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Latent Iron Deficiency Anemia in Children, Anthropometric Features

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Abstract: The article presents data on the incidence, etiology, pathogenesis and clinical manifestations of iron deficiency anemia (IDA), principles of diagnosis and treatment of the disease in children and adolescents. Various types of prevention are described: at the population level (fortification), in groups at risk of developing IDA (supplementation), primary and secondary prevention. The recommendations of the American Academy of Pediatrics for the prevention of IDA in the most vulnerable group of the population—infants and young children—are provided.

Keywords: children, adolescents, iron deficiency anemia, diagnosis, treatment, iron supplements, prevention.

Iron deficiency anemia (IDA) is an acquired disease from the group of deficiency anemias; accompanied by microcytic, hypochromic, normoregenerative anemia; clinically manifested by a combination of sideropenic and anemic syndromes. IDA includes the following nosological forms that have corresponding codes according to the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10): IDA (D50), chronic posthemorrhagic anemia (D50.0), anemia complicating pregnancy, childbirth and the postpartum period (O99.0).

In childhood, IDA accounts for 90% of all anemias.

It is found in all countries of the world, but its prevalence depends on socio-economic conditions, income, diet and other factors. Infants and young children, adolescents, women of childbearing age, pregnant and lactating women are at greatest risk of developing IDA [1]. In table Figure 1 shows the prevalence of anemia when diagnosed by reduced hemoglobin (Hb) concentration in various age groups in developed and developing countries. According to WHO experts , when IDA is detected in more than 40% of the population, the problem ceases to be medical and requires a decision at the state level [1].

Etiology of IDA The main cause of iron deficiency in the human body, according to WHO experts, is improper (poor) nutrition. Much less often, IDA develops as a result of helminthic infestations or as a result of chronic posthemorrhagic anemia.

The main reasons for the development of IDA in children and adolescents: iron deficiency at birth (fetoplacental transfusion); nutritional iron deficiency due to unbalanced nutrition; increased body needs for iron (rapid growth of a child aged 1–3 and 14–16 years, professional sports, pregnancy); loss of iron from the body exceeding physiological (bleeding of various etiologies, including heavy menstrual blood loss, postpartum anemia).



Nutritional-dependent factors in the development of iron deficiency in children are [2]: insufficient intake of iron from food (exclusive breastfeeding over 4 months, late introduction of complementary foods, unbalanced diet, vegetarianism); • reduced iron absorption; • increased iron loss.

The causes of the development of chronic posthemorrhagic anemia in men can be various diseases of the gastrointestinal tract - gastrointestinal tract (ulcerative bleeding, colon polyps, ulcerative colitis, intestinal angiomatosis, Meckel 's diverticulum, bleeding from hemorrhoids, tumors of the stomach and intestines); In women, among the causes of chronic posthemorrhagic anemia, uterine bleeding is in first place, and gastrointestinal diseases occupy second place.

Pathogenesis of IDA IDA is a group of anemic conditions of various etiologies, the main pathogenetic factor in the development of which is iron deficiency in the body (sideropenia). As a result of iron deficiency in the body, Hb synthesis is disrupted and its content in red blood cells decreases. The number of red blood cells may also decrease slightly. Since the number of red blood cells and Hb content decrease unevenly, hypo- chromia of erythrocytes, which reflects a reduced color index (CI). Impaired Hb formation leads to a decrease in erythrocyte indices - mean erythrocyte volume (MCV) and Hb content in erythrocytes (MCH). Anisocytosis becomes pronounced (due to the predominance of small erythrocytes). In the bone marrow, erythroid hyperplasia with a predominance of polychromatophilic or oxyphilic normoblasts is detected.

Erythroid cells containing hemosiderin decreases sharply. Depletion of iron reserves leads to a breakdown of redox reactions in tissues, which causes damage to the skin, mucous membranes, gastrointestinal dysfunction, and a decrease in the activity of many enzymes containing iron.

Clinical manifestations The development of IDA is preceded by *latent* iron deficiency, which does not have an independent code according to ICD-10, but is characterized by - characterized by the presence of sideropenic syndrome. Clinical manifestations of IDA represent a combination of two syndromes: sideropenic anemic [3, 4]. It is believed that the presence of 4 or more of these symptoms is pathognomonic for latent iron deficiency and IDA [5]. Anemic syndrome is associated with a decrease in Hb concentration and accompanies anemia of any origin. Clinical manifestations depend on the severity of anemia. In patients with mild or moderate anemia, significant symptoms are often absent. With the rapid development of anemia to m- pensatory mechanisms do not have time to form, which means clinical manifestations will be more pronounced than with a disease of the same severity, but developing gradually.

Consequences of iron deficiency Iron deficiency can lead to long-term and irreversible consequences. The most serious of them at an early age is delayed psychomotor development and impairment of cognitive functions [6, 7]. These children do poorly in school and have more behavior problems in middle age. Iron deficiency can also lead to delayed sexual development, cause chronic fatigue syndrome, affect the immune status, increase the risk of infectious diseases, disrupt the functioning of the endocrine glands and nervous system, and increase the absorption of heavy metals.

In older children and adults, as a result of iron deficiency, memory clearly and significantly deteriorates, shifts appear in the psyche, especially in its late-formed elements (for example, the desire for collective communication, establishing friendly relations, the ability to forgive petty insults). As a result, the individual becomes withdrawn, unjustifiably uncompromising, and nervousness and irritability increase.

Laboratory diagnostic criteria for IDA There are 3 options for laboratory diagnosis of IDA: clinical blood test performed manually; blood test performed on an automatic hematology analyzer; blood chemistry.

Hb concentration (< 110 g/l), a slight decrease in the number of red blood cells (< 3.8 1012/l), decreased CP (<0.85), increased ESR (> 10–12 mm/h), normal reticulocyte content (1–2% or 10–20 ‰). Additionally, the laboratory doctor describes the presence of anisocytosis and poikilocytosis of erythrocytes.



In a clinical blood test performed on an automatic hematology analyzer, changes not only in the above-mentioned displays can be detected; tel, but also a number of erythrocyte indices. The values of MCV decrease (<80 fl), MCH (<26 pg), the average concentration of Hb in the erythrocyte - MCHC (<320 g/l), increase - The degree of anisocytosis of erythrocytes is RDW (>14%).

In addition, modern automatic hematologist and Chemical analyzers allow you to determine the availability of functional iron. To do this, determine the proportion of hypochromic erythrocytes and the Hb content in the reticulocyte. Under physiological conditions, the proportion of hypochromic erythrocytes is <5% of the total number of erythrocytes. The Hb content in the reticulocyte > 28 pg indicates sufficient iron reserves for Hb synthesis and erythropoiesis.

In a biochemical blood test, they focus on a decrease in the concentration of serum iron - SI (< 12.5 μ mol / L), an increase in the total iron - binding capacity of serum - TIBC (> 69 μ mol / L), a decrease in the coefficient of transferrin saturation with iron - NTZ (< 17%), which is calculated by the formula: as well as a decrease in the concentration of serum ferritin (< 30 ng / ml). Serum ferritin is a generally accepted marker of iron stores in the body, but it should be remembered that serum ferritin is an acute phase protein of inflammation, so its concentration increases during inflammation, infections, and illness . liver diseases and malignant neoplasms.

Serum ferritin can be reliably used to diagnose iron deficiency only if the above conditions are excluded, i.e., there are no clinical and laboratory (C-reactive protein - CRP) signs of inflammation.

Another indicator that allows you to identify iron deficiency is erythrocyte protoporphyrin , or zinc protoporphyrin , which is formed as a result of the substitution of zinc for the iron ion in protoporph and - rine at the last stage of heme biosynthesis . Normally, such changes in heme synthesis occur in one out of 30 thousand heme molecules , however, with iron deficiency, such disturbances in heme synthesis are more common.

To determine zinc protoporphyrin, a hematofluorimeter is used - a small portable device that is relatively easy to use. Zinc protoporphyrin is determined in a drop of whole capillary blood, and the result is ready in approximately 1 minute. The disadvantages of the method include an increase in the indicator during inflammation and lead poisoning. Unfortunately, the determination of zinc protoporphyrin is rarely used in Russia, but can be used as a primary screening for diagnosing uncomplicated iron deficiency in the early stages, even before the development of anemia [8].

Iron deficiency is indicated by an indicator value $>70\text{--}80~\mu\text{mol}$ /mol of heme .

In recent years, as a diagnostic test for determining iron deficiency and the state of erythropoiesis , it has become possible to determine a solution and we are X transferrin receptors (rTFR), the concentration of which increases sharply under conditions of iron deficiency (> 2.9 μ g/ml). The advantage of this indicator over serum ferritin is the absence of changes in its values due to infection or inflammation [9], however, the method is expensive and cannot always be used in routine clinical practice.

The above changes in erythrocyte indices and biochemical parameters with normal Hb concentration indicate latent iron deficiency.

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