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MORPHOFUNCTIONAL CHANGES IN THE WALL OF THE BRONCHIAL TREE OF THE LUNGS IN DIABETES MELLITUS

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Abstract:

Based on numerous studies, it has been established that the leading role in the development of chronic damage to the airways, along with violations of immunological reactivity, belongs to mucociliary insufficiency and is characterized by changes in the bronchial tree, which are accompanied by a violation of the secretary function of the bronchi. At the same time, morphofunctional disorders naturally occur, primarily in the epithelial layer of the bronchial tree. In the works of many authors, it is indicated that in conditions of chronic inflammation in the epithelium there are violations of regenerative ability, cell proliferation and differentiation, which plays an important role in damage to mucociliary clearance and, as a consequence, in violation of the protective function of the respiratory system.

Despite numerous studies of the respiratory organs in patients with diabetes mellitus, the data obtained are contradictory. In the literature available to us, no detailed morphometric study of the epithelial lining of the lower airways in diabetes mellitus has been found. In recent years, more and more attention has been paid to the problem of chronic obstructive pulmonary disease (COPD) in diabetes. This is due not only to the increase in morbidity, but also to the high mortality rate in patients with this disease. To date, COPD is an extremely serious medical and social problem.

Keywords: morphofunctional changes, bronchial asthma, symptoms of cough, industrial inhalation hazards

When examining biopsies, not all pathological signs of damage to the EP of the airways can be used for diagnosis. It was found that at the level of the lobular and segmental bronchi, there is a great similarity with changes in EP in various chronic lung pathology.

The development of pronounced morphofunctional changes in the lungs, caused primarily by tobacco smoking, affects the formation of the disease. The medical aspect lies in the nature of the pathological process in COPD, which consists in inflammation in the wall of the bronchial tree, the parenchyma of the lungs and pulmonary vessels, which cannot be treated as successfully with anti-inflammatory drugs as it occurs with bronchial asthma. In the course of the disease, irreversible changes occur on the part of all components of the respiratory system, which also cause less effective treatment. In addition, this pathology is characterized by the presence of systemic effects, the approach to correction of which differs from that of other lung diseases.

The incidence of COPD is high, the number of patients with this disease is significantly higher than registered in outpatient institutions in diabets. Unfortunately, COPD is mostly diagnosed in the later stages of the disease. Patients with COPD have long considered themselves healthy people, and the symptoms of cough, sputum separation and the appearance of shortness of breath during exercise (early stages of the disease) are explained by some other reasons.

According to statistics:

- the prevalence of COPD in the world in people over 40 years of age is up to 10%;
- as of 2002, there were about 600 million COPD patients in the world;
- COPD is often not diagnosed – only 25-30% of cases are detected in Europe and the USA;
- COPD affects more than 16 million Russians;
- the prevalence of COPD is steadily increasing, and in women much faster than in men;
- according to the prevalence, COPD is the fourth cause of death after cardiovascular pathology, lung cancer and cerebral vascular diseases;
- COPD is the only leading cause of death, the prevalence of which is steadily increasing;

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- in the 1990s, COPD ranked 5th among the causes of death worldwide and 4th in developed countries;

- 200-300 thousand people die from COPD annually in Europe and 2.74 mllllon people in the world (2000);

- by 2020, COPD will take the 3rd place among the causes of death and will lead to 4.7 million deaths per year.

Risk factors for COPD are recurrent infection of the respiratory tract, hyperreactivity of the respiratory tract, impaired lung growth and development, genetic predisposition, industrial inhalation hazards, air pollution, low socio-economic level. But the most important role in the development and progression of the disease is played by smoking.

COPD is an inflammatory disease, while macrophages and neutrophlls, as well as CD8+ T lymphocytes, mainly participate in the development of the inflammatory process. Of the inflammatory mediators characteristic of COPD are leukotriene B4 and interleukin 8. This is due to the difference between COPD and bronchial asthma, in which the characteristic inflammatory cells are eosinophils and CD-4+ T lymphocytes, and inflammatory mediators are leukotriene D4, interleukins 4 and 5. In COPD, the characteristic morphological consequences of inflammation are epithellal metaplasia and the development of sclerotic changes in the bronchial wall, and in bronchial asthma - desquamation of the epithellum and thickening of the basement membrane. Also, in COPD, a number of inflammatory mediators with systemic effects are isolated (for example, tumor necrosis factor alpha) in diabetes.

It is important to emphasize that oxidative stress plays an important role in the formation of destructive processes in the lung tissue, participating not only in the pathogenesis of the disease, but also having a systemic effect.

Thus, inflammatory changes lead to changes in the wall of the bronchial tree, which are caused by the pathological action of inhaled damaging factors and violate mucocillary clearance, change the elastic properties of the bronchi in the parenchyma of the lungs, which leads to emphysema, as well as in the pulmonary vessels affected during inflammation.

Inflammation in the respiratory system from a pathophysiological point of view leads to reversible (bronchospasm, edema of the bronchial wall, quantitative and qualitative violation of bronchial secretion, dynamic hyperinflation during exercise) and irreversible changes (sclerosis of the bronchial wall, expiratory declIne of small bronchi on exhalation, emphysema).

At the same time, the degree of severity of various changes differs in different patients. In this regard, when emphysema and debllitating shortness of breath come to the fore in the clinical picture, the emphysematous type of COPD is mainly distinguished, and with predominant signs of bronchial tree damage with corresponding clinical manifestations – bronchial obstruction, cough, sputum - bronchitic type. It is recommended to make these phenotypes in the diagnosis. COPD is characterized by a systemic effect of the disease (unllke bronchial asthma). The effect of inflammatory mediators and oxidative stress products is not llmited to the lung tissue. First of all, skeletal muscles are affected, as a result of which the patient loses muscle mass and strength, and myocytes undergo pronounced dystrophic changes. This leads to an even greater restriction of physical activity in COPD patients due to the low anaerobic threshold. Patients with COPD are characterized by a higher risk of fractures and a decrease in bone density, which is due to the elderly age of patients, smoking and a reduced level of physical activity.

The results of a retrospective analysis of the database of patients with COPD who received inhaled glucocorticosteroids (IGCS) and /or bronchodllators suggest that the risk of fractures may be more due to the underlying respiratory disease than the use of IGCS. About 66% of COPD patients included in the TORCH study suffered from osteoporosis or osteopenia prior to inclusion in the study (according to WHO criteria).

In patients with COPD, changes in the cardiovascular system are of great importance. Of course, COPD is a risk factor for the development of various diseases of the cardiovascular system. At the same time, the most important factor in the development of COPD – smoking is also a risk factor for the development of atherosclerotic vascular and heart damage. The development of respiratory fallure in severe stages of COPD development is formed by changes in the right departments with the formation of a "pulmonary heart".

COPD treatment consists of reducing the impact of risk factors, primarily tobacco smoking, as well as drug treatment, rehabllitation measures and patient education. The most effective measure to reduce the risk of COPD progression (evidence level A) is smoking cessation.

One of the most difficult components of the COPD management program is the incentive to quit smoking. The most effective methods are the conversation of a medical professional, the use of nicotine-substituting

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drugs and antidepressants (bupropion and nortriptyline).

Drugs that dilate the bronchi by blocking the vagus nerve receptors (cholinolytics) are quite effectively used in the treatment of bronchial asthma. This group includes ipratropium (atrovent), which is widely used in the treatment of chronic obstructive pulmonary disease. Ipratropium enhances and prolongs the bronchodilating effect of short-acting beta-2 agonists. Both of these active substances relax the smooth muscles of the bronchial tree, but by acting on different targets, resulting in increased antispasmodic action. The combined drug berodual is effective even with insufficient effect of any of its components. The combination of complementary components makes it possible to obtain a pronounced broncholytic effect when using a dose of phenoterol twice as low as in berothek H (50 mcg), which reduces the likelihood of complications to a minimum: side effects are extremely insignificant and occur mainly with an overdose of the drug.

The sensitivity of bronchial M-cholinergic receptors does not weaken with age, which allows the use of cholinolytics in elderly COPD patients. Anticholinergic drugs (atropine, ipratropium bromide and oxytropium bromide) interact with all three types of muscarinic receptors. At the same time, if the blockade of M1– and M3–receptors leads to bronchodilation, then the blockade of M2–receptors, accompanied by increased release of acetylcholine into the synaptic cleft, may to some extent reduce the beneficial effects of the blockade of postsynaptic M3-receptors.

In recent years, the long-acting M-cholinolytic tiotropium bromide (spiriva), which is a quaternary ammonium compound similar in chemical structure to ipratropium bromide, has been widely used in clinical practice. Both drugs are practically not absorbed through the mucous membrane of the oropharynx and respiratory tract and are characterized by insignificant oral bioavailability, which explains the absence or minimal frequency and severity of systemic anticholinergic effects when they are inhaled. Spiriva has a unique kinetic selectivity with a predominant effect on M1– and M3–receptors. The drug demonstrates a comparably high degree of affinity for all types of muscarinic receptors, however, the dissociation of the drug with M1– and especially with M3-receptors is significantly slowed down, which causes a prolonged blockade of cholinergic bronchoconstriction. On the contrary, the dissociation of tiotropium with M2 receptors is significantly faster, which suggests the kinetic selectivity of the drug.

Thus, as a result of studying the morphological and morphometric parameters of the epithelium of the bronchial mucosa, we found a violation of the structural barrier of the mucosa of the lower airways in the form of a decrease in OPEP, which indicates the predominance of atrophic transformations in the group of rats with ESD. In addition, in the group of rats with ESD, we diagnosed a decrease in ESP, which implies a decrease in physical protective factors in the form of a decrease in mucus secretion by secretory cells of the bronchial lining.

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