MODERN MEDICINE AND PRACTICE
https://inovatus.es/index.php/ejmmp
EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE
Vol. 3 No. 11 (Nov - 2023) EJMMP ISSN: 2795-921X

MORPHOLOGICAL CHANGES IN PATHOLOGICAL FORMS OF ERYTHROCYTES

Xalimova Yulduz Salokhiddinovna
Department of Clinical Sciences, Asian International University Bukhara, Uzbekistan

Resume: The lecture describes the main types, causes, key links of the mechanisms of development of various forms of pathology in the erythrocyte system, their main manifestations and principles of elimination.

Key words: erythrocytosis, hemolytic anemia, posthemorrhagic, dyserythropoietic.

An erythrocyte is a nuclear—free cell with a diameter of 7-8 microns (normocyte). In a healthy person, the life expectancy (circulation time in peripheral blood) of an erythrocyte is 100-120 days. The special shape of the erythrocyte in the form of a biconcave disc creates the largest surface area relative to its volume, which ensures the maximum amount of gas exchange. Typical forms of changes and pathology in the erythrocyte system include: erythrocytosis, erythropenia, anemia.

Erythrocytosis (erythremia, polycythemia) is a condition characterized by an increase above the norm in the number of red blood cells per unit volume of blood. Types of erythrocytoses Distinguish primary and secondary erythrocytoses. Primary erythrocytosis (independent forms of the disease) Primary erythrocytoses include true polycythemia (Vaquez's disease) and familial (inherited) forms. The most common of primary erythrocytoses is Wakez disease.

The key links in the pathogenesis of primary erythrocytosis in Vaquez 's disease are:

- an increase in the number of malignant tumor cells associated with myelopoiesis in hematopoietic tissue;
- excessive activation of the myeloproliferative tumor process in hematopoietic tissue, which is noted not only in the bone marrow, but also often in the spleen and liver, colonized by myelopoiesis precursor cells;
- The monoclonal nature of myeloproliferation in Vaquez's disease is evidenced by the presence in erythrocytes, granulocytes and platelets of the same chromosome defect (aberration, aneuploidy, etc.) or a defective enzyme encoded by the same mutant allele. Among secondary erythrocytoses (symptoms of other diseases, pathological conditions or processes), absolute (due to increased erythropoiesis and/ or the entry of erythrocytes into the vascular bed from the bone marrow) and relative forms are distinguished. The latter may be the result of hemoconcentration (hypovolemic erythrocytosis) and/or redistribution of blood.

In addition to Vaquez's disease, a number of familial inherited non-myeloproliferative forms of pathology are attributed to primary erythrocytosis. All of them are characterized by an increase in the number of red blood cells per unit volume of blood, hypervolemia and other signs of true polycythemia. Secondary

erythrocytoses Secondary erythrocytoses are symptoms of other diseases or pathological processes, the elimination of the causes of which leads to the elimination of secondary erythrocytoses without specialized treatment.

Secondary erythrocytoses are divided into absolute and relative. Absolute secondary erythrocytoses are characterized by an increase in the number of erythrocytes per unit volume of blood as a result of activation of erythropoiesis and the release of excess erythrocytes from the bone marrow into the vascular bed. The immediate causes of secondary absolute erythrocytosis are increased erythropoietin formation and/or increased sensitivity of erythroid cells to it. Unlike true polycythemia, erythrocytoses are usually not accompanied by thrombocytosis and leukocytosis. Relative secondary erythrocytoses are characterized by an increase in the number of red blood cells per unit volume of blood without activating their production in the bone marrow and without increasing their absolute number in the blood.

Anemia is a decrease in the total amount of hemoglobin, which is characterized by a decrease in its concentration per unit volume of blood (with the exception of acute blood loss). In most cases, anemia is accompanied by erythropenia. The exceptions are some iron deficiency conditions and thalassemia. With them, the number of red blood cells may be normal or even increased. The term "anemia" reflects only changes in the blood established by laboratory methods. Thus, anemia can either characterize a specific disease (for example, iron deficiency anemia), or be one of the symptoms of other pathological conditions.

From a practical point of view, the main and mandatory characteristic of anemia is a decrease in the hemoglobin content per unit volume of blood. The essence of anemia and its significance for the body are determined primarily by a decrease in the oxygen capacity of the blood, leading to hypoxia of the hemic type. It is with hypoxia that the main clinical symptoms and disorders of vital activity in patients with anemia are associated. Hydremia should be distinguished from anemia: conditions caused by an increase in the liquid part of the blood (hemodilution) with a normal total content of Hb and erythrocytes in the body. The concentration of hemoglobin per unit volume of blood is reduced, which gives a formal picture of anemia. In this case, we are talking about false anemia, since the total amount of Hb in the blood does not decrease. False anemia can also occur after infusion into the vascular bed of a large amount of fluid, plasma or serum. It is also necessary to remember about the possibility of developing so-called latent anemia. Thus, when the body is dehydrated in patients with anemia (vomiting, diarrhea, intense and / or prolonged sweating without replenishing the lost volume of fluid), there may be a "thickening" of blood (hemoconcentration), in which the amount of Hb in a unit of its volume may be normal or even increased, despite a decrease in its total content in the body. Anemia is usually a symptom of other diseases, so there is no strict classification of anemia.

In practice, the most accessible method for determining the content of Hb in erythrocytes is the calculation of the color index. Taking into account this indicator , anemia is isolated:

- normochromic (the color index ranges from 0.8–1.0);
- hypochromic (color index less than 0.8);
- Hyperchromic (color index is more than 1.0).

The pathogenesis of anemia, depending on the features of the mechanism of anemia development, it is divided into: posthemorrhagic; hemolytic; dyserythropoietic (due to a violation of erythropoiesis).

Acute posthemorrhagic anemia, as a rule, is a normochromic normocytic hyperregenerative anemia that occurs due to acute blood loss over a short period of time.

Chronic posthemorrhagic anemia is the result of prolonged recurrent bleeding as a result of violation of the integrity of the vessel walls (for example, with infiltration of tumor cells in them, extramedullary

hematopoiesis, severe venous hyperemia, ulcerative processes in the gastrointestinal tract, skin, mucous membranes); endocrinopathies (for example, with dyshormonal amenorrhea); disorders in the hemostasis system (for example, in violation of the vascular, platelet or coagulation mechanism in patients with hemorrhagic diathesis). The pathogenesis and signs of chronic posthemorrhagic anemia are mainly due to the increasing iron deficiency in the body.

Hemolytic anemia is a group of diseases characterized by a decrease in the average life expectancy of red blood cells and the predominance of hemolysis of red blood cells over their formation. Hemolysis (destruction of the erythrocyte) can be of two types: out- (in the spleen, liver or bone marrow) and intravascular.

Membranopathy is characterized by a violation of the protein-lipid structure and the physico-chemical state of erythroid cell membranes. The causes of membranopathies are genetic defects in the synthesis of membrane or near-membrane polypeptides of erythroid cells. The mechanism of development of membranopathies consists in the synthesis of either abnormal proteins (protein-dependent membranopathies) or lipids (lipid-dependent membranopathies, for example, in acanthocytosis). Examples of hemolytic anemia developing as a result of membranopathy can be hereditary forms of spherocytosis (Minkowski–Shoffar disease), elliptocytosis, stomatocytosis, pyropoikilocytosis, Rh-zero syndrome. Fermentopathies cause a violation of the protein-lipid structure and the physico-chemical state of erythroid cell membranes. This is observed with gene mutations of a number of their enzymes.

Dyserythropoietic hypo- and aplastic anemia are a consequence of stem cell damage combined with suppression of bone marrow functions. By origin, these anemias are divided into primary (inherited and congenital, for example, Fanconi anemia) and secondary (acquired).

Iron deficiency in the body develops in cases when its losses exceed 2 mg / day. Iron deficiency in blood plasma and body cells causes a decrease in its content in the mitochondria of erythroid cells of the bone marrow. This inhibits the synthesis of heme, its connection with globin and, consequently, the formation of hemoglobin. At the same time, the synthesis of other iron-containing compounds is disrupted, both in erythroid cells (catalase, glutathione peroxidase) and in cells of parenchymal organs (cytochromes, myoglobin, peroxidase, catalase, etc.). The lack of these enzymes in erythrocytes causes a decrease in their resistance to the damaging effect of peroxide compounds, their increased hemolysis and shortening of circulation time in the blood.

Acquired sideroblastic anemia occurs under the action of substances that inhibit the enzyme systems of protoporphyrin synthesis (for example, lead, alcohol, isoniazid, cycloserine, chloramphenicol), with tumor diseases (lymphogranulomatosis, multiple myeloma), immune autoaggressive diseases (rheumatoid arthritis), with pyridoxine deficiency (vitamin B6). Approximately 10% of patients with idiopathic sideroblastic anemia develop acute leukemia. Signs of acquired iron deficiency anemia include changes in the blood system, as well as signs of damage to the nervous system and gastrointestinal tract. The number of erythroblasts and sideroblasts in the bone marrow is increased. In peripheral blood: erythropenia (erythrocytes hypochromic, target-shaped, with basophilic punctation of the cytoplasm); reticulocytosis; increased iron content (up to 60-80 mmol / l). Iron is also found in the cells of various organs and tissues, i.e. hemosiderosis develops. The content of aminolevulinic acid in the urine is significantly increased. This is one of the most characteristic signs of lead poisoning (as a consequence of lead blockade of d-aminolevulinic acid synthetase). The altered membrane makes erythroid cells targets for their own phagocytes. The destruction of red blood cells in the bone marrow leads to increased erythropoiesis and, as a consequence, to dysplasia and deformation of bones already in childhood. Chronic (often severe) anemia leads to a violation of the physical and mental development of the patient. There is a significant increase in the liver and spleen due to the massive destruction of erythrocytes, extramedullary (extra-medullary) hematopoiesis, secondary hemochromatosis (deposition of iron in cells), which occurs with numerous

transfusions. Nucleated erythroid cells with unbalanced aggregated chains are destroyed in the bone marrow, and reticulocytes and erythrocytes circulating in the blood are destroyed in the spleen. Due to the increased destruction of erythroid cells in the bone marrow and spleen, erythropenia often develops.

Literature

1. Зильбернагль С. Ланг Ф. Клиническая патофизиология. Атлас. Пер. с англ. Под ред. П. Ф. Литвицкого. М. Практическая медицина. 2015. С. 32–45.
2. Халимова, Ю. С. & Шокиров, Б. С. (2022). Современные Данные О Морфо-Функциональных Аспектов Человеческого Организма При Злоупотребление Энергетическими Напитками. *Pedagogs Jurnali*, *4*(1), 154-161.
3. Шокиров, Б. С. & Халимова, Ю. С. (2022). ДИСБИОЗ ВЫЗВАННЫЙ АНИБИОТИКАМИ КИШЕЧНОЙ МИКРОБИОТЫ КРЫС И УСТОЙЧИВОСТЬ К САЛМОНЕЛЛАМ. *Scientific progress*, *3*(2), 766-772.
4. Литвицкий П. Ф. Клиническая патофизиология. М.: Практическая медицина. 2015. С. 497–512.
5. Халимова, Ю. С. & Шокиров, Б. С. (2021). Репродуктивность и жизнеспособность потомства самок крыс при различной длительности воздействия этанола. In *Актуальные вопросы современной медицинской науки и здравоохранения: Материалы VI Международной научно-практической конференции молодых учёных и студентов, посвященной году науки и технологий,(Екатеринбург, 8-9 апреля 2021): в 3-х т.*. Федеральное государственное бюджетное образовательное учреждение высшего образования «Уральский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
6. Cpstead L., Banasic J. Pathphysilgy. 4th Edn. Philadelphia: Saunders. 2010. P. 290–329. C
7. Halimova, Y. S. (2023). Morphological Aspects of Rat Ovaries When Exposed to Caffeine Containing Drink. *BEST JOURNAL OF INNOVATION IN SCIENCE, RESEARCH AND DEVELOPMENT*, *2*(6), 294-300.
8. Ergasheva, G. T. (2022). QANDLI DIABET BILAN KASALLANGANLARDA REABILITATSIYA MEZONLARINI TAKOMILASHTIRISH. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, *2*(12), 335-337.
9. ГТ, Э. & Саидова, Л. Б. (2022). СОВЕРШЕНСТВОВАНИЕ РЕАБИЛИТАЦИОННО-ВОССТАНОВИТЕЛЬНЫХ КРИТЕРИЕВ БОЛЬНЫХ С СД-2 ТИПА. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, *2*(12), 206-209.
10. Джалилова, З. (2022). РЕАЛИЗАЦИЯ МАКСИМ ВЕЖЛИВОСТИ В АНГЛИЙСКОМ И УЗБЕКСКОМ ДИАЛОГАХ. *Zamonaviy dunyoda innovatsion tadqiqotlar: Nazariya va amaliyot*, *1*(21), 22-33.
11. Джалилова, З. (2022). ПРАГМАТИЧЕСКИЙ ВЗГЛЯД НА МЕЖЛИЧНОСТНОЕ ОБЩЕНИЕ. *Zamonaviy dunyoda ilm-fan va texnologiya*, *1*(7), 331-336.
12. Халимова, Ю. С. (2022). МОРФОФУНКЦИОНАЛЬНЫЕ ОСОБЕННОСТИ ЯИЧНИКОВ КРЫС ПРИ ВОЗДЕЙСТВИИ КОФЕИН СОДЕРЖАЩИХ НАПИТОК. *Gospodarka i Innowacje*. *23*, 368-374.
13. Халимова, Ю. С. & Шокиров, Б. С. (2022). МОРФОФУНКЦИОНАЛЬНЫЕ ООБЕННОСТИ ВНУТРЕННИХ ОРГАНОВ ПРИ ХРОНИЧЕСКОМ АЛКОГОЛИЗМЕ. *Scientific progress*, *3*(2), 782-789.

14. Halimova, Y. S. (2023). Morphofunctional Aspects of Internal Organs in Chronic Alcoholism. *AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI*, *2*(5), 83-87.
15. Ergasheva, G. (2023). METHODS TO PREVENT SIDE EFFECTS OF DIABETES MELLITUS IN SICK PATIENTS WITH TYPE 2 DIABETES. *International Bulletin of Medical Sciences and Clinical Research*, *3*(10), 104-108.
16. Djalilova, Z. O. (2023). A DISCOURSIVE TURN IN THE THEORY OF LINGUISTIC POLITENESS: TO THE FORMATION OF THE THEORY OF LINGUISTIC IMPOLITENESS. *International Journal Of Literature And Languages*, *3*(02), 15-23.
17. Djalilova, Z. (2023). THE SIGNIFICANCE AND POSITION OF TEACHING METHODS IN PROFESSIONAL TRAINING. *Solution of social problems in management and economy*, *2*(10), 31-42.
18. Salokhiddinovna, X. Y. (2023). INFLUENCE OF EXTERNAL FACTORS ON THE MALE REPRODUCTIVE SYSTEM. *EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE*, *3*(10), 6-13.
19. Yu S, H., & BS, S. (2023). Morphological changes of internal organs in chronic alcoholism.
20. Salokhiddinovna, H. Y. (2023). Morphological Features of the Human Body in Energy Drink Abuse. *EUROPEAN JOURNAL OF INNOVATION IN NONFORMAL EDUCATION*, *3*(5), 51-53.
21. Halimova, Y. S., Shokirov, B. S., & Khasanova, D. A. (2023). Reproduction and Viability of Female Rat Offspring When Exposed To Ethanol. *Procedia of Engineering and Medical Sciences*, 32-35.
22. Shokirov, B. S. (2021). Halimova Yu. S. Antibiotic-induced rat gut microbiota dysbiosis and salmonella resistance Society and innovations.
23. Халимова, Ю. С. (2021). MORPHOFUNCTIONAL ASPECTS OF THE HUMAN BODY IN THE ABUSE OF ENERGY DRINKS. *Новый день в медицине*, *5*(37), 208-210.
24. Khalimova, Y. S. BS Shokirov Morphological changes of internal organs in chronic alcoholism. *Middle European scientific bulletin*, 12-2021.
25. Djalilova, Z. (2023). THE USE OF LATIN TERMINOLOGY IN MEDICAL CASE. *Академические исследования в современной науке*, *2*(14), 9-15.