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Morphologic Changes in Red Blood Cells

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Annotation. *The lecture characterizes the main types, causes, key links of mechanisms of development of various forms of pathology in the system of red blood cells, their main manifestations and principles of elimination.*

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

Erythrocyte is a nucleus-free cell with a diameter of 7-8 microns (normocyte). In a healthy person, the life expectancy (circulation time in the peripheral blood) of an erythrocyte is 100-120 days. The special shape of the erythrocyte in the form of a double-curved disk creates the largest surface area in relation to its volume, which provides the maximum amount of gas exchange.

Typical forms of changes and pathology in the red blood cell system include:

- erythrocytosis,
- erythropenia,
- anemia.

Erythrocytosis (erythrocythemia, polycythemia) - a condition characterized by an increase in the number of red blood cells in a unit of blood volume above normal.

There are primary and secondary erythrocytosis. Primary erythrocytosis (independent forms of the disease)

To primary erythrocytosis include true polycythemia (Vaquez's disease) and familial (inherited) forms. Vaquez disease is the most common of the primary erythrocytoses. The key links in the pathogenesis of primary erythrocytosis in Vaquez disease are:

- An increase in the number of malignant myelopoietic tumor precursor cells in hematopoietic tissue;
- excessive activation of myeloproliferative tumorigenic process in hematopoietic tissue, which is noted not only in the bone marrow, but also often in the spleen and liver colonized by myelopoiesis progenitor cells; the monoclonal nature of myeloproliferation in Vaquez disease is evidenced by the presence of the same chromosomal defect (aberrations, aneuploidy, etc.) or defective enzyme in erythrocytes, granulocytes and platelets.) or a defective enzyme encoded by the same mutant allele.

Among secondary erythrocytosis (symptoms of other diseases, pathological conditions or processes) distinguish

- absolute (due to increased erythropoiesis and / or receipt of red blood cells in the vascular bed from the bone marrow) and
- relative forms. The latter may be the result of hemoconcentration (hypovolemic erythrocytosis) and / or redistribution of blood.

Erythremia is accompanied by significant changes in the bone marrow, peripheral blood, dysfunctions of the cardiovascular and other systems. Hematologic manifestations of erythremia In the bone marrow:

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- intense proliferation of the tumor pool of myeloid cells (in the proximal and distal parts of tubular bones, as well as in the flat bones, liver and spleen);
- acceleration of iron metabolism (introduction of preparations containing 59Fe and 52Fe into the blood is accompanied by an increase in the rate of iron utilization by bone marrow tissue and its subsequent excretion);
- decrease in the mass of erythropoietic bone marrow tissue (post-erythremic myelofibrosis); in the late stages of erythremia, this leads to the development of anemia and thrombocytopenia.

In the peripheral blood:

- erythro-, reticulo-, thrombocytosis, neutrophilia (with a nuclear shift to the left to metamyelocytes and even myelocytes), eosino- and basophilia, monocytosis; - hypervolemia (polycythemic);
- increase in hemoglobin concentration (Hb, usually up to 180-200 g/l); - hypochromia of erythrocytes (is the result of lagging Hb synthesis from the rate of erythroid proliferation); In the final stages of the disease develop erythro-, thrombocyto- and even pancytopenia: a decrease in the number of all or many cells of the myeloid series due to post-erythremic myelofibrosis.

In addition to Vaquez disease, primary erythrocytosis includes a number of familial inherited non-myeloproliferative (i.e., not caused by tumor transformation of erythroid cells) forms of pathology. All of them are characterized by an increase in the number of red blood cells per unit of blood volume, hypervolemia and other signs of true polycythemia

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

Secondary erythrocytosis is subdivided into absolute and relative.

Absolute secondary erythrocytosis is characterized by an increase in the number of red blood cells per unit volume of blood as a result of activation of erythropoiesis and the release of excess red blood cells from the bone marrow in the vascular bed. The immediate causes of secondary absolute erythrocytosis are increased formation of erythropoietin and/or increased sensitivity of erythroid cells to it.

In turn, these disorders are most often a consequence of:

- general, as a rule, chronic hypoxia of any genesis (hypoxia is the most important factor stimulating erythropoietin production);
- ischemia of the kidneys, and less often of the liver and spleen (with cysts in them, edema, stenosis of their arteries, inflammation), which stimulates the production of erythropoietin;
- tumor growth accompanied by excessive production of erythropoietin (e.g., neoplasms of the kidney, liver, spleen, uterus).

Unlike true polycythemia, erythrocytoses are usually not accompanied by thrombocytosis and leukocytosis. Relative secondary erythrocytosis is characterized by an increase in the number of red blood cells per unit of blood volume without activation of their production in the bone marrow and without an increase in their absolute number in the blood.

The causes of relative secondary erythrocytosis are most often:

- decrease in blood plasma volume (hemoconcentration) when the body loses fluid (diarrhea, vomiting, plasmorrhagia in burns, lymphorrhagia); this causes the development of polycythemic hypovolemia;

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- excessive release of erythrocytes into the circulating blood from organs and tissues that deposit them (in stress reaction, acute hypoxia, hypercatecholamineemia), with the development of polycythemic hypervolemia. Signs of relative secondary erythrocytosis:
- Increase in hematocrit (Ht) as a result of hemoconcentration;
- normo- or hypovolemic polycythemia (mainly due to erythrocytosis);
- increased blood viscosity.

Anemia is a decrease in the total amount of hemoglobin, which is characterized by a decrease in its concentration per unit volume of blood (except for acute blood loss). In most cases, anemia is accompanied by erythropenia. Exceptions are some iron deficiency states and thalassemia. In 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 obligatory characteristic of anemia is a decrease in the hemoglobin content in a unit of blood volume. The essence of anemia and its importance 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 associated with the main clinical symptoms and disorders of vital activity in patients with anemia. Hydremia should be distinguished from anemia: conditions caused by an increase in the liquid part of the blood (hemodilution) with a normal total body content of Hb and red blood cells. The concentration of hemoglobin per unit volume of blood is reduced, which gives a formal picture of anemia. In this case, it is a false anemia, because the total amount of Hb in the blood is not reduced. False anemia can be observed after infusion of large amounts of fluid, plasma or serum into the vascular bed. It is also necessary to remember the possibility of the development of so-called hidden anemia. Thus, with dehydration of the body in patients with anemia (vomiting, diarrhea, intense and/or prolonged sweating without replenishing the lost volume of fluid) may be observed "thickening" of the blood (hemoconcentration), in which the amount of Hb per unit volume may be normal or even increased, despite a decrease in its total content in the body.

Chronic posthemorrhagic anemia is the result of prolonged recurrent bleeding as a result of disruption of vessel wall integrity (e.g., with infiltration of tumor cells, extramedullary hematopoiesis, marked venous hyperemia, ulcerative processes in the gastrointestinal tract, skin, mucous membranes); endocrinopathies (e.g., in dyshormonal amenorrhea); disorders in the hemostasis system (e.g., in disorders 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 - a group of diseases characterized by a decrease in the average lifespan of red blood cells and the predominance of hemolysis of red blood cells over their formation.

Hemolysis (destruction of the red blood cell) can be of two types: extra- (in the spleen, liver, or bone marrow) and intravascular.

Types of hemolytic anemia According to the degree of replacement of destroyed cells with new red blood cells, compensated and uncompensated anemia are distinguished. By etiological factor anemia is differentiated into idiopathic (the cause is not detected) and symptomatic, or secondary (caused, for example, by taking medications); By the course distinguished acute, subacute and chronic hemolytic anemia. According to the origin, type of defect in red blood cells or and/or pathogenic effect on them, anemias are divided into primary (hereditary and congenital) and secondary (acquired, symptomatic; Table).

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Hemolytic anemias arise from defects in either the red blood cells themselves (intracellular factors) or from causes external to the red blood cells (extracellular factors). Usually, intracellular factors are inherited or congenital, and extracellular factors are acquired.

The microenvironment of erythrocytes is represented by blood plasma and vascular endothelium. The presence of auto- or isoantibodies, toxic substances or infectious agents in the blood plasma causes changes in the plasmolemma of the erythrocyte, which leads to its destruction. This can be observed in isoimmune hemolytic anemia in fetuses with erythroblastosis, microangiopathies (e.g., hemolytic microangiopathic anemia), paroxysmal cold hemoglobinuria, hemolysis of erythrocytes of enzymopathic genesis, exposure to certain drugs (e.g., sulfonamides, antimalarial drugs).

Intracellular defects that are generally inherited (excluding paroxysmal nocturnal hemoglobinuria) include:

- Membranopathies (e.g., inherited spherocytosis and elliptocytosis, paroxysmal nocturnal hemoglobinuria);
- hemoglobinopathies (e.g., sickle cell anemia) - there are over 300 known diseases caused by point mutations in globin genes, the defect in the globin molecule disrupts its polymerization; this alters the membranes and shape of the erythrocyte and increases susceptibility to hemolysis;
- enzymopathies (e.g., increased activity of adenosine deaminase - ADA gene, 102700, 20q13.11);
- deficiency of adenylate kinase, glucose-6-phosphate dehydrogenase, hexokinase, glutathione peroxidase, glutathione reductase, glutathione synthetase, glucose6-phosphatizomerase, diphosphoglycerate mutase, pyruvate kinase, phosphofructokinase and other enzymes. The general mechanism of erythrocyte lysis consists in disorganization of the phospholipid-protein structure of their membrane.

Hemolysis of erythrocytes in primary (inherited and congenital) hemolytic anemias is caused by genetic partial or combined defects in: the structure of their membranes (membranopathies), enzymes (enzymopathies), hemoglobin (hemoglobinopathies). Membranopathies are characterized by disruption of the protein-lipid structure and physicochemical 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 membranopathies is the synthesis of either abnormal proteins (protein-dependent membranopathies) or lipids (lipid-dependent membranopathies, such as in acanthocytosis). Examples of hemolytic anemias that develop as a result of membranopathies include hereditary forms of spherocytosis (Minkowski-Schoffar disease), elliptocytosis, stomatocytosis, pyropoikilocytosis, and Rh-zero syndrome.

Fermentopathies cause a violation of the protein-lipid structure and physicochemical state of erythroid cell membranes. This is observed in gene mutations of a number of their enzymes.

A large number of hemoglobinopathies accompanied by hemolytic anemia have been described. Thalassemias are an example of these.

Dyserythropoietic anemia is differentiated according to its origin. Dyserythropoietic hypo- and aplastic anemias are the result of stem cell damage combined with suppression of bone marrow function.

According to their origin, these anemias are divided into primary (inherited and congenital, such as Fanconi anemia) and secondary (acquired).

The causes may be factors of different nature: physical (e.g., ionizing radiation); chemical (most often drugs, such as chloramphenicol, phenylbutazone, immunosuppressants, meprobamate, chlorpromazine, cytostatics, etc.); biological (mainly viruses, for example, causing hepatitis, infectious mononucleosis and others, as well as anti-erythrocytic antibodies and T-cytotoxic lymphocytes). Pathogenesis of

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hypo- and aplastic anemias. Exposure to physical factors (e.g., high doses of ionizing radiation) causes bone marrow hypoplasia. Its severity depends on the dose of radiation. The basis of hypoplasia of hematopoietic tissue is irreversible damage and death of stem cells up to their complete disappearance, observed in aplasia. Chemical and biological factors (e.g., viruses and certain drugs) inhibit the synthesis of nucleic acids and protein in stem cells, disrupt their cellular and/or physicochemical microenvironment.

Dyserythropoietic anemia due to impaired globin DNA synthesis These anemias are typically hyperchromic macrocytic with a megaloblastic type of hematopoiesis. Megaloblastic erythropoiesis results from impaired DNA synthesis in the setting of vitamin B12 (cyancobalamin) or folic acid deficiency, as well as methionine synthetase and dihydrofolate reductase deficiencies.

Sickle cell anemia is characterized by moderate chronic hemolysis of red blood cells, recurrent acute painful crises, and increased susceptibility to infectious diseases (especially those caused by Streptococcus pneumoniae). It is caused by an abnormal form of hemoglobin, hemoglobin S (HbS). Etiology. At the molecular level, a defect in the HBB gene is identified. HbS is formed as a result of substitution of valine for glutamic acid in position 6 -chain of hemoglobin molecule, as well as other mutations. In the venous channel (in hypoxia) HbS polymerizes with the formation of long chains that change the shape of erythrocytes (they become sickle-shaped). Sickle-shaped erythrocytes increase blood viscosity and cause stasis, create mechanical obstacles in small arterioles and capillaries, leading to tissue ischemia (which is associated with pain crises). In addition, sickle-shaped erythrocytes are less resistant to mechanical influences, which leads to their hemolysis. Sickle cell anemia is manifested by moderate jaundice, trophic ulcers in the ankle area, stunted physical development (especially in boys), priapism, painful crises, splenomegaly, cholelithiasis, avascular necrosis, leg ulcers, osteonecrosis with the development of osteomyelitis.

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