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**Specificity of Risk Factors in Cerebral Circulatory and Cognitive Impairment**

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**Abstract:**  In recent years, comorbidity has been considered one of the most important problems in therapeutic practice. Its comprehensive study is carried out using epidemiological, genetic, immunological, clinical-functional and other methods to clarify the general biological patterns of a "non-random" combination of a number of diseases - syntropy.

**Keywords:** cerebrovascular disease, chronic obstructive pulmonary disease, clinical practice, cognitive impairment.

In particular, it has been established that a decrease in pulmonary functions is closely associated with an increase in mortality in ischemic stroke (IS) [10], as well as with an increase in asymptomatic cerebrovascular disorders, even among people who have never smoked [11]. An inverse linear relationship was found between the incidence of IS and forced expiratory volume in the first second (FEV1) [2]. It has been shown that in patients with COPD, compared with the general population, the incidence of hemorrhagic stroke increases by 1.3 times, IS by 1.2 times, and subarachnoid hemorrhages by 1.46 times [13]. In addition, the frequency of detection of chronic cerebral ischemia in patients with COPD is almost 3 times higher than in persons without this pathology (20 and 7.4%, respectively). It has been proven that dyscirculatory disorders in the cerebral basin develop already in the early stages of COPD [4]. As the disease progresses, remodeling of the vascular system of the brain occurs, its stiffness increases, blood flow velocity in the extra- and intracerebral arteries decreases, and the effectiveness of the mechanisms of autoregulation of the cerebral blood-cerebral pool is closely related to the severity of bronchial obstruction, arterial hypoxemia, hypercapnia, and deteriorating rheological properties. blood [10]. Neurological symptoms in these disorders are not limited to cognitive disorders and are manifested by various pathological syndromes of damage to the central nervous system (CNS), the severity of which depends on the severity of COPD [7].

The results of studies of vascular comorbidity in COPD are widely presented in the scientific literature [1]. Most of them are devoted to various pathophysiological and clinical aspects of the combination of COPD with cardiac pathology and arterial hypertension [8]. At the same time, there are only a limited number of works that consider the pathogenetic relationships between COPD and various clinical variants of CVD [9]. Currently, cerebrovascular diseases, as well as chronic obstructive pulmonary disease, remain the dominant cause of death among the adult population and are associated with large economic losses for the state. The review presents literature data on the problem of comorbidity of chronic obstructive pulmonary disease (COPD) and cerebrovascular disease (CVD). The main aspects of their pathophysiological relationships and their significance for clinical practice are considered.

Chronic systemic inflammation (CSV) is of great importance in the development of extrapulmonary, including cerebrovascular, manifestations of COPD. The spectrum of pro-inflammatory cytokines produced in this case leads to damage and death of neurons, as well as glial elements [8]. In patients with COPD, more often than in the general population, structural changes in the white matter of the brain are formed, the volume of gray matter decreases, and cerebral microhemorrhages develop [9]. These changes contribute to the development of neurological deficits and psychosomatic disorders [10]. In COPD patients, in the pathogenesis of cerebrovascular comorbidity, in addition to CVD, a number of typical pathological processes are of great importance, the implementation of which is associated with the effect of tissue hypoxia and oxidative stress products on molecular and cellular targets of the CNS - obligate manifestations of progressive respiratory failure. An analysis of the literature data indicates an insufficient amount of information on various aspects of respiratory and cerebrovascular syntropy, which dictates the need for an in-depth study of this problem.

COPD is classified as a multisystem disease, one of the manifestations of which is the defeat of the central nervous system. It has been established that about 46% of the white matter of the brain in COPD has signs of microstructural changes, which, in turn, is a diagnostic marker of cerebrovascular pathology [3]. It has been proven that tobacco smoking plays an important role in the violation of the microstructural integrity of the white matter [4]. However, brain damage in COPD cannot be fully explained by exposure to tobacco smoke. It has been shown that more pronounced changes in the white matter are associated primarily with a decrease in lung volumes, including in non-smoking patients [5]. There is evidence that a high level of CRP in blood plasma is an independent predictor of structural white matter lesions [6].

According to voxel-based morphometry, patients with COPD have a decrease in the volume of the gray matter of the brain and most subcortical nuclei, which is in a negative correlation relationship with the partial pressure of oxygen in the peripheral blood and positively with the duration of the underlying disease [7]. Moreover, a direct relationship was found between these changes and the Gensler index [19]. A decrease in the volume of the thalamus in COPD was associated with an increase in respiratory movements, and a decrease in the volume of the right anterior insular lobe was associated with the development of cardiovascular complications in this pathology [2]. In addition, in patients with COPD, damage to the anterior insular lobe is closely associated with cardiac arrhythmias [8].

Compared with the general population, among patients with COPD, there is a higher prevalence of cerebral microbleeds [relative risk (RR) - 1.7], which correlates with the severity of broncho-obstructive syndrome and the presence of concomitant microangiopathy. In severe COPD, the risk of microbleeds increases dramatically (RR - 3.3). Moreover, these indicators are not affected by gender, age, tobacco smoking experience and lipid profile [9]. Therefore, COPD is an independent risk factor for cerebral microbleeds. In addition, in this pathology, they are more often localized in the deeper structures of the brain, which is associated with the high prevalence of cerebral artery atherosclerosis and hypertensive angiopathy among this group of patients. It has been proven that the severity of brain damage increases significantly in COPD patients with frequent exacerbations [6]. Cerebral microbleeds are more frequently detected in COPD patients who quit smoking than in smokers without obstructive airway disease. These data indicate that tobacco smoking is not the only mechanism of cerebrovascular comorbidity in COPD [9].

According to pathomorphological studies, chronic ischemia of the brain against the background of multifocal atherosclerosis is detected in 40.2% of patients with COPD in 40.2% of cases [6]. In another similar study, the most common variant of comorbidity in men was a combination of COPD and chronic forms of CVD [2], which emphasizes the relevance of the problem of respiratory and cerebrovascular comorbidity [13]. The processes of ATP destabilization are of great importance in the pathogenesis of CVD. When the thickness of the fibrous cap is less than 65 μm and the volume of the lipid core increases by more than 30%, the plaque ruptures and atherothrombotic IS develops [14]. In addition, in patients with COPD, the thickness of the intima-media complex has an inverse correlation with the level of FEV1 [15].

In COPD, multidirectional disturbances in the reflex excitability of the spinal and supraspinal parts of the brain are observed. The most pronounced neurodynamic disorders occur in moderate and severe COPD: hyperreflexia is diagnosed in 72.3% of patients, and hyporeflexia in 13.7%. With increasing respiratory failure in these patients, there is a slowdown in the central conduction time along the efferent pathways, which indicates a violation of the functional state of the corticospinal tract. It has also been shown that dysfunction of the spinal and supraspinal parts of the brain is closely related to the severity of bronchial obstruction and weakness of the respiratory muscles [16].

The most severe forms of respiratory and cerebrovascular comorbidity are combinations of COPD and various types of stroke, manifested by significant functional and anatomical limitations. According to the authors, this problem requires separate coverage.

However, there is no data on what structural changes in COPD occur in the vessels of the brain, how pronounced they are and how they change depending on the stage of the disease. It is known that during exacerbation and progression of the disease, increasing hypoxia, intoxication, the impact of infectious factors on the vascular wall and nerve cells, as well as dyscirculatory disorders of a congestive nature due to right ventricular failure lead to a significant disruption of the CNS [7]. Thus, in patients with decompensated cor pulmonale and severe respiratory failure, severe encephalopathy occurs [5]. The severity of cognitive impairment, the nature and severity of the neurological deficit determine the patient's ability to continue working, the preservation of the ability to self-service. The study of structural changes in the arteries supplying the brain, the identification of the relationship and the degree of their severity in COPD at different stages of the formation of chronic pulmonary heart is of great interest to clinical medicine [11]. This will help timely prescribe corrective therapy to these patients, improve the quality of life and prognosis of the disease [6]. During PCT, after an intravenous injection of a contrast agent, it spreads through the venous and then through the arterial network, resulting in an increase in X-ray density on CT sections. The increase in CT density after contrast injection can be divided into two phases based on its distribution: intravascular and extravascular. At the initial stage after injection of a contrast agent, an increase in density is associated with the presence of contrast within the vascular bed. During the second stage, when the contrast passes through the basement membranes of the capillaries, there is an increase in density from both vessels and extravascular tissues. Thus, at the first stage, the increase in densitometric parameters is determined by the level of systemic and regional blood flow, and at the second stage, the increase depends on the blood volume and capillary permeability. By obtaining a series of fast sequence of images in the selected area, it is possible to measure the time of "washing out" of the contrast from the tissue after its intravenous injection. Quantitative indicators of perfusion are calculated using mathematical modeling methods that use densitometric indicators of native tissue and the vascular system.

A new stage in the development of techniques based on the principle of the first passage of a contrast agent became possible after the introduction of X-ray perfusion computed tomography (PCT) into clinical practice [26]. L. Axel (1980) studied the theory of indicator dilution based on the principle of central volume and developed a technique for assessing tissue perfusion of the brain using dynamic PCT [27]. The latter is a series of images obtained during the passage of a bolus of a contrast agent through the brain tissue [18].

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