

**STUDYING THE DIAGNOSIS OF PERIPHERAL BLOOD AND
BONE MARROW IN ANEMIA OF CHRONIC DISEASES.**

Madrakhimova Nigora Shokir qizi

*Master of the Department of Military Field Therapy,
Hematology and Diagnostics" of the Urgench
branch of the Tashkent Medical Academy
madraximovanigora19@gmail.com*

Fazilova Sharifa Mirhamidovna

*Associate professor, of the Department
of Military Field Therapy, Hematology
and Diagnostics" of the Urgench branch
of the Tashkent of the Medical
Academy Associate, PhD*

Summary: Thus, the detection of anemia in patients determines the need for mandatory clarification of the causes of this pathological condition. Timely verification of the etiology of anemia makes it possible to start adequate therapy without delay, which determines its effectiveness and improves the quality of life of people.

Key words: chronic disease anemia, metabolism, transferrin

At present, researchers are paying special attention to a form of anemia - chronic disease anemia (ACD). A characteristic feature of this anemia is impaired iron metabolism - a combination of iron deficiency available for erythropoiesis (reduced level of transferrin saturation with iron-TBAT) with sufficient iron supply in the reticuloendothelial system [6, 8].

ACD is a secondary anemia that occurs against the background of various infectious, inflammatory, autoimmune or tumor diseases lasting more than 1-2 months. This type of anemia is also called "chronic inflammation anemia" or "cytokine-induced anemia." ACD ranks 2nd in prevalence after iron deficiency anemia (IDA). For the first time, concomitant anemia in a chronic disease was mentioned in 1842, when French researchers discovered a decrease in the mass of red blood cells in patients infected with smallpox. Further observations of patients with typhoid fever, tuberculosis, and syphilis allowed M. Wintrobe and G. Cartwright to introduce the term "inflammatory anemia" in 1949 and in 1952 [11,15].

In the early 80s of the last century, E. Weinberg presented a theory that iron serves as a necessary component for maintaining the vital functions of all living organisms, including bacteria and tumor cells [5,7]. Based on this theory, we can conclude that anemia of the inflammatory response is a natural defense mechanism

aimed at limiting free iron (Fe^{3+}) in the blood when it enters the body pathogens, which is achieved by the rapid binding of iron to lactoferrin, as well as by its deposition (ferritin). In inflammatory processes, the synthesis of ferritin and lactoferrin increases, resulting in iron deficiency, which helps to reduce the growth of pathogenic microorganisms. Hepcidin, which plays a fundamental role in the pathogenesis of the development of ACD, is an acute-phase protein and blocks the absorption of iron in the intestine and its release from the depot. ACD is often found (8–33% of patients) against the background of chronic infectious, inflammatory, and destructive lung diseases, such as chronic obstructive pulmonary disease (COPD), bronchiectasis, abscess, pleural empyema, and tuberculosis [4,9]. According to a number of studies, the prevalence of anemia among patients with COPD is 7.5–33.0%. ACD often develops with diffuse connective tissue diseases (systemic lupus erythematosus, scleroderma), rheumatoid arthritis, systemic vasculitis, sarcoidosis, autoimmune bowel diseases (ulcerative colitis, Crohn's disease), liver diseases (autoimmune hepatitis, cirrhosis of the liver) and thyroid gland (autoimmune thyroiditis, hypothyroidism). Against the background of rheumatoid arthritis, ACD is detected in 25–64% of cases. The main role in the development of anemia in rheumatoid arthritis is played by changes in iron metabolism, inadequate production of red blood cells by the bone marrow. This may be due to exposure to various pro-inflammatory cytokines such as interferon- γ , interleukins, TNF- α . The main mechanism for the development of ACD is immunoinflammatory. Increased expression of pro-inflammatory cytokines is a key link in the pathogenesis of ACD. Against the background of infectious diseases, tumor processes, and immune conditions, T-cells and monocytes are activated, which produce cytokines such as IFN- γ , TNF- α , IL-1, IL-6, IL-10 during the immune response [1,10,14]. The pathogenetic effect of pro-inflammatory cytokines in ACD is diverse: impaired iron metabolism, decreased erythropoietin formation, and inhibition of normal erythropoiesis in the bone marrow. The pathogenesis of this phenomenon is not entirely clear. Most likely, the cause is the overproduction of cytokines (IL-1), which increase the ability of macrophages to absorb and destroy red blood cells. Due to phagocytosis of red blood cells in tissue macrophages, there is an excessive accumulation of iron in the form of hemosiderin and ferritin, which leads to a decrease in the content of the trace element in the serum depot bodies, which is considered to be the main feature of the AHZ. The clinical manifestations of ACD mainly depend on the disease with which it is associated [12,13]. In most cases, the symptoms of the underlying disease prevail over anemia, but sometimes the anemic syndrome may be its first manifestation. Patients with anemia have clinical signs of hypoxia (weakness, fatigue, general malaise, decreased concentration, shortness of breath with slight or moderate exertion, palpitations, headache). in the presence of concomitant diseases, the development of heart failure is possible. An important symptom of anemia is the

pallor of the skin, visible mucous membranes and nail beds. An objective examination shows an increase in cardiac impulse and heart sounds, the appearance of a functional systolic murmur on auscultation of the heart.

In most cases, ACD is normochromic and normocytic in nature with a low level of reticulocytes. In ACD, the indicators of serum iron and transferrin saturation are usually reduced or normal, and the level of ferritin in the blood serum is increased, the OVSS is within the normal range, low EPO production, and inadequate severity of anemia. In addition, in ACD, there is a more pronounced activation of the immune system (increased levels of hepsidin, IL-6, IFN- γ - α , TNF- α , sICAM-1, and lactoferrin) [2,3].

Conclusion. Thus, the detection of anemia in patients determines the need for mandatory clarification of the causes of this pathological condition. Timely verification of the etiology of anemia makes it possible to start adequate therapy without delay, which determines its effectiveness and improves the quality of life of people. To date, markers of chronic disease anemia (ACD) in patients have not been developed to assess the contribution of chronic disease to the progression of chronic diseases and the development of its complications, which determines the need to improve methods for early diagnosis and treatment of chronic diseases.

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