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MORPHOLOGICAL CHANGES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN ELDERLY WOMEN AND COMPARATIVE ANALYSIS.

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Abstract. Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production) and exacerbations due to airway (bronchitis, bronchiolitis) and/or alveolar (emphysema) damage, which cause persistent, often progressive airflow limitation. Exacerbations and comorbid conditions are an integral part of the disease and contribute significantly to the clinical picture. To assess the risk of COPD exacerbations, it is necessary to consider the patient's history of exacerbation frequency, including those requiring hospitalization within the last 12 months. If a patient has had even one exacerbation leading to hospitalization in the previous year (i.e., a severe exacerbation), they should be classified as high risk.
Key words: COPD, alveoli, microscopic, bronchioles, lung tissue.
Калит сузлар: СОУК, алвеолалар, микроскопик, бронхиола, упка тукимаси.
Ключевые слова: ХОБЛ, алвеолы,микроскопические, бронхиола,ткань легких.

Introduction: COPD is a disease that places a heavy burden on society and is of increasing social significance.Currently, COPD is among the most common human diseases, which is due to continuous environmental pollution, increasing smoking rates, and recurrent respiratory infectious diseases. According to the WHO, in 2019, 212.3 million people worldwide suffered from COPD. 3.3 million patients died from COPD, and the DALYs (Disability-Adjusted Life Years) indicator was 74.4 million. The prevalence of COPD was 2638.2 per 100,000 people, and the mortality rate was 42.5 per 100,000 people. In Russia, according to the Ministry of Health, 2.4 million people suffer from COPD. In reality, according to epidemiological studies by the Russian Respiratory Society, this figure is around 11 million, including undiagnosed cases.

COPD is the 3rd leading cause of death worldwide, with approximately 2.8 million deaths annually from COPD, accounting for 4.8% of all causes of death. Both endogenous factors and exposure to environmental factors play a role in the development of COPD. Smoking remains the primary cause of COPD. According to some estimates, in industrialized countries, smoking contributes to mortality in about 80% of men and 60% of women, and in developing countries – 45% of men and 20%. In developing countries, the use of biomass for cooking and heating residential premises has a significant damaging effect on the respiratory organs.

Objective: Morphological changes in chronic obstructive pulmonary disease in elderly women and a comparative analysis.

Materials and Methods: For this purpose, a study was conducted based on archival materials from the Tashkent City Scientific Research Center of Forensic Medicine. Specifically, lung

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tissue samples from 75 women (aged 45-60 years) who died in 2024-2025 and had COPD were morphologically compared and studied.

Occupational hazards, passive smoking, and outdoor air pollution can also play an etiological role. In Europe and North America, the contribution of workplace air pollution to the development of COPD is estimated to be 15-20%. This contribution is likely significantly higher in countries where occupational hazards are less rigorously controlled. Workplace air pollution from biological and mineral dust, gases, and fumes (based on patient self-assessment) has been associated with a higher prevalence of COPD. Endogenous risk factors include genetic, epigenetic, and other patient characteristics, such as bronchial hyperresponsiveness and a history of bronchial asthma (BA), as well as severe respiratory infections in childhood. Moreover, bronchial hyperresponsiveness is a risk factor for COPD development even in the absence of BA; there is also evidence that symptoms of chronic bronchitis may increase the risk of COPD development.

Congenital alpha-1-antitrypsin deficiency is an autosomal recessive inherited disease that predisposes to the development of COPD, and is detected in less than 1% of cases. Other genetic factors predisposing to COPD are complex, and their contribution to disease development is currently not well understood. The development of COPD is associated with the polymorphism of multiple genes, but only a few of these associations have been demonstrated in independent population samples. COPD is characterized by an increased number of neutrophils, macrophages, and T-lymphocytes (especially CD8+) in various parts of the airways and lungs. An increased number of inflammatory cells in COPD patients is found in both proximal and distal airways. During exacerbations, some patients may experience an increase in eosinophil counts.

Oxidative stress, i.e., the release of increased amounts of free radicals in the airways, has a potent damaging effect on all structural components of the lungs and leads to irreversible changes in the pulmonary parenchyma, airways, and pulmonary vessels.

Here's the translation of your text into English:

A significant role in the pathogenesis of COPD is played by the imbalance of the "proteinase-antiproteinase" system. This imbalance arises from either increased production or enhanced activity of proteinases, or from decreased activity or reduced production of antiproteinases. This imbalance is often a consequence of inflammation caused by exposure to damaging substances.

Airflow limitation and pulmonary hyperinflation. Expiratory airflow limitation is the primary pathophysiological abnormality in COPD. It is based on both reversible and irreversible components. Irreversible components include:

1.Fibrosis and narrowing of the airway lumen;
2.Loss of lung elastic recoil due to alveolar destruction;
3.Loss of alveolar support for the lumen of small airways.

Reversible causes include:

1.Accumulation of inflammatory cells, mucus, and plasma exudate in the bronchi;
2.Contraction of bronchial smooth muscle;
3.Pulmonary hyperinflation (PHI) (i.e., increased lung airiness).

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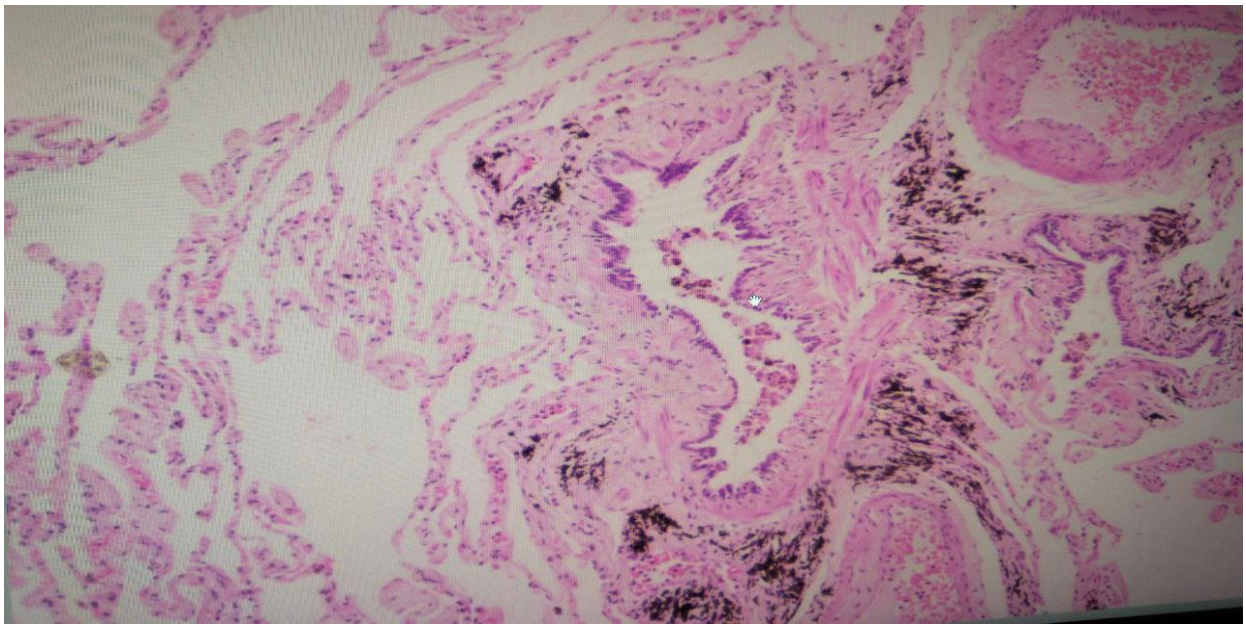
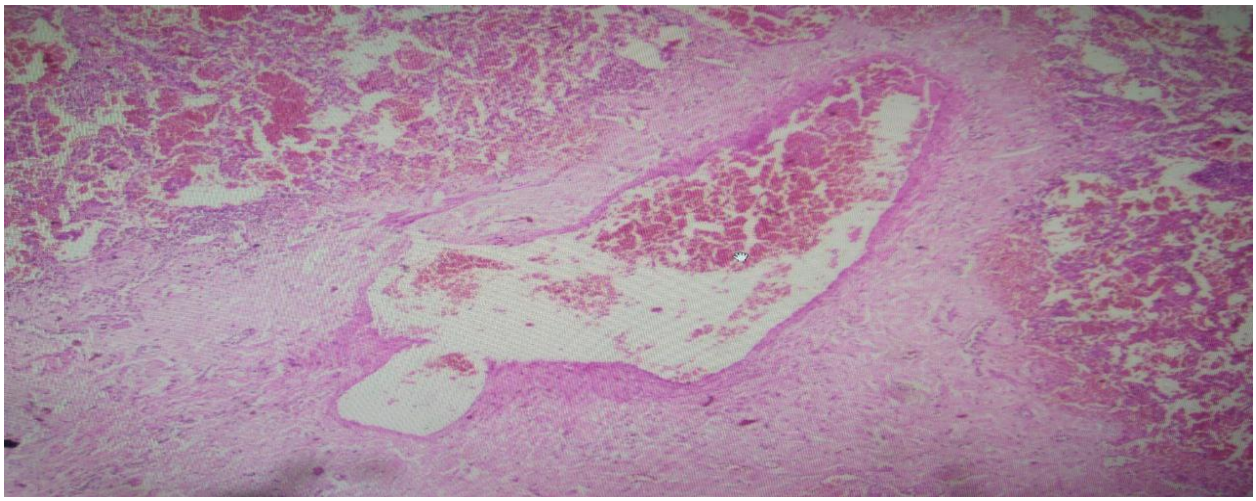
The basis of LGI (Lung Graphic Interpretation) lies in the incomplete emptying of alveoli during exhalation due to loss of lung elastic recoil (static LGI) or due to insufficient exhalation time under conditions of severe expiratory airflow limitation (dynamic LGI).
According to recently conducted studies, narrowing of the lumen and a decrease in the number of terminal bronchioles precede the development of emphysematous destruction of alveoli in both centrilobular and panlobular emphysema.

1- **Image:** Bronchial lumen is widened and contains exudate, peribronchial fibrosis, and vascular congestion, indicating emphysematous widening of the alveoli.



2- Image: The presence of vascular congestion and thickening of perivascular fibrotic tissue, as well as areas of hemorrhage, are identified.



Severe COPD is characterized by the development of hypoxemia and hypercapnia. The main pathogenetic mechanism of hypoxemia is the disruption of ventilation-perfusion relationships – the VA/Q balance (VA - alveolar ventilation, Q - cardiac output). Lung areas

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with a low VA/Q ratio contribute most significantly to the development of hypoxemia. The presence of areas with an increased VA/Q ratio leads to an increase in physiological dead space, consequently requiring an increase in overall lung ventilation to maintain a normal level of partial pressure of carbon dioxide in arterial blood (PaCO2). Blood flow shunting in COPD usually does not occur, except in particularly severe cases of exacerbation requiring respiratory support.

Pulmonary hypertension can develop in the later stages of COPD due to hypoxia-induced spasm of small pulmonary arteries, which ultimately leads to structural changes: intimal hyperplasia and later smooth muscle layer hypertrophy/hyperplasia. An inflammatory reaction, similar to that in the airways, and endothelial dysfunction are observed in the vessels. Progressive pulmonary hypertension can lead to right ventricular hypertrophy and ultimately to right heart failure (cor pulmonale).

A characteristic feature of COPD is the presence of systemic effects, the main ones being systemic inflammation, cachexia, skeletal muscle dysfunction, osteoporosis, cardiovascular events, anemia, depression, and others. The mechanisms underlying these systemic manifestations are quite diverse and not yet fully understood. It is known that hypoxemia, smoking, sedentary lifestyle, systemic inflammation, and others play an important role among them.

COPD and impaired lung function are independently associated with an increased risk of cardiovascular events. Reduced lung function (FEV1) is as strong a risk factor for cardiovascular mortality as major cardiovascular risk factors. According to a study of individuals aged 45-60 years with moderate bronchial obstruction, a 10% decrease in FEV1 was associated with a 14% increase in all-cause mortality, a 28% increase in cardiovascular mortality, and a 20% increase in the risk of developing coronary heart disease.

The development of exacerbations is a characteristic feature of COPD progression. COPD exacerbations are one of the most frequent reasons for patients to seek emergency medical care. Frequent exacerbations in patients with COPD lead to prolonged deterioration (up to several weeks) of respiratory function and gas exchange parameters, faster disease progression, a significant decline in patients' quality of life, and are associated with substantial economic costs for treatment.

Some of these features include:Characteristic black color of the lungs. This arises from the accumulation of soot and other combustion products in the interstitial tissue of the lungs.Bullae. These are sac-like protrusions that appear due to the destruction of the interalveolar septa. Bullae are a consequence of progressive emphysema, which leads to a reduction in the lung's embryonic architecture and impaired gas exchange. Changes in the bronchial system. On a lung section, it can be seen that the bronchial stumps protrude 1-2 millimeters above the cut surface. Inside these stumps, there may be mucous or purulent content, indicating the presence of chronic bronchitis, a key component of COPD. Proliferation of connective and fibrous tissue. This reflects a chronic inflammatory process and the body's attempt to compensate for the destruction of the alveolar structure.Chronic cellular infiltration in lung tissue. This indicates a prolonged reaction to inflammation, sustaining the active course of the disease.

Conclusion: The study was conducted based on archival materials from the Scientific Center of Forensic Medical Examination of Tashkent. Specifically, through comparative

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morphological examination of lung tissue samples from 75 women (aged 45-60 years) who died in 2024-2025 and had COPD, it was established that in the majority of them, the bronchial epithelium underwent metaplasia, forming precancerous morphological changes.

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