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Chronic Obstructive Pulmonary Disease and Anemic Syndrome

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ABSTRACT

This article examines the problem of anemia formation in patients with chronic obstructive pulmonary disease (COPD) and analyzes the problems and shortcomings that arise in this process. The problem of anemia formation in patients with chronic obstructive pulmonary disease (COPD) is considered. COPD has traditionally been associated with polycythemia, but systemic components can interfere with erythropoietin and lead to anemia of chronic disease.

Introduction.

The modern concept of chronic obstructive pulmonary disease (COPD) interprets it as a disease with systemic manifestations, in which lung damage is considered as one of the components of the disease [1]. New provisions have appeared in the definition of COPD: 1) the disease can be prevented and treated; 2) the disease has systemic manifestations. In the development of extrapulmonary effects in COPD, great importance is given to the role of systemic inflammation [3]. Based on numerous facts, a hypothesis has been put forward that COPD can be considered as a systemic disease with an autoimmune component. There is no doubt the greatest severity of the inflammatory process, hypoxia, and associated extrapulmonary manifestations of COPD during exacerbation of the disease.

Literary review and methodology.

Systemic effects in COPD affect the body as a whole. Since chronic obstructive pulmonary disease usually develops in middle-aged long-term smokers, such patients often suffer from other diseases associated either with smoking or with age. Chronic obstructive pulmonary disease is characterized by an increased risk of myocardial infarction, angina pectoris, osteoporosis, bone fractures, respiratory tract infections, depression, diabetes mellitus, sleep disorders, anemia and

glaucoma, and lung cancer. Systemic manifestations are aggravated with the progression of the disease, SaO2 decreases and the bode index increases, which assesses the severity of dyspnea on the MRS scale, body mass index, and a 6-minute walk test [18]. Nutritional status changes: energy expenditure at rest increases, amino acid metabolism is disturbed, and the body composition of a COPD patient becomes abnormal. Dysfunction of skeletal muscles occurs: their hypotrophy and atrophy develop, functional capabilities are impaired. The causes of systemic reactions in patients with COPD are the inflammatory activity of lung tissue cells, tissue hypoxia, the effects of shortness of breath on metabolism, as well as smoking, industrial pollutants, and genetic factors [8]. There are four main components of the pathophysiology of COPD: airway inflammation (deposition of neutrophils in them), mucociliary transport disorders (MCT), airway obstruction, structural changes in them (remodeling) with damage to the lung parenchyma, and systemic effects - endocrine dysfunction and skeletal muscle dysfunction (with their atrophy), decreased physical activity and body weight, anemia, osteoporosis.

The mechanisms underlying these systemic manifestations are quite diverse and have not yet been studied enough. Despite the large number of publications, many unresolved issues remain regarding the deciphering of the pathogenetic foundations of the disease, the tactics of complex therapy of its systemic manifestations. In practice, doctors mainly encounter COPD patients who are already aggravated by systemic inflammatory processes, i.e. most of them are disabled due to a sharp decrease in exercise tolerance and shortness of breath. Due to the insufficient effectiveness of existing methods of treating patients with COPD, new directions for the diagnosis and treatment of this serious disease and its systemic manifestations are being sought. Recently, much attention of doctors and researchers has attracted "Secondary" anemia accompanying various diseases: tumors, infections, systemic vasculitis (systemic lupus erythematosus, rheumatoid arthritis). In a wide range of diseases, comorbid anemia is increasingly recognized as a risk factor associated with increased mortality. Anemias of patients with chronic infectious inflammatory processes do not have clinical or instrumental laboratory symptoms that could characterize them as an independent type. Such anemias are possible in various diseases, and therefore they are called secondary, symptomatic, emphasizing the role of the underlying disease in their pathogenesis. Recently, the term “anemia of chronic diseases” has been commonly used; according to the morphology of erythrocytes, they, as a rule, are normocytic in nature (ACD) [2,5].

Secondary anemia occurs in various diseases of the internal organs. The list of diseases that must be taken into account in the differential diagnosis of normocytic anemia is quite large. Anemia accompanying infectious, rheumatic and neoplastic diseases are normochromic, less often moderately hypochromic. The number of reticulocytes is normal or reduced. The bone marrow is characterized by a normal or reduced number of erythrocytes, a normal or increased content of sideroblasts. Changes in iron metabolism are characterized by redistributive iron deficiency (decrease in serum iron, transferrin, and increase in serum ferritin). In some cases, in the presence of certain diseases, microblood loss is observed; treatment with cytostatics and radioactive methods is also often accompanied by anemia, which has the character of pancytopenia. Often, with anemia of chronic diseases, the processes of iron absorption are disrupted; so, for example, the absorption of iron decreases with fever; the transfer of iron from reticuloendothelial cells to bone marrow erythroblasts may be blocked or iron consumption may be increased when LPO is activated by non-erythroid cells and bacteria [14]. Various pathogenetic mechanisms for the development of ACD were discussed: redistribution of iron, a decrease in the level of erythropoietin, "toxic" effects, immune and non-immune hemolytic components, etc. However, it has become apparent that the pathogenesis of these anemias is much more complex. Therefore, sometimes this group of anemias is called anemia with mixed pathogenesis: there is a combination of two or more factors [5]. These factors can be grouped as

follows: increased consumption of iron by non-erythroid cells, including bacteria, activation of inhibitors of erythropoiesis, intravascular hemolysis due to DIC, blood loss. There are two theories for the occurrence of anemia of chronic disease. One theory is that anemia develops due to the inability to adequately respond to erythropoietin, an alternative theory is that inflammation causes a change in iron recirculation - a "reticuloendothelial block" [10]. Interleukin6 is regarded as a central mediator of anemia of chronic disease (in the range of inflammatory diseases), it provokes "reticuloendothelial block" of iron blocks the transport of iron from the duodenum.

Discussion and results.

In the pathogenesis of ACD, a certain role belongs to the activation of inhibitors of erythropoiesis. Erythropoiesis inhibitors are a large number of various substances, the common property of which is the inhibition of the maturation of erythrocytes. Inhibitors of erythropoiesis are isolated that act at the level of intercellular interaction, for example, tumor necrosis factor, cytokines, and middle molecular toxins that act at the macro level. A similar mechanism for the development of anemia is well known in patients with chronic renal failure, in sedentary people [15]. Diagnosis of ACD is carried out in two stages: – detection of anemic syndrome; - determination of the main pathogenetic mechanisms of its development in each patient. The solution of this problem is a rather complicated process, even against the background of chronic respiratory diseases, requiring not only standard approaches, but also the use of deductive methods. In each specific case of the disease, it is necessary to clarify the pathogenesis of anemia and differential diagnosis with true anemia [13].

COPD has traditionally been considered one of the most important causes of polycythemia, but recent studies have shown that anemia is also common in patients with COPD. Chronic obstructive pulmonary disease may be accompanied by anemia, which causes a complex of pathogenetic and clinical syndromes that worsen the prognosis and course of the underlying disease. The main cause of anemia in COPD patients is systemic inflammation. Serum cytokines and chemokines can disrupt the main stages of hematopoiesis; possible mechanisms of anemia in COPD are shortened erythrocyte lifespan, impaired iron mobilization and utilization, and impaired bone marrow response to erythropoietin. The important role of systemic inflammation in the development of anemia in COPD is evidenced by the data from the study of CRP and IL6 levels; in COPD patients, these parameters were significantly higher than in patients in the control group; in addition, the level of CRP was significantly increased in COPD patients with anemia compared with COPD patients without anemia. An interesting finding was the fact that the serum level of erythropoietin in patients with anemia was 3 times higher than that in patients without anemia. An inverse correlation was found between the levels of hemoglobin and erythropoietin, which indicates the presence of resistance to erythropoietin, i.e. a characteristic feature of anemia of chronic disease. In addition to inflammation, concomitant diseases (erosions and gastric ulcers), smoking, taking certain medications (theophylline can reduce erythrocyte proliferation) can contribute to the development of anemia in COPD patients. The phenomenon of anemia in COPD is little studied. There are separate foreign studies that analyze the laboratory parameters of anemic syndrome in patients with COPD. A retrospective study of the prevalence of anemia in patients with COPD was carried out in comparison with patients with chronic renal failure, chronic heart failure, bronchial asthma and oncological diseases. The prevalence of anemia in patients with COPD was 23.1%, which was comparable to the prevalence of anemia found in patients with CHF (23.3%). The cohorts of patients with COPD (n - 2524) who received long-term oxygen therapy at home were studied, the proportion of patients with anemia was 12.6% among men and 8.2% among women, and the proportion of patients with polycythemia was only 8.4%. In a study that included 101 patients with COPD, anemia was detected in 13% of patients. Stanbrook et al. investigated the prevalence of anemia in patients with chronic obstructive pulmonary disease. Anemia was defined as hemoglobin values <14 g/dl for men and

<12 g/dl for women. The prevalence of anemia in COPD in this study was 36%. According to Park M. et al. in 58 patients with chronic obstructive pulmonary disease, anemia was diagnosed in 48%. Evidence of a negative association between quality of life and hemoglobin levels in COPD patients is presented. At the same time, the prevalence of anemia was different from other studies: in 2704 patients with COPD, it was 7.3%.

Therefore, the parameter for the prevalence of anemia in COPD is controversial and requires further clarification. In a study by Attaran D. et al. anemia in COPD was diagnosed in 16% of patients (out of 80), it was proved that the reduced survival of erythrocytes is the result of an increase in the titer of cytokines IL1, IL6, and TNFα. Pro-inflammatory cytokines reduce the synthesis of erythropoietin, interfere with the absorption of iron, and weaken the bone marrow response to the action of erythropoietin. Anemia may have a negative impact on the survival of patients with COPD. The ANTADIR study found an inverse association between hematocrit (Ht) levels and survival in COPD patients. In patients with Ht<35%, the 3-year survival rate was only 24%, while in patients with Ht 55% it was 70%. In the group of smokers with COPD, a significant correlation was found between the degree of dyspnea, an increase in the number of platelets, thrombocrit, and platelet aggregation, as well as between a decrease in spirometry and a shortening of the shift time of the aggregation curve with ADP.

Conclusions.

Despite numerous studies on the pharmacotherapy of COPD, a number of unresolved issues remain regarding the formation of protocols for the treatment of patients with concomitant diseases and systemic manifestations, including anemic syndrome. Drug therapy in the combined course of anemia and COPD is one of the urgent tasks of internal medicine. Currently, there is no information about the nature of the course of the disease against the background of anemia, the principles and tactics of treating patients with anemic syndrome have not been developed, which does not allow realizing all the possibilities for managing this severe chronic disease.

REFERENCES:

1. Avdeev, S.N. Chronic obstructive pulmonary disease as a systemic disease / SN Avdeev // Pulmonology. - 2017.-№2 - P.11-16.
2. Anemia is one of the serious complications of chronic renal failure (CRF), affecting the quality of life and overall survival of patients / Yu. S. Milovanov [et al.] // The attending physician. - 2015. - No. 7. - P.45-50.
3. Arkhipov, V.V. Chronic obstructive pulmonary disease / VV Arkhipov // Pulmonology. - 2010. - No. 4. - P.99-104.
4. Atlas of hematology / S. Anderson [et al.]. - M.: - M. Logosphere, 2017. - 421 p.
5. Hemorheological disorders in patients with anemia of chronic diseases / L A. Lukina [et al.] // Hematology and transfusiology. - 2015. - No. 6.- P.17.
6. Global strategy for the diagnosis, treatment and prevention of chronic obstructive pulmonary disease: Per. from English. / A.G. Chuchalin [and others]. - M.: Atmosfera, 2017. - 96 p.
7. Drozdova, M.F. Blood diseases / M.F. Drozdova. – M.: Medicine, 2010. – 180 p.
8. Nonikov, V.E. Chronic obstructive pulmonary disease (COPD): diagnosis and treatment / V.E. Nonikov // Pulmonology. - 2014. - V.6, No. 5. -p.12-17.
9. Ovcharenko, S.I. New advances in the treatment of chronic obstructive pulmonary disease (results of the UPLIFT study) / S.I. Ovcharenko, V.A. Kapustina // Farmateka. - 2009. - No. 5. - P.33-38.

10. Fundamentals of clinical hematology / VG Radchenko [et al.]. - St. Petersburg: Medicine, 2013. – 235 p.
11. Provotorov, V.M. The role and place of erythrocytes in the directed transport system various pharmacological agents / V. M. Provotorov, G. A. Ivanova // Clinical Medicine. - 2009. - No. 9. – P.54-56
12. Provotorov, V.M. Features of the treatment of patients with CHF with anemic syndrome /– M., 2011. – P.113-114.
13. Guide to internal diseases / F.I. Komarov [i dr.]. – M.: Medicine, 2017. – 375 p.
14. Guide to hematology: in 3 volumes. T. 1 / A.I. Vorobyov [i dr.]. – M.: Nyudiamed, 2012. – 280 p.
15. Semenkova, G.G. Long-term results of treatment of patients with chronic obstructive pulmonary disease / G.G. Semenkova, O.N. Stasyuk // Bulletin of the youth innovation center. Issue. II. - Voronezh, 2009 - p. 135-139.
16. Dadajanovich, D. U., Xaydarovna, M. F., & Narzullaevna, R. O. (2020). Features Of Thrombophilia In Covid-19. *European Journal of Molecular & Clinical Medicine*, *7*(03), 2020.
17. Xaydarovna, R. M. (2020). Problems of teaching communicative English language at the Military Institute of the National Guard of the Republic of Uzbekistan and the ways of eliminating them. *EPRA International Journal of Research and Development*, *5*(3), 502-504.
18. Xaydarovna, M. F., Toirjonovna, M. N., & Narzullaevna, R. O. (2020). Prevention Of Anemia In Patients With Tuberculosis. *The American Journal of Medical Sciences and Pharmaceutical Research*, *2*(11), 62-65.
19. Гадаев, А. Г., Махмонов, Л. С., & Маматқулова, Ф. Х. (2022). Helicobacter pylori билан ассоцияланган темир ва витамин в12 танқислиги камқонликларида яллиғланиш цитокинларининг айрим лаборатор кўрсаткичлар билан ўзаро боғлиқлиги.
20. Jhasur, R. A., Lutfulla, M. S., Gadaev, G. A., & Turakulov, I. R. Helicobacter Pylori билан ассоцияланган темир танқислиги камқонлигига мойиллик туғдирувчи ташқи омилларни баҳолаш. *Samarkand-2022*, *4*, 436-445.