



Iron Deficiency Anemia in Children and Adolescents

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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. The most commonly used iron preparations in Uzbekistan for oral therapy, intramuscular and intravenous administration, their advantages and disadvantages, as well as principles for monitoring the effectiveness of treatment are presented.

Keywords:

Children, adolescents, iron deficiency anemia, diagnosis, treatment, iron supplements, prevention.

Introduction

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).

Materials And Methods

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. Those at greatest risk of developing IDA are infants and young children, adolescents, women of childbearing age, and pregnant and lactating women [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 a medical one and requires a decision at the state level [1].

Table 1. Prevalence of anemia (in%) in different age groups, based on determination of Hb concentration (cited from [1])

Population group	The developed countries	Developing countries
Children aged 0 to 4 years	20,1	39
Children aged 5 to 14 years	5,9	48,1
Pregnant women	22,7	52
All women aged 15 to 59 years	10,3	42,3
Men aged 15 to 59 years	4,3	30
Persons aged 60 years and older	12	45,2

Results And Discussion

The main cause of iron deficiency in the human body, according to WHO experts, is improper (poor) nutrition. Much less commonly, 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 that exceeds 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 of age, 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 take 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 erythrocytes and Hb content decrease unevenly, erythrocyte hypochromia develops, 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).

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 the presence of sideropenic syndrome (Table 2). Clinical manifestations of IDA represent a combination of two syndromes: sideropenic and anemic [3, 4] (see Table 2). 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, compensatory 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.

Table 2. Symptoms of sideropenia and anemia characteristic of latent iron deficiency and IDA

Symptoms of sideropenia	Symptoms of anemia
<ul style="list-style-type: none"> • Perversion of taste (addiction to clay, chalk, raw meat, dough, dumplings) • Perversion of the sense of smell (addiction to strong odors of perfume, varnish, paint, acetone, car exhaust) • Dysphagia • Drowsiness 	<ul style="list-style-type: none"> • Weakness • Lethargy • Decreased appetite • Decreased performance • Decreased exercise tolerance • Irritability • Dizziness

<ul style="list-style-type: none"> • Skin changes (dryness, appearance of small café au lait pigment spots) • Hair changes (dryness, brittleness, split ends, hair loss up to alopecia areata) • Changes in nails (cross-striations of the nails of the thumbs, in severe cases, toenails, brittleness, softness, thinning) • Changes in the mucous membranes (angular stomatitis, cheilosis, glossitis, atrophic esophagitis and gastritis) • Dyspeptic symptoms, tendency to constipation • Menstrual irregularities • Low-grade fever • Pain in the calf muscles 	<ul style="list-style-type: none"> • Noise in ears • Headache • Fainting • Shortness of breath • Paleness of the skin and mucous membranes • Arterial and muscular hypotension, including hypotension of the bladder muscles with the development of urinary incontinence • Tachycardia • Expansion of the boundaries of the heart • Muffled heart sounds • Systolic murmur at the apex of the heart
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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]. 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, changes 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.

IDA laboratory diagnostic criteria

There are 3 options for laboratory diagnostics of IDA:

- clinical blood test performed by the “manual” method;
- blood test performed on an automatic hematological analyzer;
- blood chemistry.

In a clinical blood test performed by the “manual” method, they are guided by a decrease in Hb concentration (< 110 g/l), a slight decrease in the number of erythrocytes (< 3.8 × 10¹²/l), a decrease in CP (< 0.85), an increase in 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.

Disease prognosis

The prognosis of the disease is favorable: cure occurs in the vast majority of cases.

So-called “relapses” of the disease are possible:

- when using low doses of iron supplements;
- ineffectiveness of oral iron supplements, which is rare;
- reducing the duration of treatment for patients;
- treatment of chronic posthemorrhagic anemia with an unidentified or unresolved source of blood loss.

It is extremely rare that a cure cannot be achieved with standard iron therapy. American scientists have found that the poor response to iron therapy in IDA is due to the presence of a mutation in the TMPRSS6 gene, which leads to excess production of hepcidin, which blocks the absorption of iron in the intestine and prevents its release from macrophages [6]. This is why anemia in such patients cannot be treated with either oral or intravenous iron supplements.

Conclusion

Taking iron supplements for preventive purposes is intended for people at risk who do not have the opportunity to receive foods fortified with iron. It should be remembered that iron from fortified foods is included in erythropoiesis to a lesser extent than when prescribing iron supplements.

Unfortunately, such scientifically based recommendations for the prevention of iron deficiency in various population groups have not yet been formulated by domestic scientists. The development of national recommendations for the prevention and treatment of iron deficiency conditions and their approval at the state level will make it possible to solve a complex medical and social problem.

named after G. N. Speransky. — 2018; 87(1):67–74.

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