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IRON DEFICIENCY ANEMIA IN CHILDREN

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Annotation*: This is the literature review devoted to modern trends of diagnostics and treatment of iron deficiency anemia in children. Forms of iron deficiency, the etiology of iron deficiency and laboratory diagnostics standards, the modern view on the selection of iron supplementation for the treatment of children with iron deficiency anemia are presented.*

Keywords: *children, iron-deficiency anemia, iron supplementation.*

ПVital signs; more than 700 patients with mild to moderate IDA sought advice at a hematologist's appointment. IDA is a polyethological disease, the occurrence of which is associated with D in the body due to a violation of its intake, assimilation or increased losses, characterized by microcytosis and hypochromic anemia. LV — an acquired condition in which there is latent (latent) For example, a decrease in iron reserves in the body and its insufficient content in tissues (sideropenia, hyposiderosis), but there is no anemia yet. Categories assigned to various iron-deficient conditions (IDDS) in the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) [4]:

Anemia that complicates pregnancy, childbirth and the postpartum period O99. 0 Iron is an essential trace element, is part of the structure of proteins, participates in the work of enzymatic systems that ensure systemic and cellular aerobic metabolism, as well as redox homeostasis of the body. Iron, being a structural component of a number of enzymes, is involved in the transport of electrons (cytochromes, ironseroproteins), oxygen (myoglobin, hemoglobin), as well as in redox reactions (oxidases, hydrolases, superoxide dismutase). Disruption of the functioning of iron-containing proteins present in all organs and tissues leads to changes in a number of vital processes [5, 6]. It is known that iron is not only a component of various heme proteins, which are necessary for the normal implementation of redox reactions, but also a cofactor of some enzymes. Trivalent iron from food is reduced to divalent iron by means of copper-dependent ferroreductase on the enterocyte membrane under the action of vitamin C and enters the enterocyte through manganese-dependent divalent metal transport proteins (DMT proteins), from where it passes through the protein ferroportin on the basement membrane into the blood, where it is oxidized to a trivalent state by means of copper-dependentfer rooxidases to form a ferric acid. Contact with the transport protein transferrin [6, 7]. This complex (transferrin — iron) interacts with specific receptors on the membrane of bone marrow erythroid cells, and then penetrates into cells, where iron is transferred to mitochondria and incorporated into protoporphyrin, participating in the formation of heme. Normally, transferrin is approximately 30% saturated with iron. Iron-free transferrin can be involved in iron transport several times. Iron is stored in the depot as part of the ferritin and hemosiderin proteins. Iron can also enter the depot during the natural destruction of red blood cells. With a normal iron balance in the body, a balance is established between the content of ferritin in plasma and depot. The level of ferritin in the blood reflects the amount of iron deposited. In the erythron system, iron deficiency develops, which leads to a decrease in the production of red blood cells and reticulocytes and to reduce their hemoglobin. Thus, ineffective erythropoiesis may develop. Then there is a depletion of iron

reserves in the depot — the content of ferritin and hemosiderin decreases. Hormonal factors play an important role in the development of IDA, especially in adolescence, as androgens stimulate erythropoiesis, iron absorption and utilization, and hemoglobin synthesis, while estrogens inhibit these processes. The main reasons for the development of IDA in children: insufficient intake of microelements with food (alimentary deficiency); increased body need for iron due to the rapid growth rate of the child, excessiveweight gain;reducedabsorption of the microelement; iron loss from the body exceeding physiological. Depending on the time of exposure, the following causes of IDA development in children are distinguished [6]: Protein, Enzyme Localization Function Hemoglobin Erythrocytes Oxygen transport to tissues Myoglobin Muscles Oxygen storage Iron-containing mitochondrial proteins (cytochromes, etc.) Most cells Energy production in cells (ATP) Transferrin Blood Iron transport Ferritin/hemosiderin Liver, spleen, bone marrow Iron depot Iron-containing enzymes (catalases, peroxidases, xan the oxidases) There are various functions in the metabolism everywhere: insufficient food intake; increased iron requirements at high growth rates; increased iron loss due to bleeding; impaired ironin take during malabsorption; impaired iron metabolism due to hormonal changes in puberty. The development of cancer in the body has a clear stage. There are consistently developing stages of IDC: L J, characterized by a decrease in iron reserves in the depot and beginning iron-deficient erythropoiesis; IDA, characterized by a combination of sideropenic and anemic syndromes. Most authors believe that laboratory methods of investigation are of primary importance in the diagnosis of IDA, but clinical data representing a combination of two syndromes — sideropenic and anemic-also play a certain role [8]. Sideropenic syndrome is characterized by the following symptoms: dystrophic changes in the skin and appendages, mucous membranes; perversion of taste and smell; muscle pain;muscle hypotension; changes in the nervous system in the form of delayed psychomotor development and impaired cognitive functions. Anemic syndrome is characterized by the following symptoms: asthenovegetative disorders; pallor of the skin and mucous membranes; changes in the cardiovascular system [6]. The presence of J at an early age has delayed adverse effects, as evidenced by the results of a study by B. Lozoff and her colleagues published in 1987 [9, 10]. Children in the first years of life have lower indices of psychomotor development on the Bayley scalethan their peers, and later they develop a decrease in memory, learning ability and concentration, delayed speech development, and decreased physical activity. The diagnosis of IDA is based on the detection of microcytic hypochromic normo- or hyporegenerative anemia in a clinical blood test performed by a "manual" method or on an automatic hematological analyzer that allows you to measure a number of red blood cell indices. Additionally, the laboratory assistant describes the morphological changes of red blood cells — anisocytosis and kilocytosis. IDA criteria are: decrease in hemoglobin concentration (Hb less than 110 g / l in children under 6 years of age, less than 120 g/l in children over 6 years of age);a slight decrease in the numberof erythrocytes (less than 3.8 x 1012/l); a decrease in the vetовoro index (less than 0.85); an increase in ESR (more than 10-12 mm / h) reduced or normal reticulocyte count (10-20%); decreased red blood cell volume (MCV less than 80 fl), average Hb content in red blood cells (MCH less than 26 pg.), average Hb concentration in red blood cells (MCHC less than 320 g/L), increased red blood cell anisocytosis (RDW more than 14%). IDC is confirmed by a biochemical blood test, which focuses on a decrease in the concentration of serum iron (LVL less than 12.5 mmol/l), an increase in the total iron binding capacity of serum (LVL more than 69 mmol/L), a decrease in the transferrin saturation coefficient with iron (LVL less than 17%), a decrease in the concentration of serumferritin (SF less than 30 ng/l).ml or mcg/l). In recent years, it has become possible to determine soluble transferrin receptors (RTFRS), the concentration of which increases under conditions of iron deficiency (more than 2.9 mcg/ml) [11]. In recent years, several new biochemical parameters

have been proposed for the diagnosis of J. P. Blavatsky. One of them is zinc protoporphyrin (CPP) — the first biochemical sign that reflects changes in red blood cells when the body's iron supply decreases. CPP is formed as a result of the substitution of zinc for the iron ion in protoporphyrin at the last stage of heme biosynthesis. CPP is measured in a drop of whole capillary blood using a portable device-a hematofluorometer (the device is not registered in Russia). The result is ready in 1 minute. The cost of one measurement is about $ 5. Normal CPP values are in the range of 30-80 mmol/ mol of heme. An increase in the indicator above this level indicates that J. The advantage of this method is the speed and simplicity of execution, low cost and high sensitivity. This method is widely used abroad as a screening test for identifying individuals with J. The disadvantages of CPP include its increase in lead poisoning, infection, inflammation, and pregnancy, as well as its lower specificity compared to SF [6]. In complex cases, as a differential diagnosis of anemia, it is also necessary to determine the concentration of hepcidin in the blood serum. Hepcidin is a protein synthesized by hepatocytesand is a key regulator of iron homeostasis, inhibiting its absorption in the small intestine and release from macrophages and hepatocytes. With inflammation or infection, the concentration of hepcidin increases, and with hypoxia and IDA decreases, which contributes to rapid mobilization of iron from the depot, increased iron absorption in the duodenum and saturation of erythron the required amount of iron [6]. Along with the early diagnosis of IDD, monitoring the effectiveness of treatment of patients with IDD with iron preparations is of great importance. For this purpose, early (up to 1 month from the start of treatment with iron preparations) and late (from 1 to 6 months) criteria are currently used. Early criteria for the effectiveness of therapy include: an increase in the average Hb content in reticulocytes (ReticulocyteHemoglobinContent, CHr); an increase in the difference between CHr and MCH; the appearance of immature reticulocyte fraction an increase in the number of reticulocytes relative to the initial one on days 7-10 from the start of treatment with jellyza (reticulocytic reaction); increased Hb oncentration by 10 g / l and hematocrit by 3% by the end of the 4th week of treatment. Late criteria for the effectiveness ofiron therapy include: the disappearance of clinical symptoms of the disease after 1-3 months from the start of treatment; overcoming tissue sideropenia after 3-6 months from the start of treatment (depending on the severity of anemia), determined by the normalization of SF concentration (more than 30 mcg/l). Reticulocyte indices are the earliest criteria for response to therapy, and their use to assess the effectiveness of treatment allows you not to wait for 1 month for an increase in the concentrationof Hb. Special attention should be paid to the CHr indicator, which allows us to judge the current supply of iron to the bone marrow, reflects the synthesis of Hb in bone marrow precursors of red blood cells and the availability of iron for erythropoiesis. It reflects the state of erythropoiesis over the last 1-2 days, allowing us to assess the effectiveness of therapy in the first days from its start and timely correct the treatment regimen if necessary [12]. The goal of IDA treatment is to eliminate the underlying cause of the disease (correct nutrition, identify and eliminate the source of blood loss), and compensate for iron deficiency in the body. Substances that reduce the absorption of this trace element include soy protein, phytates, calcium, dietary fiber, and polyphenols. Thus, when planning a proper diet for children, it is important to take into account the above features of iron metabolism. A large number of iron preparations have been developed for the treatment of IDA, and there are even attempts to classify them. Iron preparations, depending on the route of administration into the body, are divided into oral and parenteral. Further separation of iron preparations is carried out on a different basis: depending on the chemical composition, iron preparations can be simple (sulfate, gluconate, lactate, fumarate), most often containing iron salts, and combined, which, in addition to iron compounds, include other substances (ascorbic and folic acids, vitamin B12). In addition to salt preparations of iron, there is a group of preparations based on polymaltose hydroxide.

Complex (HPA) of ferric iron, which are produced both in pure form and in combination with other substances (for example, folic acid) [14]. The main requirements for oral iron preparations used in children's practice are: high efficiency, high safety, good organoleptic properties, and availability of dosage forms that are convenient for patients of all ages. Iron (III) preparations based on HPA meet these requirements to the greatest extent [6]. Randomized studies in recent years have shown that the effectiveness of iron salt preparations and iron (III) preparations based on HPC in the treatment of IDA is the same [15].

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