ANEMIA OF CHRONIC DISEASE IN THE ELDERLY (LITERATURE REVIEW)

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Anemia is a common disease among the elderly. Its prevalence in this population ranges from 8 to 44% and continues to grow in the elderly. It used to be thought that anemia was an inevitable consequence of aging, but in reality, many healthy older people have normal hemoglobin levels. The root cause of anemia can be found in most older people.

Anemia of chronic disease (ACD) develops in many chronic debilitating diseases. Their clinical picture is masked by a variety of symptoms of the underlying disease, the prevalence of cardiovascular and cerebral manifestations. There is a high frequency of lesions of the upper gastrointestinal tract in the form of superficial or deep gastritis with impaired acid-forming function. The criterion for diagnosis is a decrease in serum iron levels while maintaining its reserve in the form of ferritin [1, 10].

Among them, the most common infections, tumors, autoimmune diseases, chronic kidney disease.

The pathogenesis is based on a combination of several factors.

Firstly, the lifespan of erythrocytes is slightly reduced, secondly, there is iron sequestration in the cells of the reticuloendothelial system, which cannot be effectively utilized by the body, and finally, an insufficiently high level of erythropoietin relative to the degree of anemia.

The main distinguishing feature of ACD from iron deficiency anemia is normal iron stores in the body, which can be confirmed by bone marrow biopsy.

It is believed that all these changes occur under the influence of cytokines such as interleukin-1, tumor necrotic factor, interferons alpha, beta and gamma. In recent years, another mediator involved in iron metabolism, called hepcidin, has been discovered. In the presence of an inflammatory process, the concentration of hepcidin can increase by 100 times. Hepcidin is secreted in response to increased iron stores in the liver. At the same time, it blocks the absorption of iron from the gastrointestinal tract and stimulates the uptake of iron molecules by macrophages, which are part of the reticuloendothelial system.

It should be noted that anemia of chronic disease is usually not severe. Its degree correlates with the severity of the chronic disease that caused the disease.

Pathogenesis anemia of chronic disease is extremely complex and insufficiently studied. It is believed that the leading role in the development of such anemia is played

by a change in iron metabolism, a shortening of the life of erythrocytes, or their inadequate production by the bone marrow (BM). This may be due to the influence of various pro-inflammatory cytokines (interferon γ, interleukins (IL), tumor necrosis factor- (TNFα)), the level and activity of which increases significantly in many chronic diseases of internal organs, as well as cells of the reticuloendothelial system (histiocytes, monocytes, macrophages) [9].

In recent years, it has been established [2] that hepcidin, an amino acid peptide synthesized in the liver, plays the role of a universal humoral regulator of iron metabolism. It was noted [2] that under the action of pro-inflammatory cytokines, in particular IL-6, hepcidin hyperproduction occurs, which blocks the receptors of ferritin, a transmembrane protein that transports iron adsorbed by enterocytes. Thus, the export of iron from cells containing this protein (macrophages, enterocytes, etc.) to the blood is disrupted. This assumption was confirmed in an in vitro experiment [23]. Another type of anemia common in the elderly is hyperchromic, B12- and folate-deficient, which, as a rule, are diagnosed at the age of over 50-60 years, manifested by lesions of the hematopoietic, gastrointestinal and nervous systems. systems. These anemias develop as a result of insufficient intake of vitamin B12 or folic acid, which is associated with a violation of the content of gastromucoprotein (intrinsic factor produced in the fundus of the stomach), which combines with food vitamin B12 and ensures its absorption. Clinically, anemia is manifested by powdery pallor of the skin with an icteric tint, glossitis with smoothing of the papillae, and burning on the tip of the tongue. Gastric secretion is depressed, a slight increase in the liver and (or) spleen is possible. The patient's gait can be changed: clapping, unsure, tendon reflexes of the extremities are disturbed (clinic of funicular myelosis). produced in the fundus of the stomach), which combines with dietary vitamin B12 and ensures its absorption. Clinically, anemia is manifested by powdery pallor of the skin with an icteric tint, glossitis with smoothing of the papillae, and burning on the tip of the tongue. Gastric secretion is depressed, a slight increase in the liver and (or) spleen is possible. The patient's gait can be changed: clapping, unsure, tendon reflexes of the extremities are disturbed (clinic of funicular myelosis). produced in the fundus of the stomach), which combines with dietary vitamin B12 and ensures its absorption. Clinically, anemia is manifested by powdery pallor of the skin with an icteric tint, glossitis with smoothing of the papillae, and burning on the tip of the tongue. Gastric secretion is depressed, a slight increase in the liver and (or) spleen is possible. The patient's gait can be changed: clapping, unsure, tendon reflexes of the extremities are disturbed (clinic of funicular myelosis).

In addition to the typical Addison-Birmer B12 deficiency anemia, agastral B12 deficiency anemia can be observed in old age, which develops on average 7–8 years after gastrectomy, as vitamin B12 reserves are used up. As a causative factor, functional agastria is not excluded in case of damage to the fundus of the stomach, where gastromucoprotein is produced, due to severe recurrent gastric ulcer or cancer,

or chronic alcohol poisoning with many years of alcohol abuse. These processes contribute to the development of B12-deficiency anemia, the clinical and morphological symptoms of which are somewhat blurred compared to the classical form of the disease, but the need for emergency treatment remains.

Folate deficiency anemia is characterized by similar clinical and morphological signs, although less pronounced, as well as the absence of neurological lesions - funicular myelosis. The cause of folic acid deficiency is usually an inflammatory and (or) infectious lesion of the mucous membrane of the small intestine, including with sprue, during its removal, as well as with prolonged alcohol abuse. The diagnosis is confirmed by a decrease in the level of folates in the blood serum: up to 2.1 ± 2.08 ng / ml (norm 5.8-10.0 ng / m) and in erythrocytes - up to 1.6 ± 0.44 ng (norm 5, 0-35 ng). At the same time, the content of vitamin B12 in blood serum can moderately decrease (260±45 pg/l), while in erythrocytes it does not change [10].

The prevalence of B12- and folate deficiency anemia in the elderly and senile age is 2%, but often it remains unrecognized. American doctors on more than a thousandth contingent of patients showed that the frequency of undiagnosed B12-deficiency anemia reaches 1.9% (it was not detected among immigrants from Latin America and Asia); when extrapolating these data to the entire US population, it turns out that this disease remains unrecognized in 800 thousand Americans of advanced age [1,11,12]. This circumstance testifies to the unfavorable diagnosis of anemia in this age group and to the underestimation of their impact on the health of the elderly, resulting in their late detection and lack of treatment.

The development of anemia in the elderly is accompanied by a significant deterioration in the quality of life (decrease in mental and physical activity, fatigue, depressed mood), aggravates the course of the existing pathology and creates a threat of premature death. So, when observing 63 patients aged 70 to 90 years, the 5-year survival rate in the group of patients without anemia was 67%, and in the group with low Hb levels - 48%.

When observing 1002 disabled women older than 65 years in the group with Hb < 120 g/l, the risk of death was 1.5 times higher than with Hb 130 g/l. In the analysis of data concerning 2280 patients with symptoms of heart failure, anemia was noted in 48% of them; the ratio of mortality in this group and in patients without anemia was 4:1, and a decrease in hematocrit by 1% was accompanied by an increase in the risk of death by 2% [11,12].

The development of anemia in old age is associated with a violation of cognitive functions - a decrease in intelligence, memory, concentration. Greek scientists [12] report a decrease in cognitive function in 55.6% of older men with anemia and in 34.4% without it (p = 0.016), in older women the corresponding figure was 47.5 and 40.1% (p=0.016).

It has been established that B12-deficiency anemia in the acute stage is one of the determining risk factors for the development of fractures due to osteoporosis.

These data indicate the need for timely and accurate diagnosis of anemia with the establishment of its form and origin and adequate therapy in geriatric patients.

With iron deficiency in the body in the elderly and senile age, the erythron system, bone marrow hematopoiesis, as a rule, suffers last of all, due to the compensatory mobilization of reserve and tissue iron for these needs, which creates a vicious circle when the maintenance of hemoglobin formation in the bone marrow negatively affects the tissue respiration, causing pathological disturbances in the system of redox processes in tissues and, ultimately, in the bone marrow itself. In addition to this, it should be noted that a decrease in the functional activity of cytochromes of enterocytes of the duodenal mucosa negatively affects the absorption of iron [9,12,]. A significant decrease in tissue respiration is noted due to a decrease in functional activity, in particular, cytochrome oxidase in the tissues of the liver, myocardium, kidneys in elderly and senile patients. They also indicate that with aging, the content of myoglobin, a heme-containing protein in muscles, decreases. Thus, it should be noted that the above disorders of tissue respiration with age have a complex genesis, are multifactorial in nature, and disturbances in the activity of iron-containing and iron-dependent enzymes of the mitochondrial respiratory chain due to iron deficiency in the body in old age is one of the leading factors.

The impact of developing iron deficiency on metabolic processes during aging is not limited to dysfunctions of erythropoiesis and tissue respiration. Of interest is information about the role of iron in the functioning of immunity in the body, in particular, it indicates a decrease in immunological reactivity due to inhibition of RNA reductase and impaired DNA synthesis in lymphocytes due to iron deficiency, and disorders of the immune system have recently been considered as one of the leading factors in the mechanism of aging.

Conclusion: It has been established that in most cases anemia is a manifestation of some other disease. Therefore, even if anemia is minor and the patient does not experience symptoms, investigation of its causes is absolutely necessary in order to identify the underlying disease that causes it. Another reason is the fact that 80 percent of older people with anemia can find the cause. For persons of elderly and senile age, a characteristic feature is polymorbidity, therefore, in an isolated form, anemia is rare. Background and concomitant diseases obscure the signs of anemic syndrome, which is often the cause of delayed diagnosis and treatment of these patients. Moreover, anemic syndrome aggravates the clinical course of diseases of the cardiovascular and nervous systems. Due to this, clinicians face significant diagnostic, therapeutic and organizational difficulties. A better understanding of the mechanisms that regulate the progression of anemia is essential for developing definitive treatment strategies for elderly patients.

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