ANEMIA IN CARDIOVASCULAR DISEASES

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Abstract

Anemia, defined according to World Health Organization guidelines organization shealth care decline hemoglobin level <130 g/l in men and 120 g/l in women is an independent risk factor for cardiovascular diseases. The presented review touches on the prevalence of anemia and iron deficiency (ID) in CHF, their impact on the course and prognosis of this condition. A definition of anemia and ID is formulated based on the assessment of various laboratory data. In particular, the diagnostic significance of determining serum iron and serum ferritin is discussed blood, transferrin saturation coefficient, total iron binding capacity of blood serum and the level of soluble transferrin receptors. The importance of determining the level of iron in the bone marrow is emphasized, although it is the “gold standard” for diagnosing ID, but is rarely used in widespread clinical practice. The pathogenetic mechanisms of the development of ID in CHF are covered, including lack of iron intake, the role of inflammation, erythropoietin, RAAS and the influence of certain pharmacological drugs. The article describes the main pathophysiological mechanisms of the relationship between anemia and cardiovascular diseases systems, summarizes the results of relatively recent research in this area and discusses their implications for clinical practice.

Keywords: anemia, cardiovascular disease, currents.

Relevance

According to WHO estimates, 1.63 billion people suffer from anemia in the world [1]. The incidence of anemia, as well as coronary artery disease, increases significantly with age. More than 10% of people over 65 years of age and up to 50% of elderly patients with chronic diseases living in nursing homes have signs of anemia [3].

The increasing incidence of chronic diseases and their associations causes difficulties for the general practitioner in timely diagnosis and selection of adequate comprehensive treatment, in particular for chronic heart failure (CHF), combined with anemia of various etiologies. In the practice of a general practitioner and a cardiologist, a combination of cardiovascular pathology Withanemia. Anemia is determined in 25–40% of patients with chronic heart failure (CHF) and 10–20% of patients with coronary artery disease [2,6].

European and American recommendations for the management of patients with stable angina, issued in the last two years, require mandatory determination of hemoglobin levels in all patients and consider anemia as a factor provoking coronary insufficiency [3,5]. In the latest recommendations for the diagnosis and treatment of patients with CHF, American and

European experts note that anemia not only increases the symptoms of CHF, worsens the quality of life of patients, reduces exercise tolerance, and can cause the development of acute decompensation of CHF and an increase in the frequency of hospitalizations, but also an independent negative predictor of prognosis [2,4]. The risk of death in CHF in patients with anemia is twice as high as without it, even when taking into account additional variables (renal dysfunction, severity of CHF, and others) [5]. Already latent iron deficiency can have a negative impact on the prognosis of CHF [3], which makes it advisable to determine its markers in all patients with CHF.

Purpose of the study

To study the frequency and nature of anemia in patients with systolic CVD, its effect on clinical and hemodynamic parameters.

Etiopathogenesis of anemia in CHF. Despite the fact that anemia is a fairly common condition in patients with CHF, a direct cause-and-effect relationship with either HF or other concomitant diseases has not yet been established. The following have been proposed as independent or interrelated causes of anemia: iron deficiency, inflammation, the role of EPO, pharmacotherapy of CHF, hemodilution and BM dysfunction, among which iron deficiency and inflammation have the most reliable levels of evidence [6,7]. A relatively simple and convenient algorithm for determining the main cause of anemia in severe HF was recently proposed by the Greek research group of E. Kaldara [7].

Results

The frequency of anemia diagnosed in accordance with WHO criteria in patients with systolic CHF II-IV class according to NYHA is 28.8%. Iron deficiency anemia was detected in the majority of patients (46.1%), the second most common is anemia of chronic diseases (36.5%), and anemia with vitamin B12 deficiency (7.9%) and folic acid deficiency (9.5%) are less common.). The incidence of anemia increases with increasing functional class of CHF (33% in patients with NYHA III and IV compared with 15% in NYHA II). The severity of anemia is directly proportional to the duration of CHF and the level of NT-proBNP, but does not depend on the Left Ventricle. The presence of anemia is associated with significantly higher LVH Andsystolic-diastolic dysfunction of the LV myocardium. The presence of anemia was directly correlated with elevated hf CRP levels, the presence of proteinuria, and lower GFR and cholesterol values.

Cardiorenal anemia syndrome was identified in 20.7% of patients with systolic CHF, which were characterized by a combination of lower hemoglobin and GFR values with higher levels of NT-proBNP as well as inflammatory markers (HF CRP, IL-1 and TNF-a) compared with patients without renal pathology.

Correction of absolute iron deficiency in patients with cardiorenal anemia syndrome using intravenous administration of iron preparations (sucrat and iron carboxymaltose) leads to a significant decrease in the FC of CHF, improved quality of life and increased exercise tolerance according to the results of the six-minute walk test. In all patients, the levels of Hb, Ht and red blood cells normalized, and hf CRP values decreased ($p < 0.001$). The use of intravenous iron supplements in this group of patients is safe and well tolerated.

The introduction of statins, and vabradine and inotropic drugs (dopamine, levosimendan, ularitide) into the basic therapy of systolic CHF does not affect hemoglobin levels and does not worsen the functional state of the kidneys.

Conclusions. The main hemodynamic compensation factor is an increase in cardiac output. It is due to a decreasefastload, increasingpreloadand positive inotropic and chronotropic effects. Decreasepost loadis a consequence of decreased blood viscosity (low hematocrit) and vascular resistance. Simultaneously with a decrease in post-load and an increase in cardiac output during anemia, venous return (pre-load), filling of the heart chambers, and their end-diastolic volume increase, which naturally leads to an overload of the heart with volume and work. Work overload also occurs due to an increase in myocardial contractility and heart rate under the influence of an increase in the tone of the sympathetic nervous system and the concentration of catecholamines. In the long term, these hemodynamic changes lead to the gradual development of myocardial hypertrophy, increasing dilatation of the heart chambers, primarily the left ventricle (LV), and the formation of relative insufficiency of the valve apparatus. In addition, the presence of anemia leads to persistent myocardial ischemia. Thus, congestive heart failure causes anemia, anemia worsens the course of CHF, and CHF itself aggravates anemia. IDA meets in 46.1% of cases, CHF and is easily diagnosed by the level of hemoglobin and such indicators of iron metabolism as serum iron, ferritin, transferrin and transferrin saturation with iron.

Treatment of CHF includes general measures, non-drug treatment, pharmacotherapy and surgical methods. Iron supplements for anemia and iron deficiency lead to an improvement in clinical and laboratory parameters in CHF, including in patients with impaired renal function. The administration of intravenous iron supplements in this category of patients is safe and well tolerated.

Renal dysfunction, activation of neurohumoral mechanisms and the influence of anti-inflammatory cytokines can lead to the development of anemia of chronic diseases with impaired iron utilization, decreased erythropoietin production and suppression of bone marrow function [8]. Impaired proliferation, differentiation, mobilization and iron incorporation into hematopoietic stem cells negatively affects bone marrow function [9]. A decrease in renal perfusion in patients with CHF leads to ischemia of the renal tissue and increased production of erythropoietin. Against the background of kidney dysfunction, the reninangiotensin-aldosterone system is activated, the production of angiotensin II is stimulated, which in turn

promotes increased production of erythropoietin and proliferation of erythroid progenitor cells in the bone marrow [10]. However, the bone marrow, as a result of exposure to anti-inflammatory cytokines, ceases to be sensitive to erythropoietin. As a result of the action of anti-inflammatory cytokines, the amount of iron available for erythropoiesis decreases, the amount of ferroportin (a transmembrane iron transporter) decreases, and the amount of hepcidin increases. Hepcidin reduces the reabsorption of iron in the duodenum, prevents the release of iron from macrophages and reduces the ability of red bone marrow to absorb it. Concentration of anti-inflammatory cytokines, incl. tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), increases in HF and inversely correlates with hemoglobin levels [10]. In patients with CHF, hemodilution contributes to the development of anemia. In patients with anemia, blood viscosity is reduced and total peripheral vascular resistance is reduced as a result of nitric oxide-mediated vasodilation. A decrease in blood pressure causes neurohumoral activation with an increase in sympathetic and renin-angiotensin-aldosterone activity, leading to insufficient renal perfusion and expansion of the extracellular space. Considering the above, it can be assumed that anemia worsens the prognosis in patients with CHF as a result of a decrease in oxygen supply to the myocardium. ventricular remodeling, neurohumoral changes, actions of pro-inflammatory cytokines and, in some cases, the development of renal failure. It is likely that anemia is both a mediator and a marker of poor outcome in CHF.

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