Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

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METHODICAL GUIDELINES

for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Respiratory system diseases: chronic obstructive pulmonary disease (COPD), chronic nonspecific lung diseases”

Duration: 3 hours

Approved at the meeting of the Department of Pathological Anatomy
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EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, classification, etiology, pathogenesis, morphology, complications, and outcomes of chronic nonspecific lung diseases (CNSLD). To analyze chronic obstructive pulmonary disease (COPD), including chronic bronchitis (simple, mucopurulent, atrophic, hyperplastic), emphysema (centriacinar, panacinar, paraseptal, irregular), and bronchial asthma. To examine chronic cor pulmonale as a complication of chronic lung disease. To describe the morphological features of chronic bronchitis (hyperplasia of mucous glands, increased Reid index, goblet cell metaplasia, inflammation, fibrosis). To analyze the types of emphysema and their pathogenesis (protease-antiprotease imbalance, oxidative stress). To discuss the role of smoking, air pollution, occupational exposures, and genetic factors (alpha-1 antitrypsin deficiency). To study bronchiectasis (saccular, cylindrical, varicose) and its causes. To examine the complications: respiratory failure, pneumothorax, chronic hypoxemia, polycythemia, and cor pulmonale with right ventricular hypertrophy and failure.

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity, understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

***Know*:**

- The definition and classification of chronic nonspecific lung diseases;
- The etiology, pathogenesis, and morphological features of chronic bronchitis;
- The types of pulmonary emphysema and their morphological characteristics;
- The definition, causes, and morphology of bronchiectasis;
- The morphological changes in chronic cor pulmonale;
- The complications and outcomes of chronic lung diseases.

Be able to:

- Diagnose different types of emphysema based on macroscopic and microscopic features;
- Identify chronic bronchitis on histological specimens;
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

Master:

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

Chronic nonspecific lung diseases, particularly COPD, are among the leading causes of chronic morbidity and mortality worldwide, with smoking being the primary risk factor. These diseases result in progressive respiratory disability, reduced quality of life, and significant healthcare costs. Understanding the morphological changes in chronic bronchitis, emphysema, and cor pulmonale is essential for pulmonologists, internists, general practitioners, and pathologists for accurate diagnosis, assessment of disease severity, and management of complications.

QUESTIONS

1. Chronic obstructive pulmonary disease (COPD). Chronic bronchitis. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
2. Emphysema. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
3. Bronchiectasis. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
4. Bronchial asthma. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes. Morphological features in status asthmaticus.
5. Chronic interstitial lung diseases. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.

COURSE OF THE SESSION

Chronic nonspecific lung diseases (CNSLD) is an older medical term that was historically used to describe a group of long-term respiratory conditions characterized by persistent airway obstruction, inflammation, or structural changes in the lungs, **without a single identifiable cause** (hence "nonspecific").

Conditions typically included under CNSLD:

- Chronic Obstructive Pulmonary Disease (COPD) – Includes chronic bronchitis and emphysema.
- Chronic Bronchitis (without clear obstruction).
- Bronchial Asthma (in some classifications, especially if chronic and severe).
- Diffuse Pulmonary Fibrosis (in some older definitions).

Current equivalent terms are:

- **Chronic Respiratory Diseases (CRDs)** – Broader term including COPD, asthma, etc.
- **Obstructive Lung Diseases** – If airflow limitation is the main feature.

Pathophysiological classification of CRDs

1) Obstructive Lung Diseases. Airway obstruction → Increased airflow resistance

Features:

- Chronic bronchitis (mucus hypersecretion)
- Emphysema (alveolar destruction)
- Asthma (bronchospasm & inflammation)

Key Dysfunction: Impaired bronchodilation, air trapping

2) Restrictive Lung Diseases. Reduced lung expansion → Decreased lung volumes

Features:

- Pulmonary fibrosis (scarring of interstitium)
- ILDs (inflammation → fibrosis)

Key Dysfunction: Reduced lung compliance, impaired gas exchange

Underlying Mechanisms:

1) **Bronchitogenic** (airway inflammation)
2) **Pneumoniogenic** (alveolar damage)
3) **Pneumonitogenic** (interstitial fibrosis)

Chronic obstructive pulmonary disease (**COPD**) or chronic obstructive airways disease (COAD) are commonly-used clinical terms for a group of pathological conditions in which there is chronic, partial or complete, obstruction to the airflow at any level from trachea to the smallest airways resulting in functional disability of the lungs.

The **obstructive pulmonary disease** must be distinguished from **restrictive pulmonary disease**.

Chronic bronchitis and emphysema are quite common and often occur together.

More recently, small airways disease involving inflammation of small bronchi and bronchioles (bronchiolitis) has been added to the group of COPD.

Chronic bronchitis is a common condition defined clinically as persistent **cough** with expectoration on most days for at **least three months of the year** for **two or more consecutive years**. The cough is caused by oversecretion of mucus.

In spite of its name, chronic inflammation of the bronchi **is not a prominent feature**. The condition is more common in middle-aged males than females; approximately 20% of adult men and 5% of adult women have chronic bronchitis, but only a minority of them develop serious disabling COPD or cor pulmonale. Quite frequently, chronic bronchitis is associated with emphysema.

Etiopathogenesis. The two most important etiologic factors responsible for majority of cases of chronic bronchitis are: **cigarette smoking** and **atmospheric pollution**. Other contributory factors are occupation, infection, familial and genetic factors.

Etiopathogenesis of chronic bronchitis

1. **Smoking**. The most commonly identified factor implicated in causation of chronic bronchitis and in emphysema is heavy smoking. Heavy cigarette smokers have 4 to 10 times higher proneness to develop chronic bronchitis. Prolonged cigarette smoking appears to act on the lungs in a number of ways:

2. **Atmospheric pollution**. The incidence of chronic bronchitis is higher in industrialized urban areas where air is polluted. Some of the atmospheric pollutants which increase the risk of developing chronic bronchitis are sulfur dioxide, nitrogen dioxide, particulate dust and toxic fumes.

3. **Occupation**. Workers engaged in certain occupations such as in cotton mills (byssinosis), plastic factories, etc. are exposed to various organic or inorganic dusts which contribute to disabling chronic bronchitis in such individuals.

4. **Infection**. Bacterial, viral and mycoplasmal infections do not initiate chronic bronchitis but usually occur secondary to bronchitis. Cigarette smoke, however, predisposes to infection responsible for acute exacerbation in chronic bronchitis.

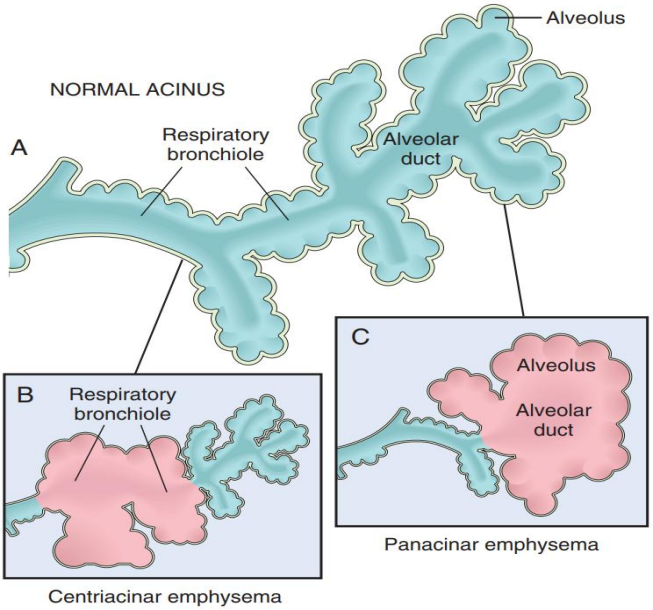
5. **Familial and genetic factors**. There appears to be a poorly-defined familial tendency and genetic predisposition to develop disabling chronic bronchitis. However, it is more likely that nonsmoker family members who remain in the air-pollution of home are significantly exposed to smoke (passive smoking) and hence have increased blood levels of carbon monoxide.

Emphysema

The WHO has defined **pulmonary emphysema** as combination of permanent dilatation of air spaces distal to the terminal bronchioles and the destruction of the walls of dilated air spaces.

Thus, emphysema is defined morphologically, while chronic bronchitis is defined clinically. Since the **two conditions coexist frequently** and show considerable overlap in their clinical features, it is usual to label patients as 'predominant emphysema' and 'predominant bronchitis’.

Classification. Lobule normally is composed of about 5 acini distal to a terminal bronchiole and that an acinus consists of 3 to 5 generations of respiratory bronchioles and a variable number of alveolar ducts and alveolar sacs. By strictly adhering to the WHO definition of pulmonary emphysema, it is classified, according to the portion of the acinus involved, into 5 types: 1) **centri-acinar**, 2) **panacinar** (panlobular), 3) **para-septal** (distal acinar), 4) **irregular** (para-cicatricial), 5) and **mixed** (unclassified) emphysema.



Bronchiectasis is defined as **abnormal and irreversible dilatation of the bronchi and bronchioles** (greater than 2 mm in diameter) developing **secondary to inflammatory weakening** of the bronchial walls. The most characteristic clinical manifestation of bronchiectasis is persistent cough with expectoration of copious amounts of foul-smelling, purulent sputum.

Etiopathogenesis. The origin of inflammatory destructive process of bronchial walls is nearly always a result of two basic mechanisms: **obstruction** and **infection**.

1) **Endobronchial obstruction** by foreign body, neoplastic growth or enlarged lymph nodes causes resorption of air distal to the obstruction with consequent atelectasis and retention of secretions.

2) **Infection** may be secondary to local obstruction and impaired systemic defense mechanism promoting bacterial growth, or infection may be a primary event i.e. bronchiectasis developing in suppurative necrotizing pneumonia.

Bronchiectasis represent the **end stage of a variety of unrelated disorders** and can be divided into localized and diffuse forms.

Localized bronchiectasis often results from **partial or total obliteration of the bronchial lumen** by a neoplasm, foreign body, localized inflammatory process, inspissated mucus, or external compression. It can occur in any area of the lung and follows the branching pattern of the obstructed bronchus. If the source of obstruction is relieved at an **early stage**, the bronchiectatic **changes will regress**; **otherwise**, the

secondary inflammatory and fibrotic changes will render the condition **irreversible**. Localized bronchiectasis is a common finding in patients with middle lobe syndrome, a condition characterized by fixed radiological abnormalities limited to the right middle lobe and/or lingula.

Diffuse bronchiectasis is a consequence of **inflammation and postinflammatory destruction** of airway walls that is usually the result of repeated episodes of infection. This is the form of bronchiectasis often seen in patients with **cystic fibrosis**. The distribution of diffuse bronchiectasis depends on the underlying condition. For example, bronchiectasis patients with cystic fibrosis and allergic bronchopulmonary aspergillosis usually presents with upper lobe predominant disease.

Pathologic changes in bronchiectasis

The disease characteristically affects **distal bronchi and bronchioles** beyond the segmental bronchi.

The pleura is usually fibrotic and thickened with adhesions to the chest wall. Cut surface of the affected lobes, generally the lower zones, shows characteristic **honeycombed appearance**.

The bronchi are extensively dilated nearly to the pleura, their walls are thickened and the lumina are filled with mucus or mucous. The dilated airways, depending upon their gross or bronchographic appearance, have been subclassified into the following different types:

1) **Cylindrical**: the most common type characterized by tube-like bronchial dilatation.

2) **Fusiform**: having spindle-shaped bronchial dilatation.

3) **Saccular**: having rounded sac-like bronchial distension,

4) **Varicose**: having irregular bronchial enlargements.

Pathologic changes in bronchiectasis

Microscopically, fully-developed cases show the following histologic features:

1) The **bronchial epithelium** may be normal, ulcerated or may show squamous metaplasia.

2) The **bronchial wall** shows infiltration by acute and chronic inflammatory cells and destruction of normal muscle and elastic tissue with replacement by fibrosis.

3) The intervening **lung parenchyma** shows fibrosis, while the surrounding lung tissue shows changes of interstitial pneumonia.

4) The **pleura** in the affected area is adherent and shows bands of fibrous tissue between the bronchus and the pleura.

Bronchial asthma

Asthma is a disease of airways that is characterized by increased responsiveness of the tracheobronchial tree to a variety of stimuli resulting in **widespread spasmodic narrowing of the air passages** which may be relieved spontaneously or by therapy.

Asthma is an episodic disease manifested clinically by paroxysms of dyspnea, cough and wheezing. However, a severe and unremitting form of the disease termed status asthmaticus may prove fatal.

Based on the stimuli initiating bronchial asthma, two broad **etiologic types** are traditionally described: **extrinsic** (allergic, atopic) and **intrinsic** (idiosyncratic, non-atopic) asthma. A third type is a mixed pattern in which the features do not fit clearly into either of the two main types.

Grossly, the lungs are overdistended due to over-inflation. The cut surface shows characteristic occlusion of the bronchi and bronchioles by viscid mucus plugs.

Microscopically, the following changes are observed:

1. The mucus plugs contain normal or degenerated respiratory epithelium forming twisted strips called **Curschmann's spirals**.

2. The sputum usually contains numerous eosinophils and diamond shaped crystals derived from eosinophils called **Charcot-Leyden crystals**.

3. The bronchial wall shows thickened basement membrane of the bronchial epithelium, submucosal edema and inflammatory exudate consisting of lymphocytes and plasma cells with prominence of eosinophils. There is **hypertrophy** of submucosal glands as well as of the bronchial smooth muscle.

4. Changes of bronchitis and emphysema may supervene, especially in intrinsic asthma.

Clinical features of bronchial asthma

Asthmatic patients suffer from episodes of acute exacerbations interspersed with symptom-free periods.

Characteristic clinical features are **paroxysms of dyspnea, cough and wheezing**.

Most attacks typically last for a **few minutes to hours**. When attacks occur continuously it may result in more serious condition called status asthmaticus.

The clinical diagnosis is supported by demonstration of circulation **eosinophilia** and sputum demonstration of **Curschmann's spirals** and **Charcot-Leyden crystals**.

More chronic cases may develop **cor pulmonale**.

Chronic interstitial lung diseases

Interstitial lung disease (ILD) is a group of lung diseases characterized by a primary inflammatory process in the **interalveolar pulmonary interstitium** and the development of bilateral diffuse **pneumofibrosis**.

ILD is divided into 2 groups based on **etiology**:

1) ILD with **established etiology** includes **pneumoconiosis** caused by organic and inorganic dust, exogenous allergic alveolitis. Bacteria, fungi, dust containing **antigens of animal and plant origin** are the most common etiological factors. It is widespread among people employed in agriculture, as well as among those working in the textile, pharmaceutical industry, etc.

2) ILD with **unknown etiology** includes idiopathic **fibrosing alveolitis** (**Hamman-Rich disease**), secondary fibrosing alveolitis in rheumatic diseases,

sarcoidosis, fibrosing alveolitis in **Goodpasture's syndrome**, idiopathic pulmonary hemosiderosis, eosinophilic pneumonia, alveolar proteinosis.

Interstitial lung diseases are divided into acute and chronic.

Chronic interstitial lung diseases

Morphological changes in ILD has **3 stages**:

1. Stage of **alveolitis**. It is characterized by infiltration of the interstitium of the alveoli, alveolar ducts, walls of the respiratory and terminal bronchioles with neutrophils, lymphocytes, macrophages, and plasma cells.

2. Stage of disorganization of alveolar structures and **pneumofibrosis**. It is characterized by the destruction of endothelial and epithelial membranes, elastic fibers. Cellular infiltration of the alveolar interstitium increases with subsequent spread to large vessels and perivascular tissue. Diffuse pneumofibrosis develops in the interstitium of the alveoli.

3. Stage of formation of a **honeycomb lung**. An alveolar-capillary block and panacinar emphysema, bronchiolectasis develop, cysts with fibrously altered walls appear in place of the alveoli. It is complicated by pulmonary hypertension, hypertrophy of the right ventricle, heart failure.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

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2. Klatt, E. C. Robbins and Cotran Atlas of Pathology : [with Student Consult Online Access] / Edward C. Klatt. - third edition. - Philadelphia : Elsevier, 2015. - 587 p. : il.

3. Robbins Basic pathology / [ed. by] Vinay Kumar, Abul K. Abbas, Jon C. Aster. - 9th ed. - [Philadelphia] : Elsevier ; Saunders. - 910 p. : ill., scheme, tab.

ADDITIONAL LITERATURE

1. Sobotta. Atlas of human anatomy. [V. 1]. General anatomy and musculoskeletal system / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 400 p. : ill., col. ill.

2. Sobotta. Atlas of human anatomy. [V. 2]. Internal organs / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 259 p. : ill., col. ill.

3. Sobotta. Atlas of human anatomy. [V. 3]. Head, neck, and neuroanatomy / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 370 p. : ill., col. ill.

4. Weir & Abrahams' imaging atlas of human anatomy / ed.: J. D. Spratt [and oth.] ; consultant eds.: J. Weir, P. H. Abrahams. – 5th ed. - [S.l.] : Elsevier, 2017. - xvi, 263 p. : ill.

NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: http://patan.by/menyu/administrativnyie-proczeduryi.html - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: http://www.rosmedlib.ru/ . – Access date: 03/15/2022.

2. Springer Link [Electronic resource] / Springer International Publishing AG. – Access mode: https:link.springer.com. – Date of access: 03/15/2022.