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Anemia in Chronic Diseases

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Summary: Anemia that occurs during infectious and inflammatory processes, non-infectious inflammatory diseases and tumors is called "anemia of chronic diseases" (ACD). With all the diversity of pathogenetic mechanisms for the development of anemia in these situations, one of the main ones is the redistribution of iron into the cells of the macrophage system, which is activated during various inflammatory (infectious and non-infectious) or tumor processes. In terms of the prevalence of ACD, they occupy 2nd place after iron deficiency anemia (IDA). The identification of ACD as a separate pathogenetic variant and awareness of it are important because of the similarity of this variant with IDA and some sideroachrestic anemias, although the essence and therapeutic approaches for these anemias are different.

Key words: anemia of chronic diseases; Iron-deficiency anemia; prevalence.

Relevance. Anemia is detected in 4% of men and 8% of women, and among middle-aged and elderly people - in 8-44%. One common type of anemia is anemia of chronic disease (ACD), or anemia of chronic inflammation, or iron-distributing anemia, which occurs in patients with chronic activation of cellular immunity and lasts more than 1-2 months. ACD ranks second among anemias in terms of prevalence after iron deficiency anemia (IDA) [3].

Morbidity in elderly and senile age is characterized by polymorbidity, i.e. accumulation of diseases, among which anemia occupies a significant place.

The main pathogenetic mechanism in ACD is considered to be the redistribution of iron in the cells of the macrophage system, which is activated during various inflammatory or tumor processes. Anemia is very diverse in etiology, pathogenesis and clinical and hematological characteristics. In the clinical and pathogenetic classification of anemia, a section is dedicated to iron metabolism disorders (iron deficiency, iron distribution, sideroachrestic anemia).

In the morphological classification of anemia, ACD is classified as normocytic anemia, and according to the degree of regeneration, it is classified as regenerative. Clinical manifestations in a patient with anemia are determined by the pathology causing the anemia and the severity of tissue hypoxia. ACD is one of the symptoms of a common disease, often masking or concealing the underlying disease. There is a direct relationship between the degree of ACD and the severity of the underlying disease. Anemia enhances clinical manifestations in case of damage to the arteries supplying the brain and lower extremities, aggravates heart failure, and in lung diseases anemia aggravates hypoxic syndrome. The body adapts to low levels of hemoglobin (Hb) and red blood cells, and patients often get used to their illness, explaining this by overwork at work, psycho-emotional overload and other factors. Changes in internal organs appear when the Hb level

decreases to 80-70 g/l, and when the Hb concentration decreases by less than 40 g/l, the likelihood of developing an anemic coma is high [6]. In normocytic anemia, the MCV is 81-100 µl. Regeneration is observed in the bone marrow - the number of reticulocytes is 1.5-5%. The number of leukocytes corresponds to the concomitant pathology. In cases of infection and severe intoxication, toxic granularity of neutrophils is detected. To make a diagnosis, it is necessary to assess the adequacy of iron metabolism: serum iron (LC), serum ferritin (FS), transferrin iron saturation (NTJ), and serum transferrin receptor (TfR) levels. Ferritin levels are the gold standard for assessing the amount of iron stored in the body: it is directly proportional to the accumulation of iron in macrophages and hepatocytes, unless there is an infection or inflammatory process. Its reduction has 100% specificity for identifying iron deficiency conditions. Ferritin concentrations may increase in ACD associated with infection, inflammation, and malignancy. An Hb content in reticulocytes of more than 28 pg indicates sufficient iron reserves for Hb synthesis and erythropoiesis. It should be borne in mind that transferrin (Tf) has the qualities of a "negative" acute phase protein, i.e. acute inflammation helps to reduce its level. Malignant neoplasms, liver disease, nephrotic syndrome and poor nutrition can reduce the concentration of Tf in the blood serum, while pregnancy and oral contraceptives can increase this indicator. Total serum iron binding capacity (OGSS) reflects the degree of serum starvation and correlates with Tf levels. With iron (J) deficiency, there is an increase in heart rate. A decrease in this indicator is observed in diseases accompanied by significant loss or increased protein consumption (nephrotic syndrome, chronic renal failure, severe burns, chronic infections and active inflammatory processes, malignant neoplasms, severe liver diseases). There is no data on the effect of inflammatory reactions on the concentration of GFR [7]. A distinctive feature of AHZ is the combination of J and, accordingly, iron deficiency in the hematopoietic tissue of the bone marrow with intense iron uptake by macrophages and dendritic cells of the reticuloendothelial system (RES). Iron released from decaying red blood cells, which under normal conditions is reused in the synthesis of new hemoglobin molecules, enters the iron-containing depot. As a result, the FS content increases. AHD is diagnosed in the presence of hypoferremia and elevated or normal PS levels. This occurs as a result of stimulation of iron accumulation in the RES and immune activation of ferritin synthesis. With IDA and AHZ, a decrease in LC concentration and saturation of Tf with iron are observed. A decrease in the saturation of Tf with iron in ACD reflects a decrease in the concentration of iron in the blood serum, while in IDA there is an increase in the content of Tf (in the first case, this indicator is within the normal range or increased), which makes the decrease in the saturation of Tf with iron more pronounced. When ACD is combined with concomitant IDA, microcytosis and a more severe degree of pathological changes are more often observed. To identify functional disorders during erythropoietin (EPO) therapy in patients with ACD, it is proposed to determine the percentage of erythrocyte hypochromia and Hb levels in reticulocytes [8].

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Anemia in chronic inflammatory (infectious) diseases. Anemia most often develops with purulent diseases of the lungs, kidneys and other organs a month after the onset of the disease. Hb decreases to 110-90 g/l. ACD can become the leading symptom of a hidden disease. With such ACD, special corrective therapy is not required; treatment should be aimed at the underlying disease.

Anemia due to HIV infection. The viral load in HIV infection is inversely proportional to all hematological parameters. The disease can cause anemia by affecting cytokine production and inhibiting erythropoiesis, reducing EPO concentrations and increasing the risk of opportunistic

infections. Therapies used to treat HIV also cause a decrease in the number of red blood cells (myelotoxicity). Anemia is associated with decreased life expectancy and poorer quality of life in HIV-positive patients [9].

Anemia in chronic kidney disease (CKD). Diabetes mellitus and arterial hypertension are the main causes of end-stage CKD. For patients newly diagnosed with CKD, the primary goal is to slow disease progression by optimizing treatment of the underlying disease. Anemia in CKD develops due to the inability of the kidneys to secrete sufficient amounts of EPO to stimulate adequate erythropoiesis and is aggravated by obesity, severe hyperparathyroidism, acute or chronic inflammatory diseases, and shortened erythrocyte lifespan. Anemia worsens as CKD progresses. Anemia in systemic connective tissue diseases is caused by impaired synthesis of EPO, J due to blood loss from ulcers and erosions of the gastrointestinal tract, developing with long-term use of anti-inflammatory drugs. Rheumatoid arthritis is accompanied by anemia in 16-65% of cases. In the group of patients with anemia, 77% of patients have hypertension and 23% have IDA.

The development of anemia in rheumatoid arthritis is promoted by increased levels of inflammatory cytokines. Approximately half of patients with systemic lupus erythematosus have anemia with an Hb content of less than 100 g/l, which is either hypochromic or normochromic. In all cases of anemia due to connective tissue diseases, treatment of the underlying disease is necessary.

Diagnostic criteria for anemia in chronic hepatitis and liver cirrhosis. Clinical signs (anemia secondary to impaired liver function, the most common cause of non-megaloblastic macrocytic anemia, observed in approximately half of cases of liver disease), pathology varied (hemolysis, decreased bone marrow response, folic acid deficiency; blood loss, lipid disorders of the red blood cell membrane) .

Endocrine pathology occurs quite often. In this case, the development of all morphological variants of anemia is possible: normal, hypo-, macrocytic. Thus, with primary hyperparathyroidism, ACD is observed in every second patient. Hypothyroidism is accompanied by anemia in 30-60% of patients. Anemia with hypopituitarism occurs in 32-46% of cases. The number of people with diabetes continues to rise worldwide. As diabetes progresses, the glomerular basement membrane thickens as a result of glycosylation, which leads to increased intrarenal pressure. This damage leads to decreased EPO production and the development of anemia [12]. Diagnostic criteria for anemia in endocrine diseases. Clinical signs (symptoms are specific: hyperthyroidism, hypothyroidism, hyperfunction of the adrenal cortex, hypofunction of the adrenal cortex, hypoandrogenemia, diabetes mellitus), pathology (hyperthyroidism - an increase in the total mass of erythrocytes due to accelerated proliferation; hypothyroidism - a decrease in the total mass of erythrocytes due to a decrease in oxygen demand; hyperfunction of the cortex adrenal glands - moderate polycythaemic state due to increased androgen levels; hypofunction of the adrenal cortex - a state of hemoconcentration with a normal or slightly increased hematocrit level due to a lack of mineralocorticoids; hypogonadism - anemia due to decreased androgen levels; diabetes mellitus - falsely elevated hematocrit level, acute hemolysis can develop with ketoacidosis) [13].

Anemia in malignant neoplasms (ZN). The prevalence of anemia in patients with ZN ranges from 5 to 90%. Mild anemia after chemotherapy is observed in 100% of patients, and the incidence of moderate and severe anemia can reach 80%. The occurrence of anemia contributes to the progression of the underlying disease. Anemia in ZN is the result of cytokine-mediated regulation of erythropoiesis: impaired iron utilization, suppression of differentiation of erythroid

progenitor cells and insufficient production of EPO, as well as a decrease in the lifespan of erythrocytes. Approximately 75% of patients with ZN report symptoms of fatigue (weakness, apathy), trouble starting and completing tasks, and needing sleep during the day (61% of patients reported that fatigue had a more adverse impact on their lives than pain associated with with ZN). Other side effects associated with anemia include increased heart rate, cognitive impairment, nausea, decreased skin temperature, impaired immune system function, dizziness, headache, chest pain, shortness of breath, depression, and decreased performance. The presence of anemia in patients with ZN increases the risk of death. Anemia has an adverse effect on quality of life and prognosis in patients with heart disease. Treatment of anemia reduces the need for blood transfusions by 7-47%. Only 50% of cancer patients have a complete therapeutic response to conventional doses of EPO. Intravenous iron administration significantly improves response to EPO therapy [14].

Examination algorithm for the presence of normocytic anemia. If a patient with anemia has red blood cells of normal appearance and size, the first step is to count the reticulocyte count. With an increased level of reticulocytes, differential diagnosis between posthemorrhagic and hemolytic anemia is necessary. If the reticulocyte level is normal or reduced, GI is determined. When its content decreases, it is necessary to differentiate early J and AHZ. If cholesterol levels are normal or elevated, the first step is to conduct an examination to exclude diseases of the kidneys, liver or endocrine system. If cholesterol levels are normal and there are no chronic diseases, it is necessary to examine the bone marrow. This will identify possible infiltration of the bone marrow by leukemic cells, tumor metastases, proliferation of fibrous tissue, identify myelodysplastic syndrome, masked megaloblastic anemia, and also establish aplastic anemia or a rare type of hereditary dyserythropoietic anemia, in which multinucleated erythroblasts are present. In case of normocytic anemia, first of all, chronic renal failure, nutritional defects, and hemolytic anemia should be excluded [16]. Anemia is not a diagnosis, but a syndrome. After laboratory testing and identification of ACD, it is necessary to conduct a comprehensive examination of the patient to diagnose the underlying disease. For infectious and inflammatory diseases of the lungs (bronchiectasis, abscesses, pleural empyema), the main methods are X-ray and tomographic examination of the lungs, bronchological examination (bronchography, bronchoscopy). Diseases of the abdominal organs (cholangitis, liver abscesses, subphrenic abscess, interdigital abscess, peritonitis, inflammatory processes in the pelvis) are detected by ultrasound, abdominal laparoscopy, and gynecological examination.

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