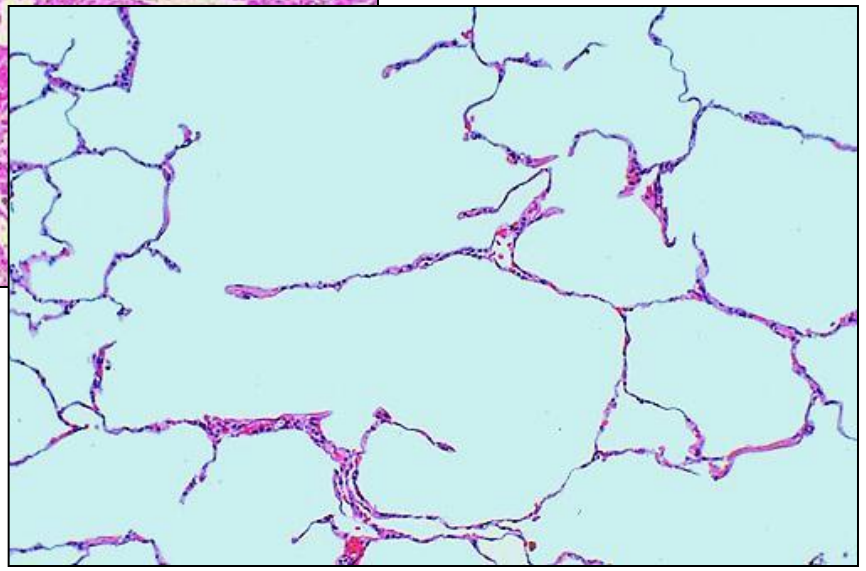


Morphology of COPE

- Microscopic picture:
  - The lumens of respiratory bronchi and alveoli are enlarged
  - The walls of the alveoli are thinned and straightened, the disappearance of the elastic fibers (when stained with fuxelin)
  - The capillary network is reduced, which leads to the development of the capillary-alveolar block and the violation of gas exchange (pulmonary insufficiency)
- As a result of sclerotic changes in the pulmonary capillaries and increase of pressure in the pulmonary artery system, a pulmonary heart develops.

COPE

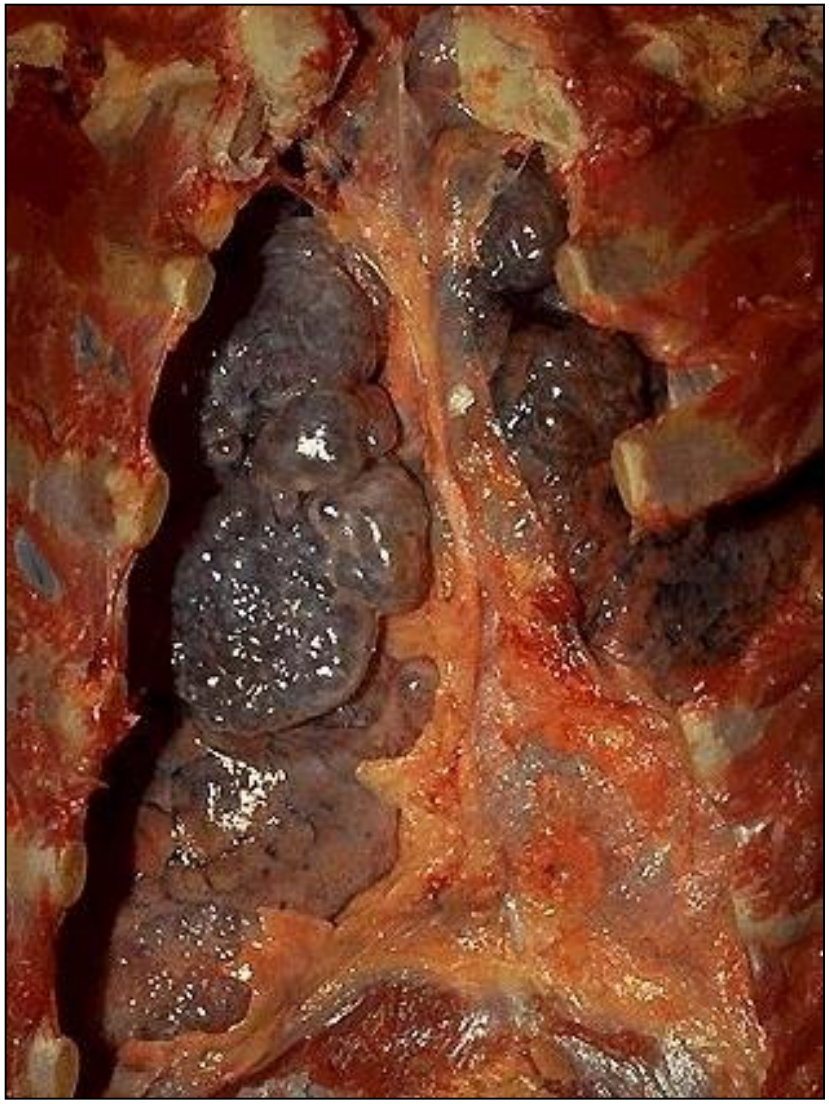


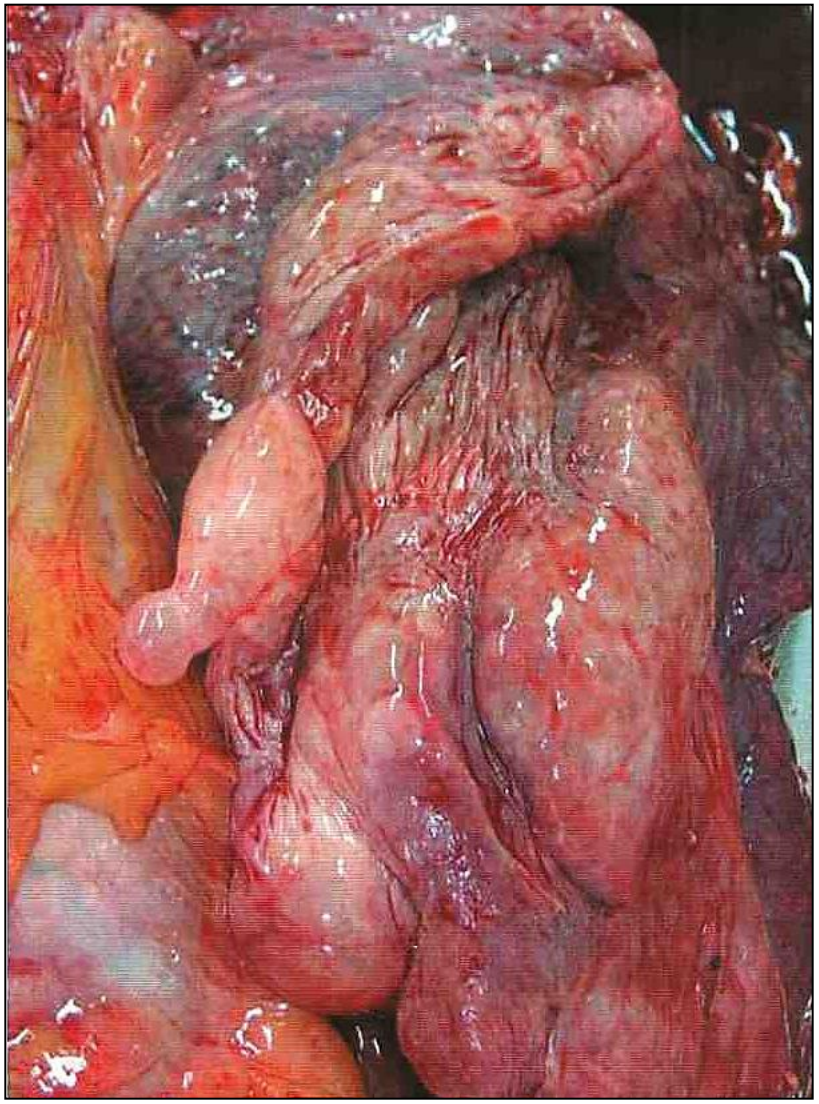


Bullous emphysema

- As a result of rupture of interalveolar septa and fusion of alveoli, bubbles and blisters are visible, visible to the naked eye.
- Large bubbles (bullae) can reach more than 1 cm in diameter and are usually located subpleural.
- Such changes are more typical for irregular emphysema.
- Bubbles can sometimes burst and cause pneumothorax.

Bullous emphysema





Chronic lung abscess

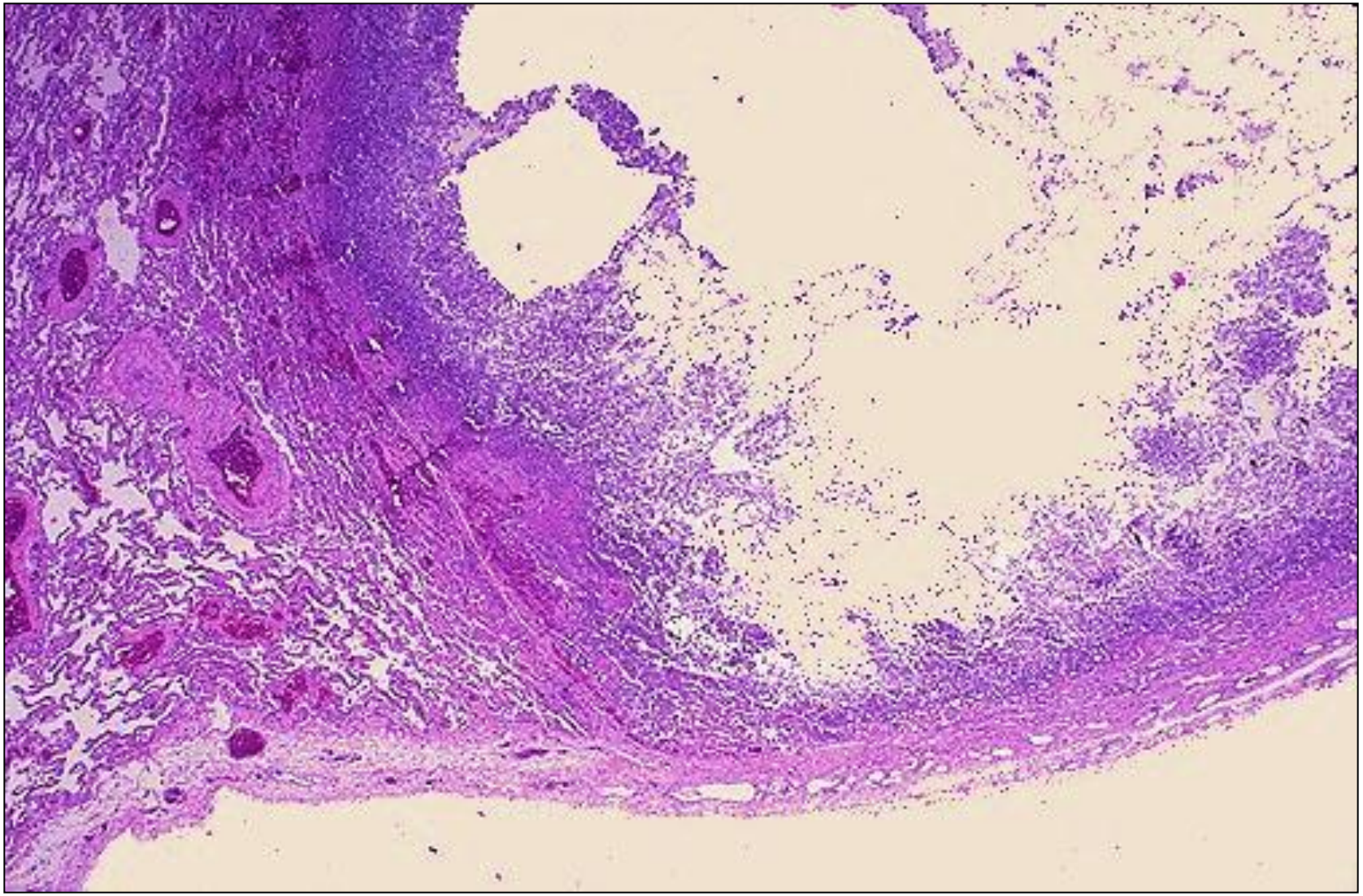
- It develops from the acute and is more often localized in the II, VI, IX, X segments of the right lung.
  It is the source of bronchogenic spread of purulent inflammation in the lung.

- Macroscopic picture:
  The abscess is a cavity filled with pus and surrounded by a dense capsule.

- Microscopic picture:
  The outer layers of the capsule are represented by a connective tissue,
  Internal - granulation tissue and pus (pyogenic membrane).

Chronic lung abscess

Chronic lung abscess



Chronic interstitial lung diseases (CILD)

- CILD is a group of diseases characterized by a primary inflammatory process in the interstitium of the lungs.
- Synonyms: fibrosing alveolitis, pneumonitis.
- Rapidly developing diffuse pneumofibrosis, panacinar emphysema, small-scale hypertension, pulmonary heart and chronic pulmonary-cardiac failure.

Etiopathogenesis of CILD

- Etiological factors:
  - Household, plant and industrial dust (pneumoconiosis)
  - Viruses (measles virus, HIV infection, herpes viruses)
  - Drugs that have a toxic pneumotrophic action (antitumor antibiotics, antidiabetics, cytostatic, immunosuppressive drugs)

Etiopathogenesis of CILD

- The development of the disease is associated with the appearance of autoantigens, animal and plant antigens, viruses, which causes the formation of immune complexes that circulate in the blood, deposited in the interalveolar septa, bind complement and cause a cellular inflammatory reaction in interstitium interalveolar septa.

- In this case, macrophagal granulomas can form.

- In the outcome of a chronic inflammatory process, diffuse interstitial fibrosis is formed.

Classification of CILD

- On the etiology of CILD are:

- With established etiology:
  Pneumoconiosis
  Exogenous allergic alveolitis

- With unidentified etiology (predominate):
  Idiopathic fibrosing alveolitis (Hammain-Rich disease)
  Secondary fibrosing alveolitis in rheumatic diseases and HBV infection
  Fibrosing alveolitis with Goodpasture syndrome, Sarcoidosis
  Histiocytosis X
  Eosinophilic pneumonia, and others

Classification of CILD

- By the nature of inflammation:
  - Interstitial inflammation and fibrosis without granuloma formation
  - Interstitial inflammation with granulomas and fibrosis:
    Pneumoconiosis
    Exogenous allergic alveolitis
    Sarcoidosis
    Histiocytosis X

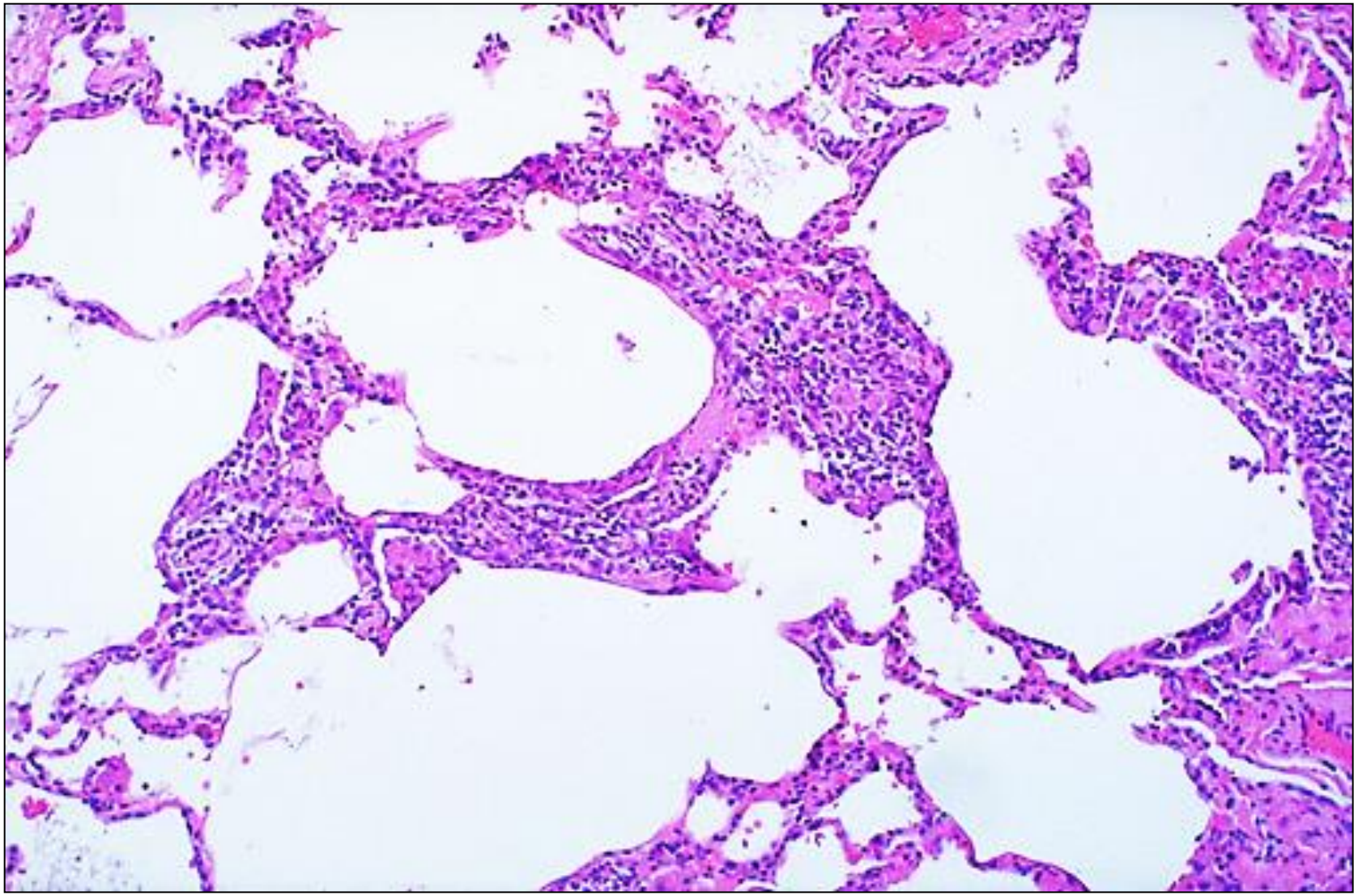
Morphology of CILD

- Three consecutive stages of morphological changes in CILD were established:
  - Alveolitis
  - Disorganization of alveolar structures
  - Formation of a "cellular lung"

Alveolitis

- Infiltration of interstitial lung tissue with neutrophils, lymphocytes, macrophages, plasma cells.
- In the lumens of the alveoli there is a serous exudate, often hyaline membranes, hyperplasia of type II pneumocytes that protrude into the alveolar lumens (diffuse alveolitis).
- In some cases, the process is not diffuse, and focal, with the formation of macrophage granulomas (granulomatous alveolitis).
- The interalveolar septa significantly thicken, their capillaries squeezed.

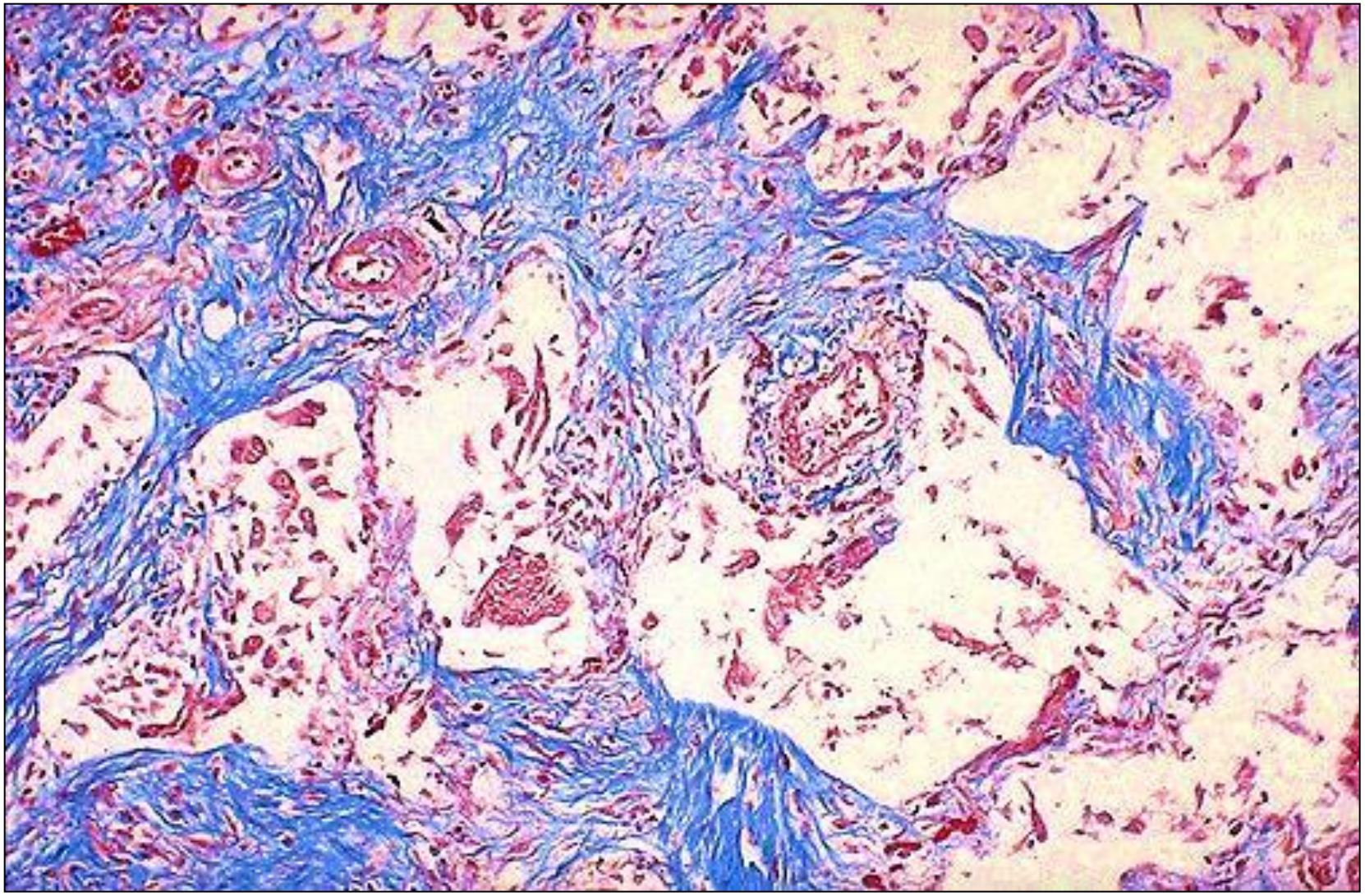
Alveolitis



Disorganization of alveolar structures

- Destruction of endothelial and epithelial membranes,
- Reinforcement of cellular infiltration in the interstitium,
- The spread of inflammation to the walls of the vessels and perivascular tissue - develops diffuse pneumofibrosis.

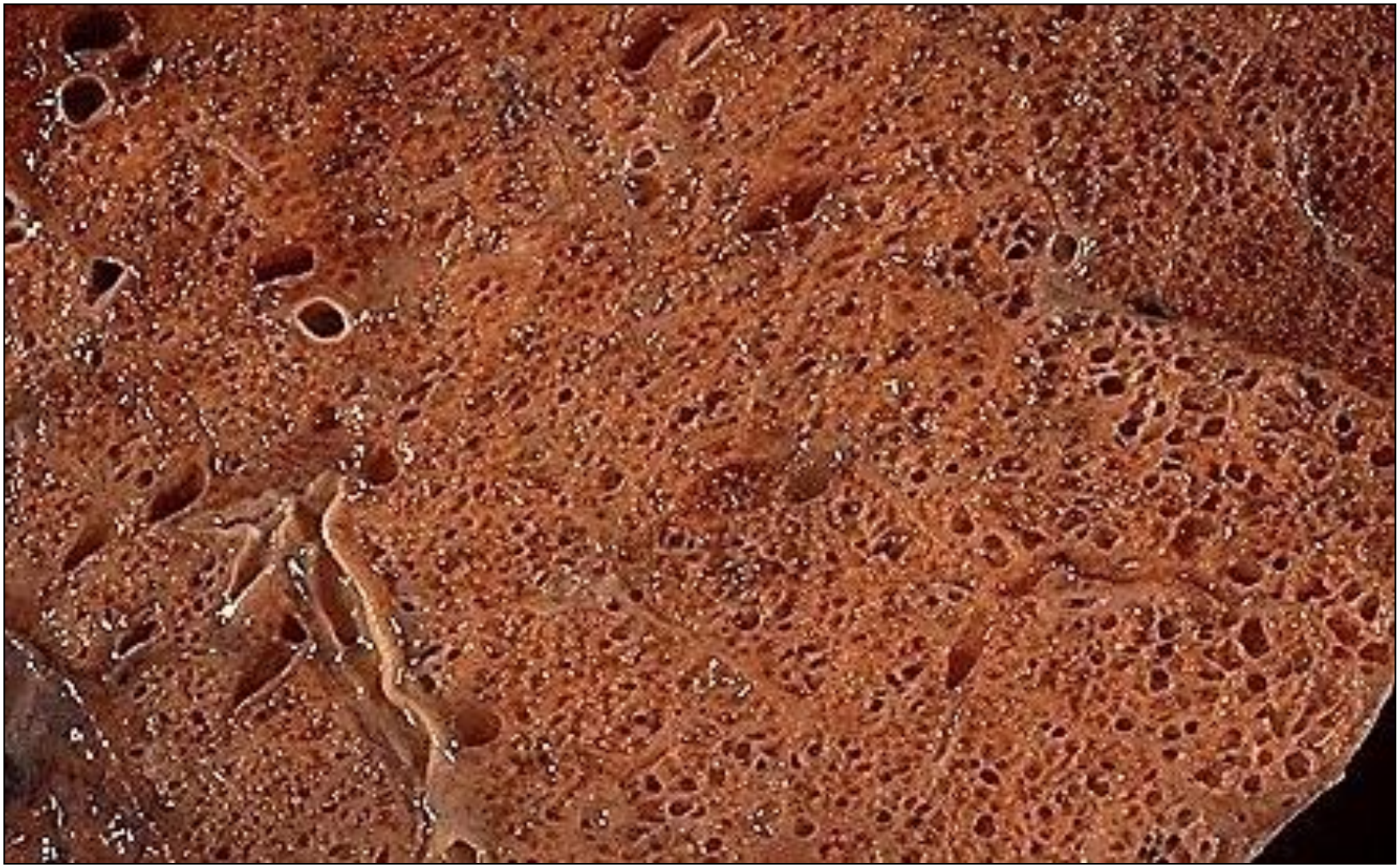
Diffuse pneumofibrosis



Formation of "cellular lung"

- There is a capillary block,
- Along with pneumofibrosis develops panacinar emphysema, bronchiectasis (bronchioloectasis),
- Cysts with fibrous walls are formed on the site of the altered alveoli - "honeycomb lung" (resembling bee honeycombs).

"Cellular lung"



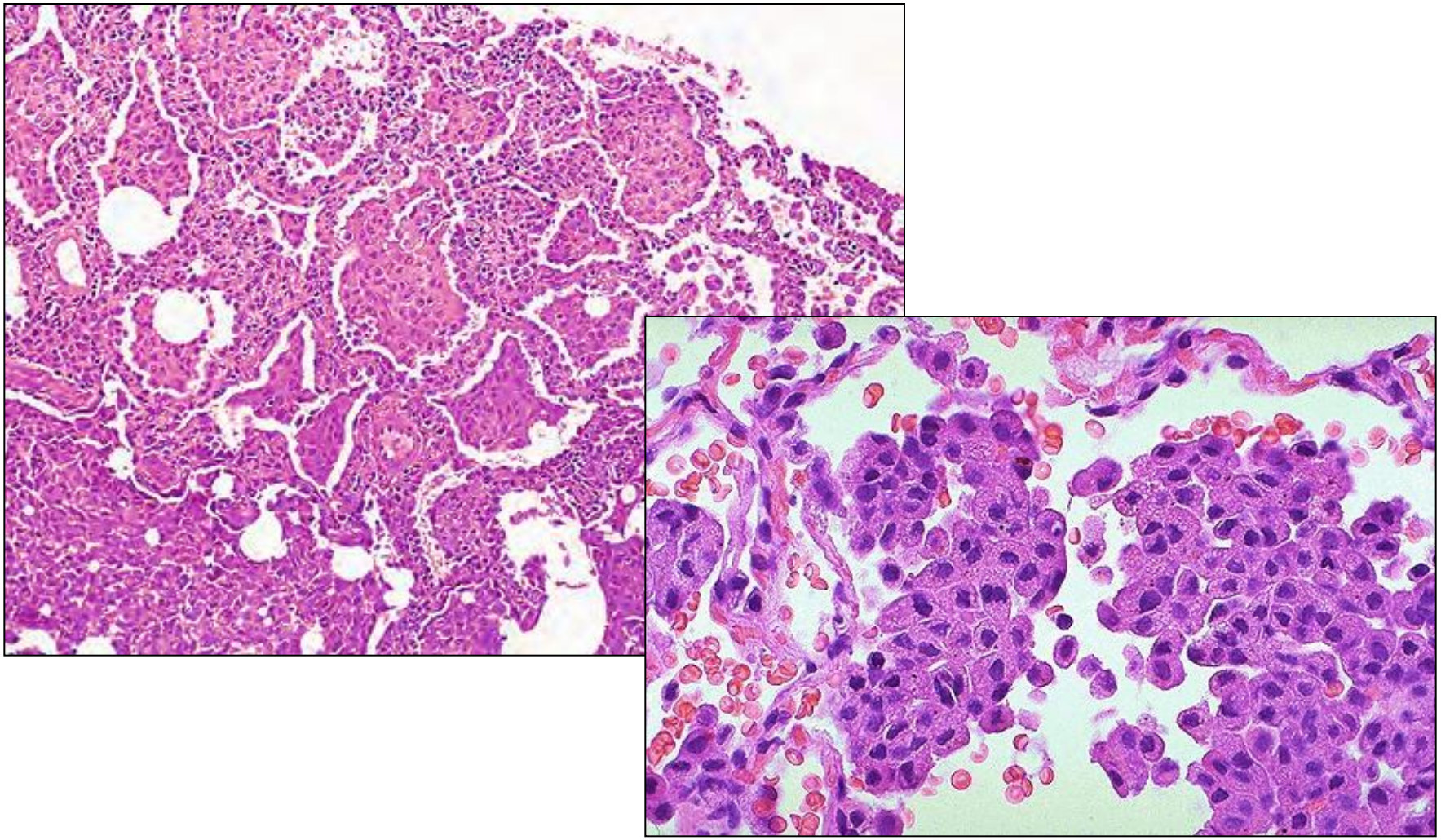
Clinical and morphological forms of pneumonitis

- Desquamative interstitial pneumonitis
- Hypersensitive pneumonitis

Desquamative pneumonitis

- The etiology is unknown.
- Morphologically in the lumens of alveoli, clusters of macrophages are identified, which were mistaken for desquamated cells of the alveolar epithelium.
- In the cytoplasm of macrophages, lipid and PAS-positive granules, or lamellar structures, are identified that may be particles of the surfactant.
- In the future there is hyperplasia of alveolocytes, which are really desquamated into the lumens of the alveoli.
- Gradually, signs of interstitial fibrosis increase.
  The disease can be treated with steroid drugs.

Hyperplasia of alveolar macrophages with desquamative pneumonitis



Hypersensitive pneumonitis

- A group of interstitial lung diseases that develop with prolonged, intense contact with organic dust.

- Synonym: exogenous allergic alveolitis.
  They lead to the development of a clinical and morphological picture of "light farmer", "light poultry farmer", "light mushroom picker", etc.

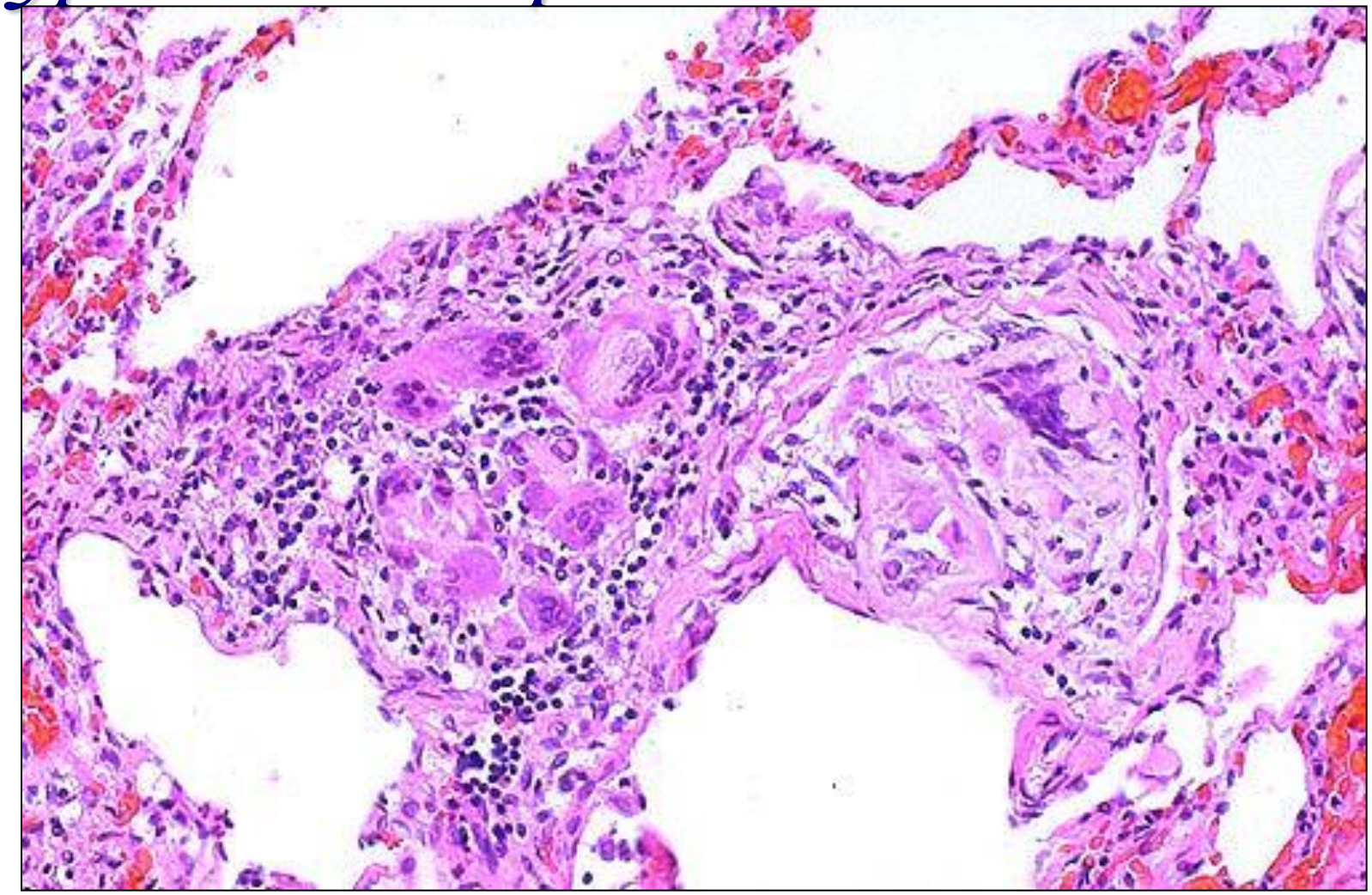
- The mechanism of the disease in the early stages of development (acute form) is caused by immunocomplex reactions of the III type, in the late stages - by type IV reactions with the formation of granulomas (chronic form).

"Poultry-farmer's lung"

Hypersensitive pneumonitis

- In acute form in interalveolar septa, infiltration of lymphocytes, plasma cells, macrophages appears.
- A characteristic morphological sign of the chronic form is the presence of granulomas scattered over the lung tissue, containing multinucleated giant cells of foreign bodies.
- Vasculitis that occur with immunocomplex diseases, with hypersensitive pneumonitis absent.

Granulomas of foreign bodies with hypersensitive pneumonitis



Lung cancer

- Occupies the first place among malignant tumors in terms of morbidity and mortality of men in most countries of the world.
- It is characterized by a poor prognosis.
- All cases of lung cancer are etiologically related to smoking.

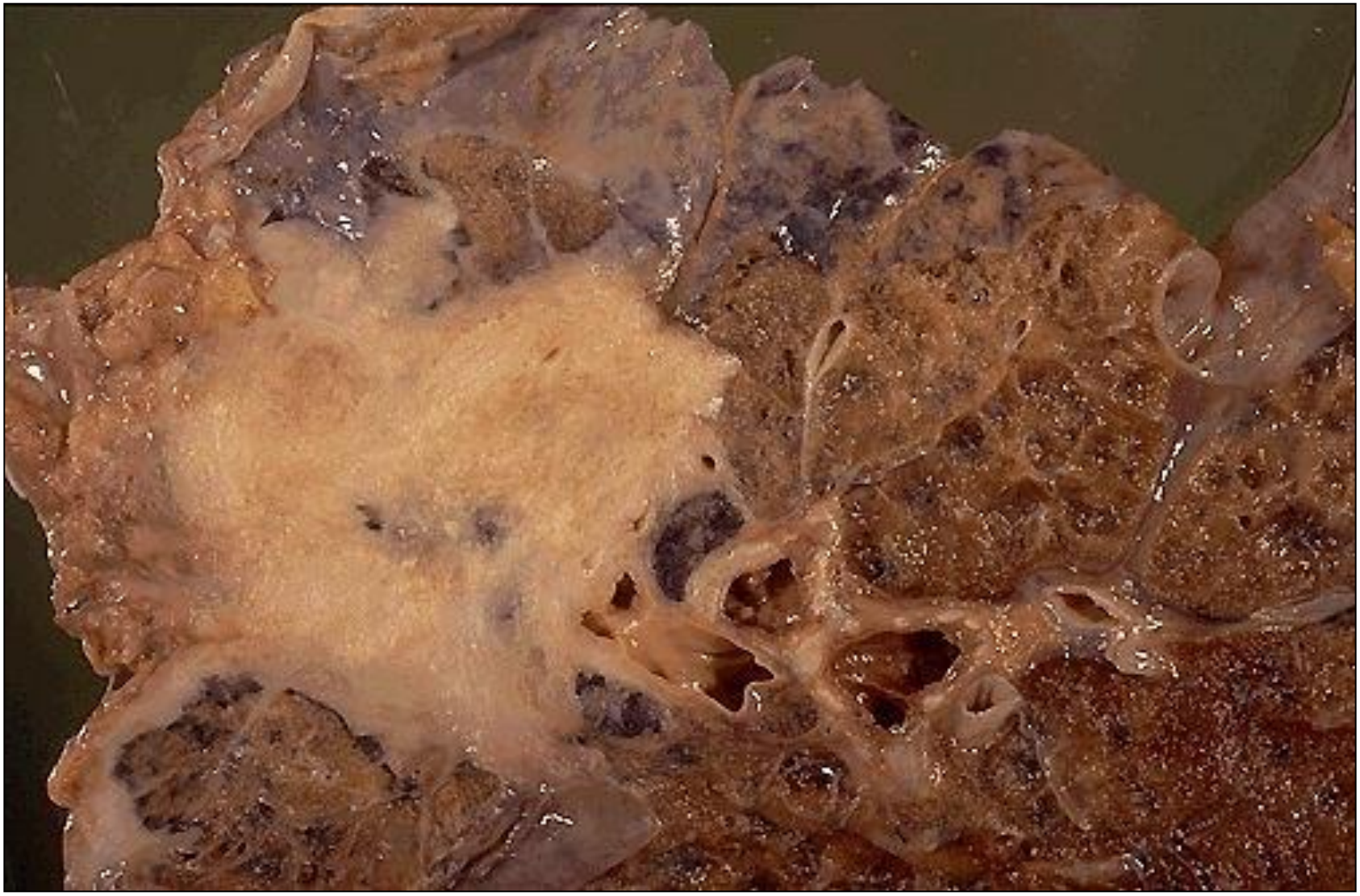
Classification of lung cancer

- By localization:
  - Basal (central) cancer.
  - Peripheral cancer.
  - Mixed cancer.
- By the nature of growth:
  - Exophytic (endobronchial).
  - Endophytic (exo- and peribronchial).

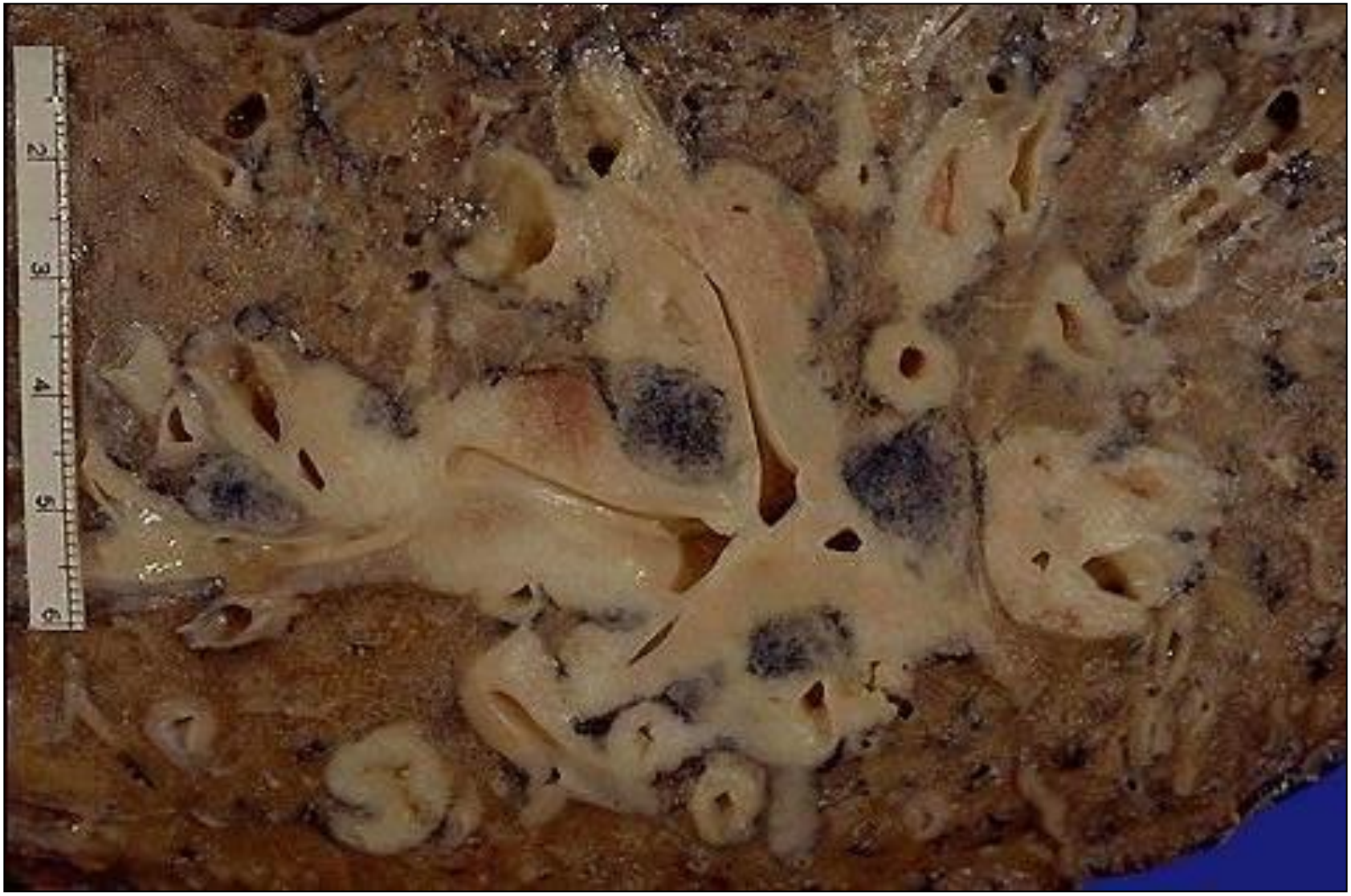
Classification of lung cancer

- According to the macroscopic form:
  - Plaque-like
  - Polyposis
  - Endobronchial diffuse
  - Knotted
  - Ramified
  - Knot-branched
  - The cavitary
  - Pneumonia-like

Nodular lung cancer



Branched lung cancer



Classification of lung cancer

- According to the microscopic form (histogenesis):
  - A squamous cell (epidermoid), its variant is spindle cell
  - Small cell: oat cell (lymphocyte-like), intercellular, combined
  - Adenocarcinoma: acinar, papillary, bronchioloalveolar carcinoma, solid with mucus production
  - Large-celled, its variants are giant-celled, light-celled
  - Iron-squamous cell carcinoma
  - Cancer of bronchial glands: adenoid-cystic cancer, mucoepidermoid cancer, etc.

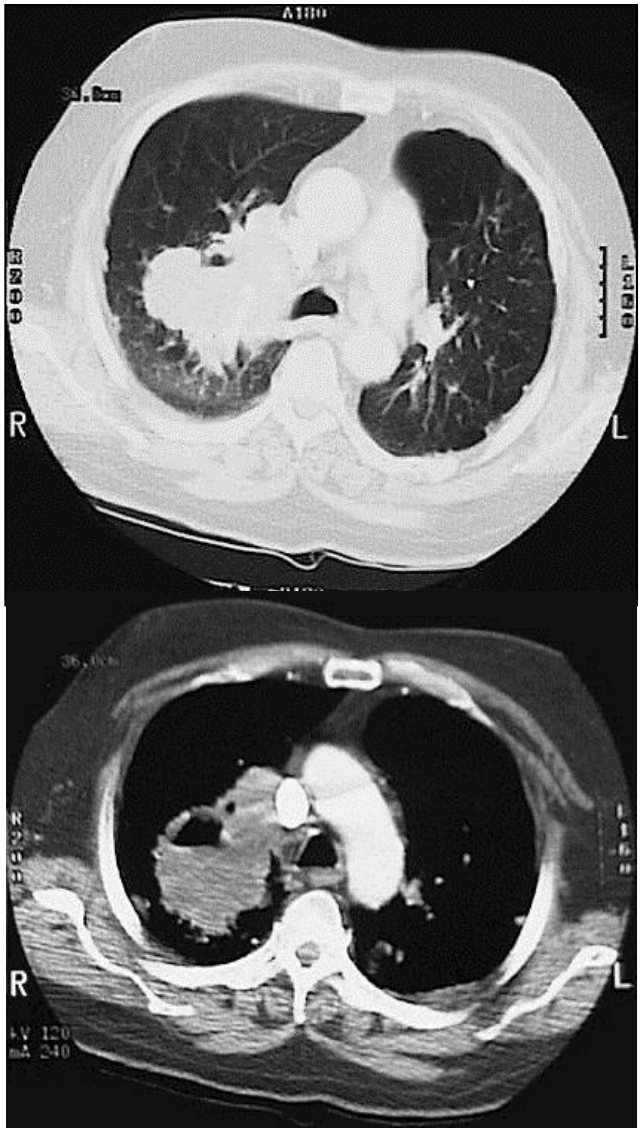
Basal (central) lung cancer

- It develops in the large bronchi (stem, lobar bronchi and proximal part of the segmental bronchus).
- Precancerous processes: squamous cell metaplasia and dysplasia of the bronchial epithelium against a background of chronic inflammation.
- Early violations of bronchial patency occur, which leads to atelectasis and lung abscesses.
- Basic diagnostic methods: bronchoscopy with biopsy, cytological examination of sputum, X-ray examination.

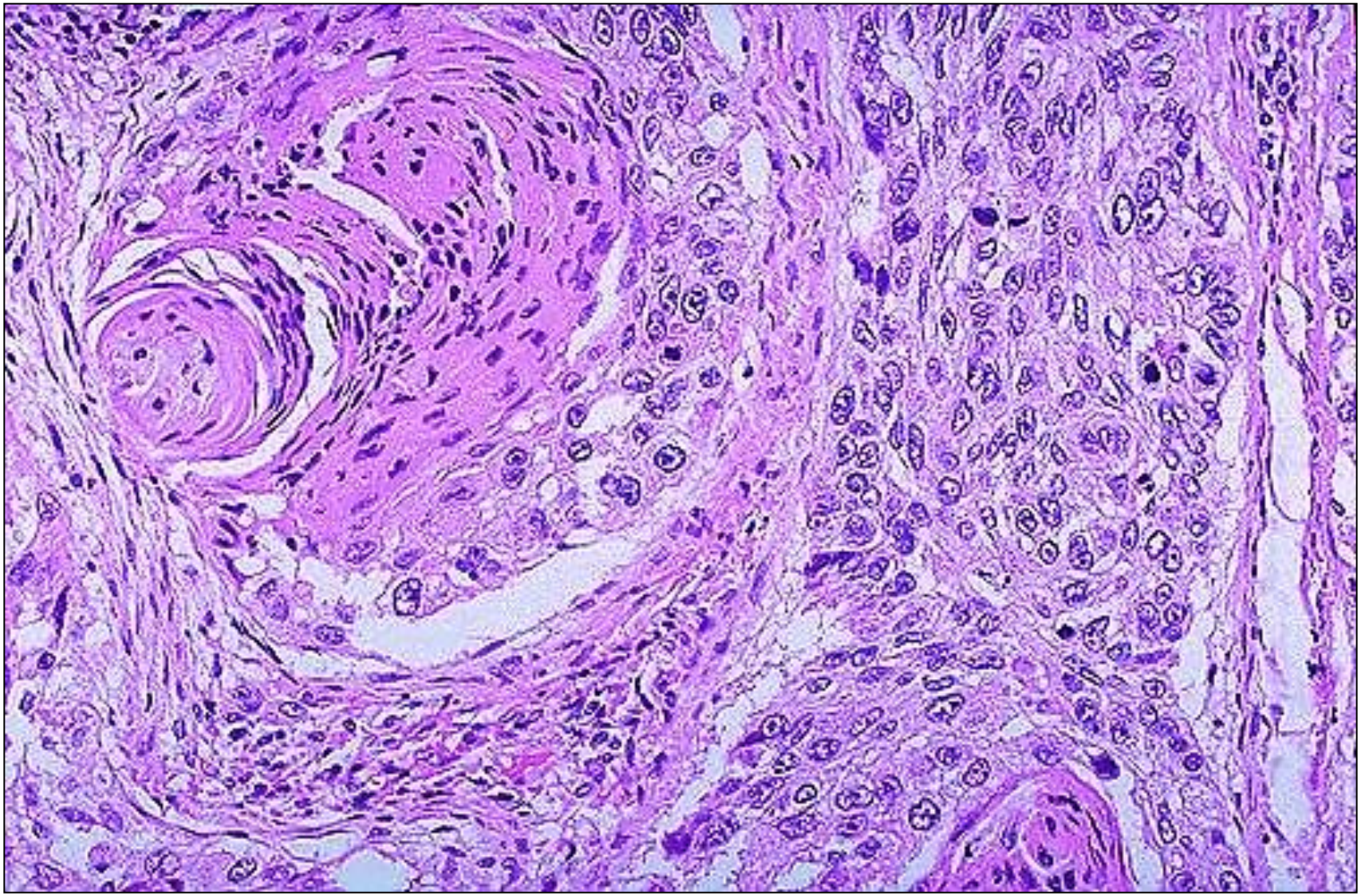
Basal (central) lung cancer

- The predominant macroscopic forms: polypous, nodular, branched, nodular-branched.
- The most common microscopic types: squamous and small cell.
- Small cell lung cancer is associated with the worst prognosis.
- Small cell lung cancer is able to secrete ACTH, which will be clinically manifested by Cushing's paraneoplastic syndrome.

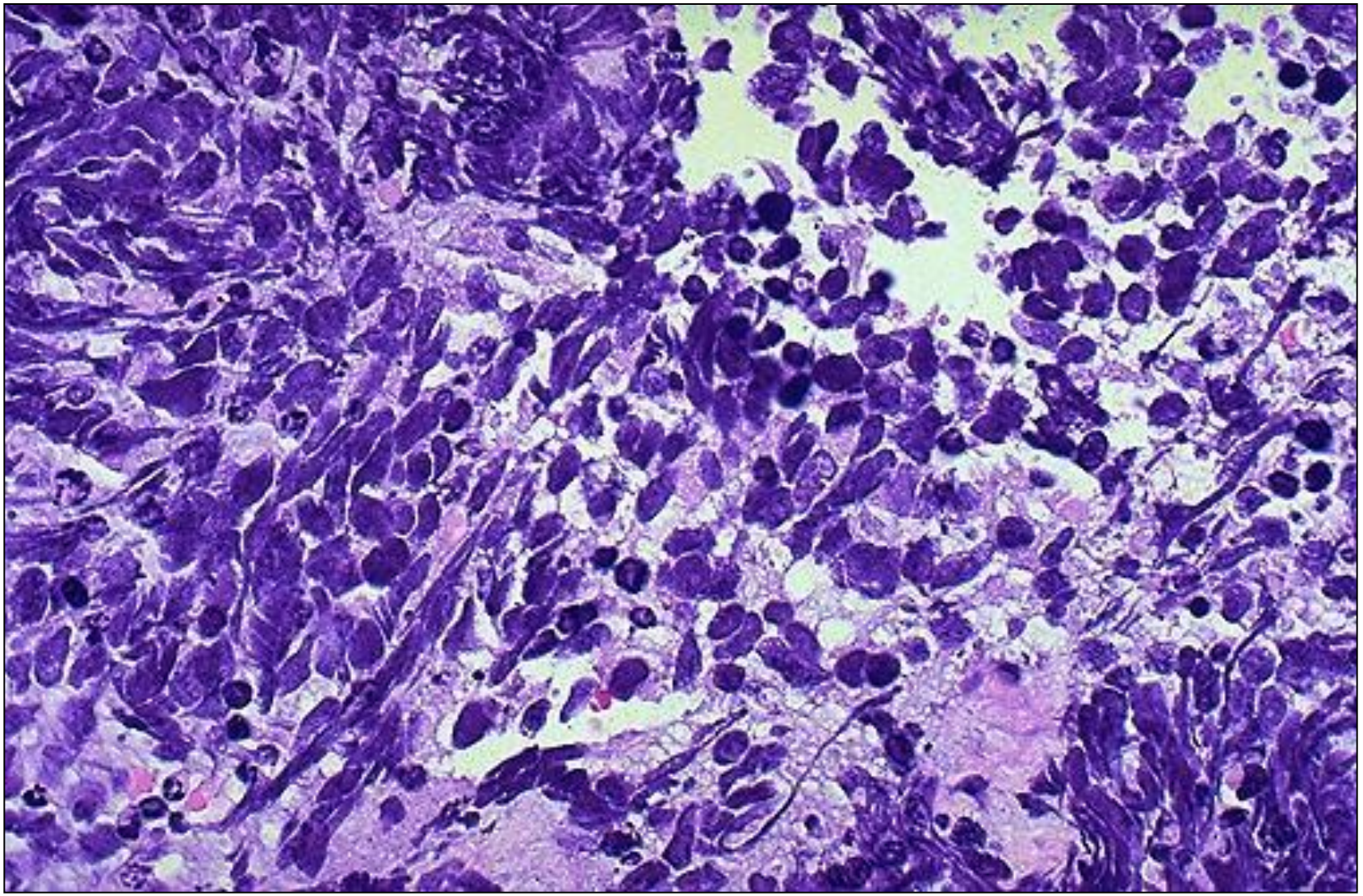
Basal (central) lung cancer



Squamous cell carcinoma



Small cell lung cancer

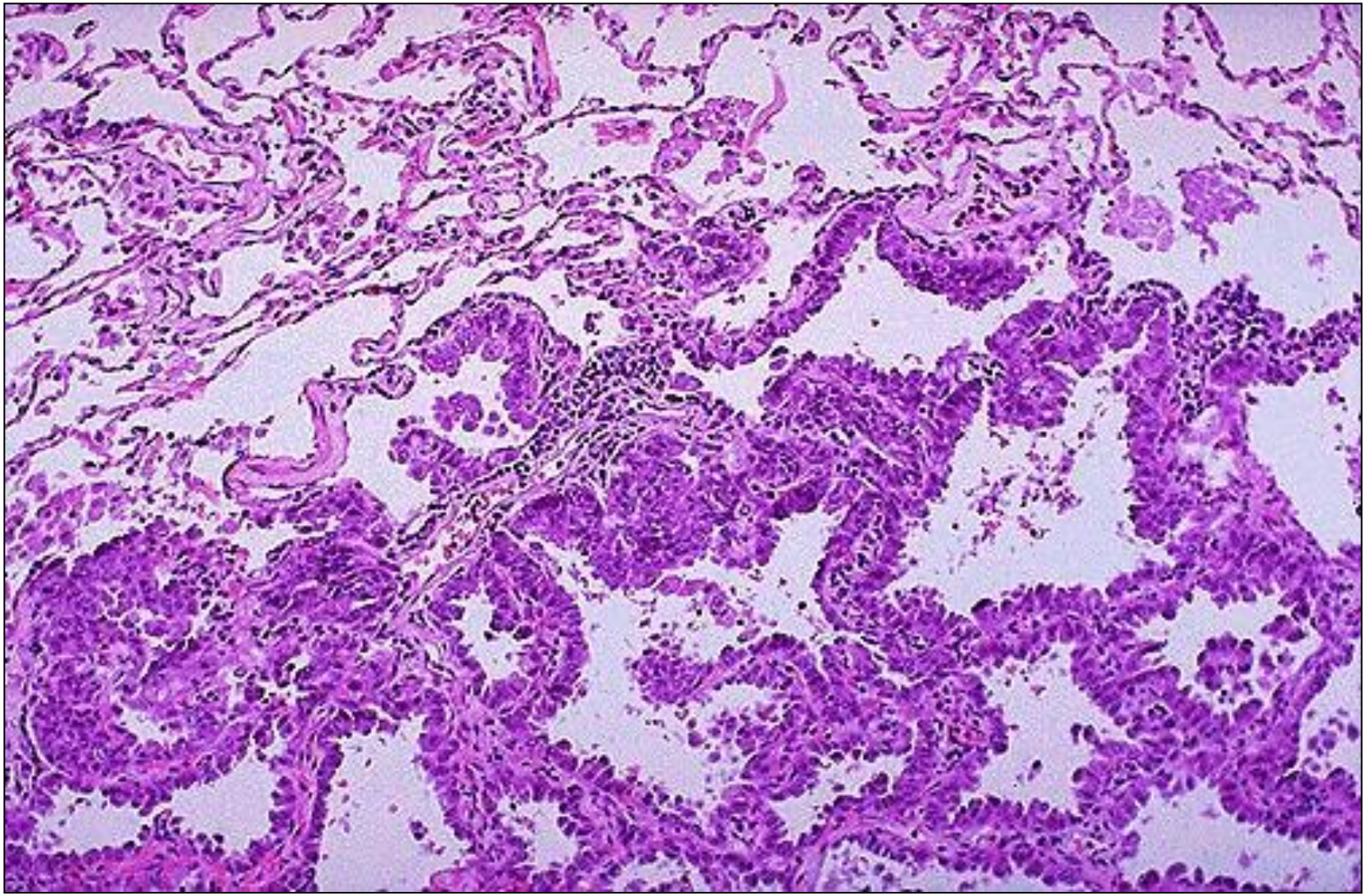


Peripheral lung cancer

- It comes from bronchi of smaller caliber, bronchioles and, probably, alveoli.
- It often develops in the rumen (a hotbed of pneumosclerosis). The main diagnostic method is X-ray.
- On the roentgenogram, peripheral lung cancer can simulate tuberculoma.
- The predominant macroscopic forms: nodose, nodular-branched, cavitary and pneumonia-like.
- Microscopic forms are diverse, glandular carcinomas predominate, bronchiolo-alveolar carcinoma is more common.

Peripheral lung cancer

Broncho-alveolar carcinoma



Mixed lung cancer

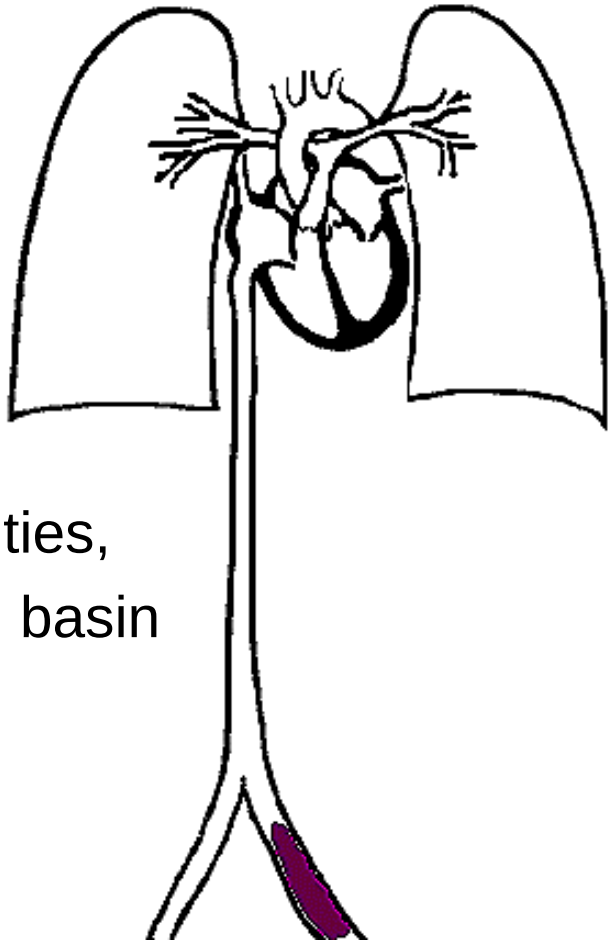
Metastasis of lung cancer

- The first metastases are found in the regional (peribronchial) lymph nodes.
- Further, bifurcation, paratracheal, mediastinal and cervical lymph nodes are involved.
- Pleural and peritoneal carcinomatosis may develop.
- Hematogenous metastasis is carried out mainly in the liver, bones, adrenals and brain.

Pulmonary thromboembolism (PE)

- PE – one of the most common causes of sudden death in patients with heart failure.

Pulmonary thromboembolism (PE)



- The source of thromboembols are:
  - Thrombi of the veins of the lower extremities,
  - Thrombi of veins of a cellulose of a small basin
  - Blood clots of the right heart.

Pulmonary thromboembolism (PE)

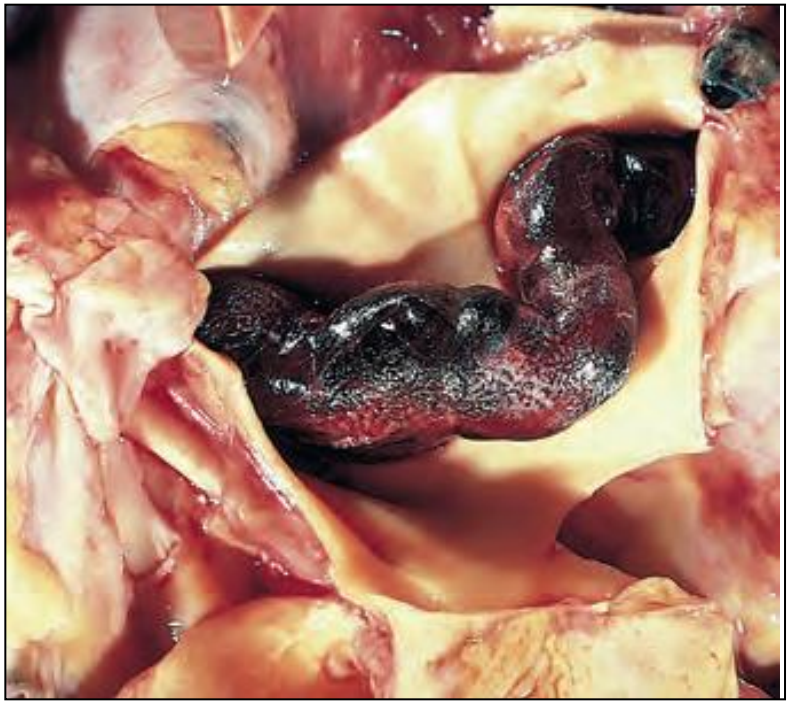
- In the genesis of death in PE they have the meaning:
  - Closure of the lumen of the vessel with the development of acute right ventricular failure,
  - Pulmonocoronary reflex:
    Spasm of the bronchial tree,
    Spasm of the branches of the pulmonary artery,
    Spasm of the coronary arteries of the heart.

Pulmonary thromboembolism (PE)

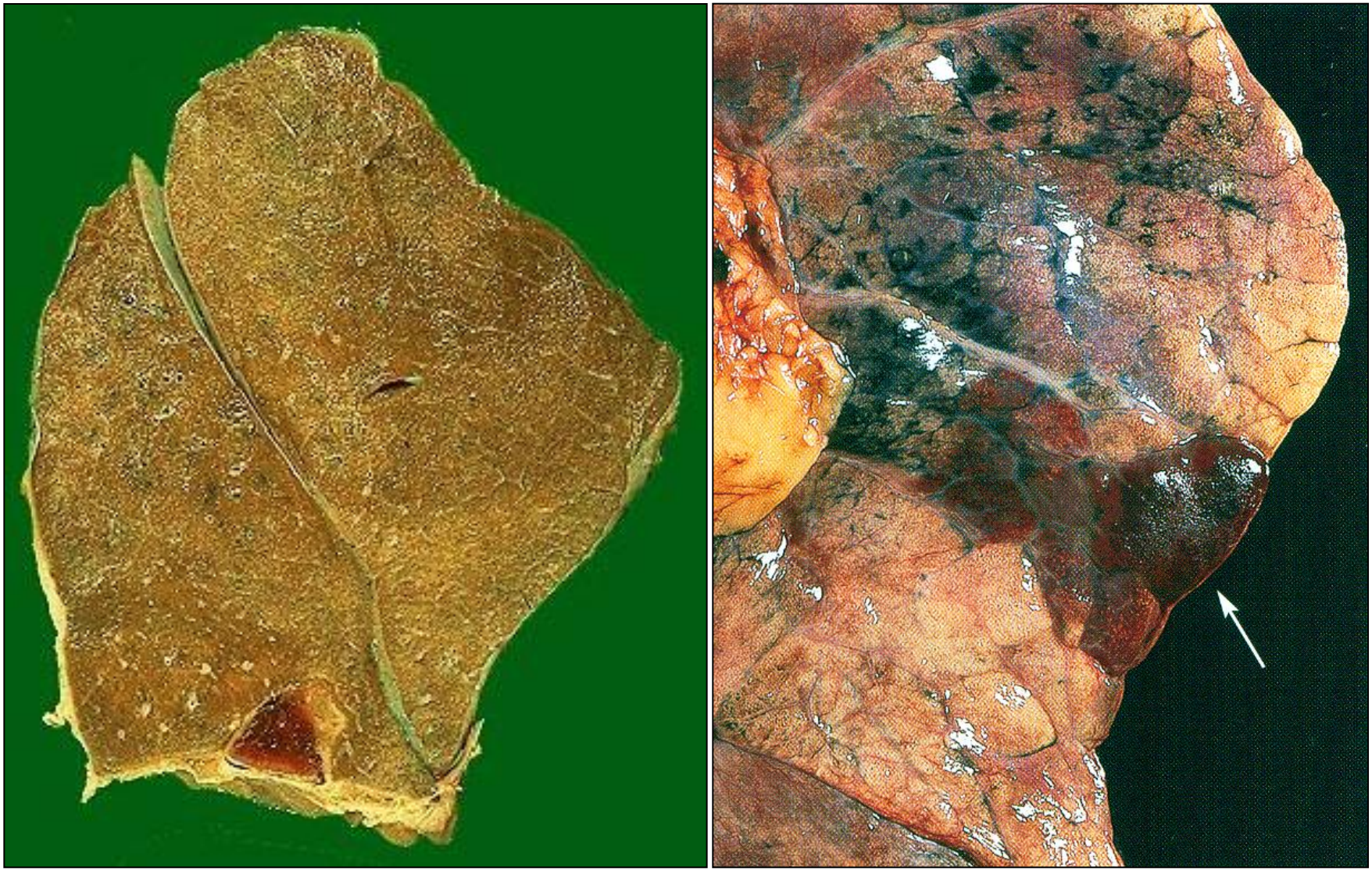
- On the autopsy in the common pulmonary artery trunk, free-lying dense gray-red color wormlike masses with a dull surface (thromboemboles) are visible.

- When thromboembolism of small branches of the pulmonary artery develops hemorrhagic lung infarction.

Pulmonary thromboembolism (PE)



Hemorrhagic lung infarction



Pulmonary edema

- Pulmonary edema complicates many diseases of the lungs, heart and other organs.
- In the tissue of the edematous lung, more than 4-5 ml of liquid per 1 gram of dry matter is accumulated.
- Lung edema is most often caused by two groups of causes:
  - Increased hydrostatic pressure in the venous part of the small circle of circulation (with acute left ventricular failure, mitral stenosis, hypervolemia of the small circulation, pulmonary vein obstruction)
  - Local increase in the permeability of capillaries of the alveolar septum (with adult respiratory distress syndrome).

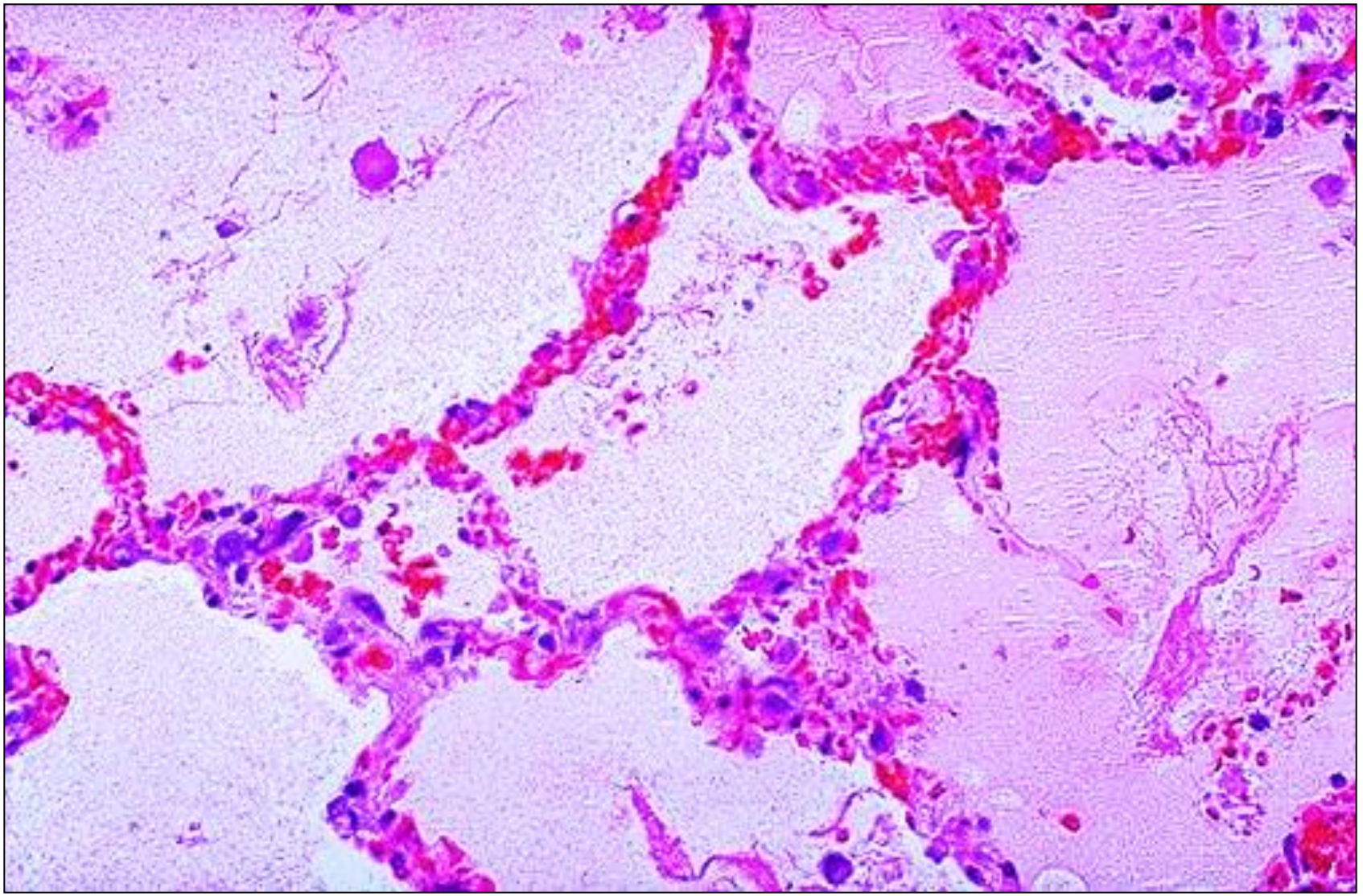
Pulmonary edema

- In addition, pulmonary edema can develop with a decrease in oncotic blood pressure in nephrotic syndrome, liver disease, enteropathy accompanied by hypoalbuminemia, and edema caused by obstruction of lymphatic drainage.

Pulmonary edema

- Macroscopic picture:
  - Lungs are enlarged in size
  - Heavy
  - Pink colour
  - Testate consistency
  - When pressed from the surface of the cut, foamy pink liquid flows down
- Microscopic picture:
  - The first stage: edematous fluid accumulates in the interstitium (interalveolar septa) of the lung
  - The second stage: the edematous fluid accumulates in the lumen of the alveoli
  - Also erythrocytes and other cellular elements of blood can leave.

Pulmonary edema



Respiratory distress syndrome in adults (RDSA)

- RDSA is associated with damage to the endothelium of the capillaries and often of first-order pneumocytes in the airgematic barrier zone with the subsequent development of respiratory failure.

- More than 60% of RDSA patients die, despite modern methods of treatment.

- Particularly high mortality in the outcome of RDSA with aspiration of gastric contents (93.8%), sepsis (77.8%) and pneumonia (60%).

Acute respiratory distress syndrome (ARDS)

- ARDS as a complication can occur when:
  - Aspiration of gastric contents
  - DIC-syndrome
  - Infectious diseases of the lungs (pneumonia)
  - Shock (septic, traumatic, etc.)
  - Inhalation of toxic substances, incl. Excess amounts of oxygen, paraquat
  - Overdose of narcotic drugs
  - Operations on the heart with extracorporeal circulation
  - Radiation effects

The stages of the ARDS

- ARDS takes place in three stages:
  1. Preclinical
  2. Acute
  3. The organization of exudate and proliferation of pneumocytes of the second order

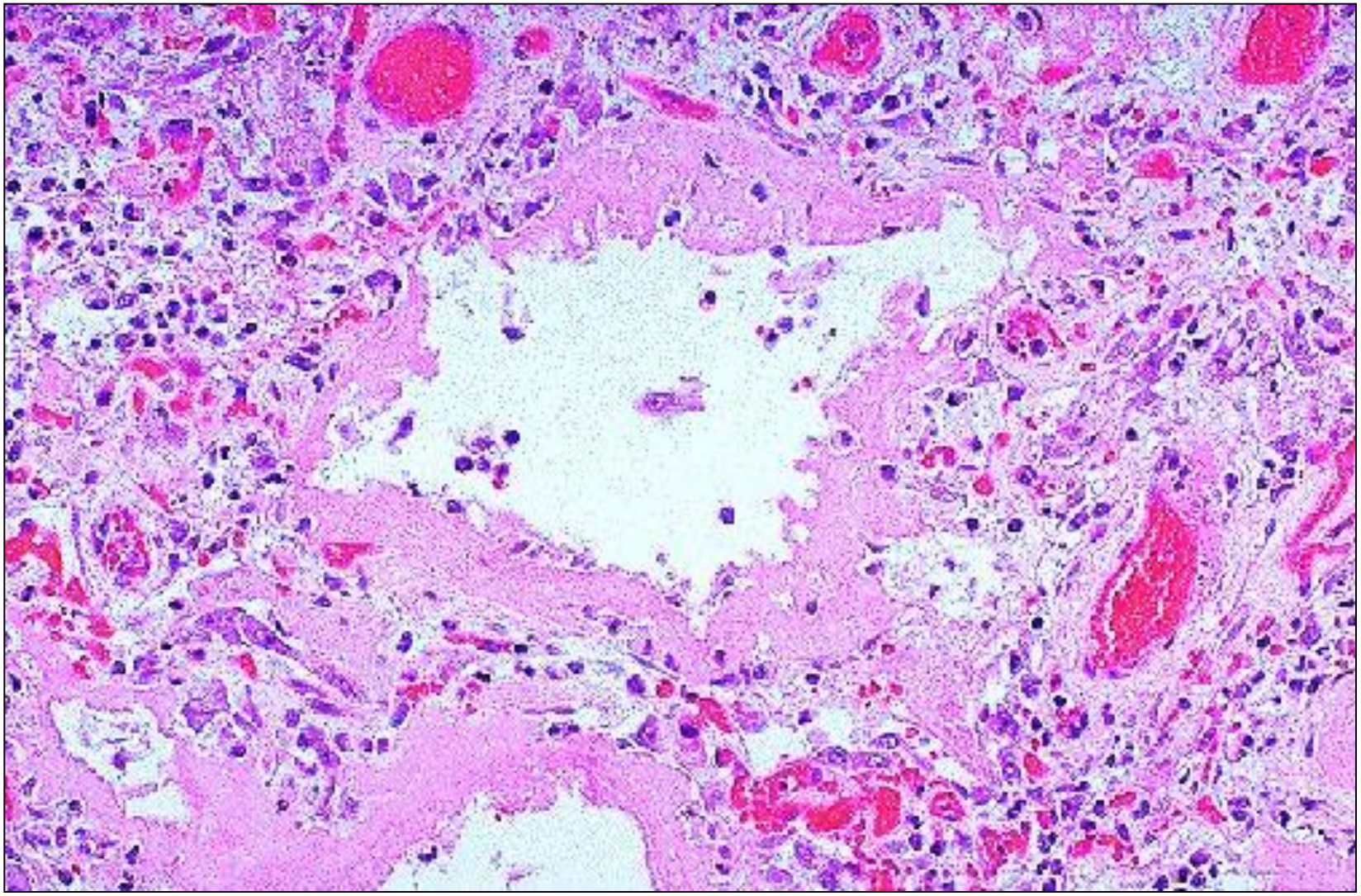
Preclinical stage of ARDS

- Characterized by morphological signs of damage to the capillaries of the alveolar septa.

Acute stage of ARDS

- Characterized by the development of pulmonary edema.
- Develops within the first week after the action of the damaging factor.
- Morphological manifestations:
  - Intra-alveolar and interstitial edema
  - Inflammatory changes with a large number of PMNL and fibrin both in intraalveolar exudate and in tissue infiltrates
  - Hyaline membranes
  - Atelectasis

Hyaline membranes with ARDS



Stage of exudates organization of ARDS

- It ends with interstitial fibrosis.
- Organization processes start from the 2nd - 3rd day of the disease.
- Death comes from pulmonary heart failure.

Organization of exudates with ARDS