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Various Methods of Treatment of Chronic Obstructive Pulmonary Disease

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Abstract: Many of the risk factors for COPD come from the external environment and the main one is smoking (both active and passive). Also, an important influence on the development of COPD is exerted by: occupational hazards (constant contact with fine biological and mineral dust, work in polluted and dusty rooms), air pollution of residential premises as a result of the use of solid fuel for room heating and cooking, air pollution outside working and residential premises. Among the professions with an increased risk of COPD are miners, builders and workers in the metallurgical industry. In addition, internal factors play a sufficient role in the development of COPD – bronchial hyperreactivity, the presence of bronchial asthma in a person or his close relatives, frequent bronchitis in childhood, and a slightly less common deficiency of alpha—antitrypsin.

Keywords: chronic inflammation, pulmonary function, chronic respiratory, bronchial M-cholinergic receptors.

An important difference between COPD and other chronic lung diseases is irreversible obstruction of the airways, which develops due to narrowing of their lumen, fibrous changes, loss of elasticity of lung tissue. The main cause of shortness of breath in COPD is the formation of "air traps" that develop due to impaired emptying of the alveoli due to low elasticity of the lung tissue. It is the formation of "air traps" that leads to the development of shortness of breath, an increase in respiratory rate, which again leads to incomplete emptying of the alveoli, forming a vicious circle of lung tissue damage.

COPD affects not only the lung tissue. Due to the constant lack of oxygen and stagnation of sputum in the alveoli, patients with COPD are characterized by the development of chronic inflammation, damage to the cardiovascular system, and a long-term current disease can lead to the development of clinical depression.

Of crucial importance is the study of the function of external respiration with the determination of FEV1 and the Tiffno index (FEV1/FVC) with mandatory bronchodilation test with both a short-acting β2-agonist and ipratropium bromide (atrovent) to prove irreversible and only partially reversible airway obstruction.

COPD treatment begins with the cessation of exposure to polyutants, primarily with the cessation or, if this is not possible, a sharp restriction of smoking, and training of patients according to special educational programs, as well as with the professional orientation of the patient with the exception of professional polyutants.

The nature of therapeutic measures varies somewhat depending on the phase of the process: remission or exacerbation.

Meanwhile, for a number of years, doctors in various countries have noted higher efficacy and higher safety of the use of AHP, especially tiotropium, before β2-agonists and even more so before xanthine preparations. This is explained as follows:

1. COPD is characterized by an increase in the bronchomotor tone of the vagus nerve, which plays a major role in the development of bronchial smooth muscle spasm and hypersecretion. Thus, AHPs act pathogenetically to the greatest extent, reducing not only bronchospasm, but also hypersecretion. This explains the advantages of AHP in COPD over other bronchodilators.
2. The initial effectiveness of AHP does not decrease with prolonged regular use, that is, they do not have tachyphylaxis (a rare positive quality of the drug), along with this, there is practically no withdrawal syndrome.
3. The sensitivity of bronchial M-cholinergic receptors (unlike β2-adrenoreceptors) does not weaken with age. This is especially important because COPD is a "privilege" of the elderly.

AHPs are practically not absorbed and do not cause systemic side effects, and therefore can be used in concomitant cardiovascular pathology (coronary artery disease, arterial hypertension). They have absolutely no cardiotoxic effect.

In patients over 50 years of age, the annual decrease in FEV1 sharply slowed down under the influence of treatment: the average level of reduction in post-bronchodilatory FEV1 was 38 ml, approaching the physiological age decrease of this indicator, compared to 58 in the control group. Along with this, there was a more significant improvement in the quality of life and a reduction in the risk of developing exacerbations of COPD.

The inflammatory nature of COPD justifies the use of GCS, mainly iGCS, in this disease. GOLD-2008 [13] provides: "Long-term therapy of iGCS is indicated for patients with clinically significant symptoms with FEV1 < 50% of due (stages III and IV) and recurrent exacerbations (for example, three in the last 3 years)." The use of iGCS (along with bronchodilators) is especially indicated in exacerbation of COPD. Such patient with severe respiratory insufficiency, when they are unable to synchronize the inhalation with the activation of the aerosol generator, nebulizer therapy of iGCS is indicated. Preference should be given to topical iGCS budesonide (pulmicort), which practically has no systemic effect. With a particularly severe course of COPD, when due to shallow breathing and widespread bronchial obstruction, the inhalate is unable to penetrate into the shallow airways even when using a nebulizer, systemic GCS per os (30-40 mg) or intravenous GCS is prescribed for 10-12 days. After that, bronchial patency improves, and iGCS have their effect.

It was possible to increase the effectiveness of iGCS in stable COPD by combining iGCS with long-acting β2-agonists (salmeterol or formoterol). In this case, there is a mutual enhancement of the effects of iGCS and long-acting β2-agonists: iGCS increase the expression of β2-adrenoreceptors, and β2-agonists activate receptors for GCS. Thus, long-acting β2-agonists make it possible to overcome steroid resistance in COPD, and therefore the combination of these drugs has a synergistic effect. It is not surprising that the treatment of COPD with combined drugs turned out to be much more effective than using each component in the form of monotherapy .

Fixed combinations of long-acting β2-agonists and iGCS have become widespread: seretide (a combination of salmeterol with fluticasone propionate at a dose of 50/500 mcg) and symbicort (a combination of formoterol with budesonide at a dose of 9/320 mcg). Both the one and the other drug is used 2 times a day.

Initially, these drugs were used for the treatment of AD, but later for the treatment of COPD. In COPD, both drugs can be prescribed with equal success. At the same time, with AD, symbicort has some advantage, which serves not only for prevention, but also for the relief of asthma attacks due to the fact that formoterol, which is part of it, begins to have a bronchodilatory effect after 2-3 minutes.

Indeed, the addition of formoterol to tiotropium significantly improved the results of treatment, in particular, higher indicators of pulmonary function were achieved compared to both tiotropium monotherapy and the combination of salmeterol with fluticasone. The same high treatment results

were achieved with the addition of iGCS to therapy, that is, with the addition of seretide to tiotropium, although this position still needs to be confirmed with the help of large-scale evidence-based studies.

Bronchodilators used in the stable course of COPD are represented by short-acting anticholinergic drugs (ipratropium bromide) and long-acting (tiotropium bromide), p2.short-acting agonists (phenoterol, salbutamol, terbutalin) and long-acting (formoterol, salmeterol) and methylxanthines (theophylline, aminophylline, theopec, theo-tard). Considering that parasympathetic tone is the leading reversible component of bronchial obstruction in COPD, inhaled administration of anticholinergic drugs is advisable for almost all patients with COPD.

Inhaled glucocorticosteroids (IGCS) are added to routine bronchitis therapy in COPD patients with significant clinical symptoms with FEV1<50% of the due (stages III and IV -severe and extremely severe COPD) and with recurrent exacerbations of the disease in the anamnesis that required the appointment of systemic glucocorticosteroids and/or antibiotics (evidence level A). The anti-inflammatory effect of IGCS is associated with an inhibitory effect on inflammatory cells and their mediators, the use of IGCS increases the sensitivity and number of smooth muscle P-receptors, increases the synthesis of anti-inflammatory proteins, helps to stabilize cell membranes and reduce vascular permeability. Unlike systemic steroids, IGCS are characterized by rapid inactivation and a short half-life from blood plasma. It is also important that the therapeutic effect of IGCS is utopian in nature and develops directly in the bronchial tree.

It is known that the diaphragm accounts for more than 90% of the work on the implementation of inspiration. At the same time, due to progressive hypoxemia and tissue hypoxia, metabolic processes in this vital muscle for breathing are significantly disrupted, which inevitably accelerates and aggravates the process of progression of pulmonary insufficiency and CHL. Therefore, percutaneous electrical stimulation of the diaphragmatic muscle is indicated for patients with chronic respiratory (pulmonary) insufficiency to train the respiratory muscles.

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