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SOME RISK FACTORS FOR PROGRESSION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract
The increase in the number of methods for diagnosing COPD and their constant improvement, the need to establish an accurate diagnosis in the early stages raises the question of the urgent need for researchers to use a minimal but sufficient set of studies. The use of each subsequent method should ensure the obtaining of data that could not be established using the previous one. When comparing different methods, one should find out their limits and capabilities, advantages and disadvantages, the possibility of replacing them with less invasive ones, as well as the reliability and value of the information obtained.

Keywords COPD, FEV1, TGF-β1, α1-antitrypsin.

INTRODUCTION

Chronic obstructive pulmonary disease(COPD) is a disease that has become one of the causes of morbidity and mortality throughout the world. This disease is characterized by the fact that it occurs as an exacerbation and is not always treatable. This article presents an analysis of literature data over the past 10 years on risk factors and pathogenetic aspects of chronic obstructive pulmonary disease [1].

X-ray comparisons, and in recent years, biopsy comparisons carried out in the treatment of many thousands of patients, have shown that in specialized centers the error rate is 4-5% and tends to decrease. Correct use of standard examination methods for patients with COPD, including, first of all, spirometry, in non-specialized general practitioners' offices, allows us to increase the percentage of correct diagnoses to 80–85%. The use of various biopsy options in specialized pulmonology institutions helps to increase this percentage to 95-96% [2,3].

As practice shows, general practitioners often make a diagnosis of COPD based on such shaky signs as shortness of breath, physical and radiological data, especially in patients with frequent exacerbations of lung diseases, without taking into account risk factors for the onset and progression of the disease [4].

Diagnosis of COPD using the spirometric method is gaining new positions every year. Currently, a classification of COPD has been adopted, in which the main indicator of the severity of the disease is FEV1, all values of which relate to post-bronchodilation. The work of the last decade has

confirmed the correctness of the fundamental principles about the decisive role of the FEV1 indicator in the diagnosis of COPD. However, in recent years, justified concerns have emerged that some overdiagnosis of this disease is possible, especially when trying to take into account the so-called microsymptoms and symptoms that cannot be accurately recorded and measured [5].

I would like to note that reproaches against modern pulmonology, as well as other medical disciplines, for departure from clinical thinking are only fair when the use of a particular technique becomes an end in itself, and the data obtained are not scrupulously studied and generalized. According to experts, the diagnosis of COPD is possible if the patient's shortness of breath is more pronounced than that of healthy people of the same age and gender, and the exacerbation of the infectious process significantly disrupts the patient's lifestyle and if the malaise is long-lasting [1,5].

Combating risk factors is the most important strategic goal in the prevention and treatment of COPD. There remains a nihilistic attitude towards COPD among a wide range of physicians due to the disappointing results regarding primary and secondary prevention of COPD, in particular regarding the possibility of eliminating factors causing the initiation and progression of COPD. Identification of risk factors is an important step towards developing strategies for the prevention and treatment of any disease. Risk factors for COPD are divided into two groups: exogenous and endogenous [6].

In addition, the prevailing opinion is that in most cases, the patient causes the disease to himself. Thus, smoking continues to be the cause of COPD and other serious diseases. It has been shown that smokers have a higher prevalence of symptoms of respiratory dysfunction, a greater decline in annual FEV1, and a higher mortality rate compared to non-smokers. The results of twenty-five-year observations of smokers in the general population showed that a large proportion of smokers develop COPD [7].

Risk factors for COPD include genetic predisposition; the most studied genetic disorder that may cause the disease is alpha-1-antitrypsin deficiency, which is the main cause of emphysema in non-smokers, organic and inorganic industrial hazards, household chemicals, and poorly ventilated areas. In addition, insufficient growth and development of the lungs in the perinatal period, oxidative stress, gender, age, respiratory tract infections, a history of tuberculosis, socioeconomic status, poor nutrition, and comorbid conditions are considered risk factors for COPD [7,8].

It is known that COPD is a polygenic inherited disease. In particular, deficiency in the production of α1-antitrypsin, the most important circulating inhibitor of serine proteases, is a documented risk factor for the development of COPD. Although α1-antitrypsin deficiency is a recessively inherited and relatively rare disease, this disease is a clear example illustrating the interaction between genotype and environmental factors leading to the development of COPD [9].

Scientists have conducted a number of studies to draw parallels between hereditary biological defects and the risk of developing COPD. In particular, the role of transforming growth factor β1 (TGF-β1), microsomal apoxide hydrolase 1 (mEPHX1), and tumor necrosis factor α (TNFα) in the pathogenesis of COPD was studied. However, the unconditional role of these factors in the pathogenesis of COPD has not been proven [7,9].

Infectious agents (viral and bacterial agents) may play a role in both the initiation of COPD and the progression of the disease through exacerbations. Researchers have proven the connection between severe infectious lung diseases suffered in childhood and an increased risk of clinically significant COPD in adulthood. A high susceptibility to viral infections of the respiratory tract may be associated with birth weight, which is also a risk factor for the development of COPD [10,11]. One of the risk factors for the development of COPD may be individual underdevelopment of the lungs in the perinatal period.

The course of COPD varies among patients and depends on the severity of symptoms (especially breathing and decreased physical activity), systemic manifestations, and comorbidities that contribute to airflow limitation. At the same time, COPD itself has a number of systemic (extrapulmonary) manifestations that lead to the development of comorbid conditions. [12]

The statement that bronchial asthma may be a risk factor for the development of COPD is not conclusive. COPD can coexist with bronchial asthma. It has been established that in bronchial asthma, a sensitizing antigen promotes inflammation through CD4+ T lymphocytes and eosinophils. In this case, the obstruction is reversible. Whereas in COPD, the damaging agent promotes inflammation through CD8+ T lymphocytes, macrophages and neutrophils, and the obstruction is only partially reversible [13].

In recent years, malnutrition, or malnutrition or trophological deficiency observed in patients with COPD, has been the subject of close study, due to the fact that it is considered by scientists as an independent unfavorable factor that aggravates the prognosis and course of the disease [14]. The cause of malnutrition is probably the progressive loss of muscle mass in COPD, as well as muscle weakness due to increased apoptosis or muscle inactivity.

It is believed that post-transplant osteoporosis is one of the unfavorable consequences of immunosuppressive therapy (taking cytostatics and glucocorticosteroids). However, in patients with so-called terminal pulmonary pathology, including COPD, there are a number of risk factors for the development of osteopenic syndrome: hypogonadism, physical inactivity, poor nutritional status leading to vitamin D deficiency and calcium absorption, long-term therapy with glucocorticosteroids, hypoxemia, systemic inflammation [15].

Systemic manifestations of COPD, in addition to musculoskeletal disorders associated with apoptosis and hypodynamic atrophy, include depression, anemia (usually normochromic, normocytic), depression, diabetes, and sleep disturbances. These combinations obviously worsen the prognosis of patients with COPD [16].

Thus, COPD is a disease that (according to the definition of GOLD experts) can be prevented and treated by carefully studying the comorbid background, conducting a differential diagnosis of the severity of comorbid conditions in each patient with obstructive syndrome. The strategy to combat COPD should include early identification of patients with COPD, timely diagnosis, identification and monitoring of factors contributing to the progression of the disease.

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