FEATURE IRON DEFICIENCY ANEMIA IN CHILDREN

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Abstract. The article describes various types of prevention: at the population level (fortification), in groups at risk of developing IDA (saplimentation), primary and secondary prevention. Pediatric recommendations for the prevention of IDA in the most vulnerable group of the population—infants and young children—are provided.

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

INTRODUCTION

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 a medical one and requires a decision at the state level [1].

MATERIALS AND METHODS

The main cause of iron deficiency in the human body, according to WHO experts, is improper (poor) nutrition. Much less frequently, 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).

Table 1. Prevalence of anemia (%) in different age groups based on Hb concentration determination

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 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, stomach and intestinal tumors); In women, among the causes of chronic posthemorrhagic anemia, uterine bleeding is in first place, and gastrointestinal diseases take second place.

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 are 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
• 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 • 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	• Weakness • Lethargy • Decreased appetite • Decreased performance • Decreased exercise tolerance • Irritability • Dizziness • 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

Differential diagnosis of IDA The differential diagnosis of IDA should be carried out, first of all, with other “deficiency” anemias, which (in addition to IDA) include megaloblastic anemia due to deficiency of folic acid and vitamin B12. Clinical manifestations of these anemias are glossitis, premature graying of hair, signs of severe anemia, bleeding, jaundice, depression or psychosis, subacute degeneration of the spinal cord (only with vitamin B12 deficiency). The main distinguishing sign of folic acid and vitamin B12 deficiency is the macrocytic nature of anemia (MCV > 120 fL). In addition, there is a decrease in the concentration of Hb, the number of erythrocytes, hematocrit (Ht), pancytopenia

(decrease in the number of erythrocytes, leukocytes, platelets), hypersegmentation of neutrophil nuclei (5- and 6-lobed nuclei), the presence of megaloblasts in the bone marrow and its hypercellularity [4]. In difficult cases, it is necessary to determine the concentration of folic acid in blood serum (norm: 7.2–15.4 ng/ml) and red blood cells (norm: 125–600 ng/ml), as well as the concentration of vitamin B12 in blood serum. (normal range is 208–964 pg/ml). Differential diagnosis is also carried out between IDA and anemia in chronic diseases (ACD). The main causes of the development of ACB: chronic infections (tuberculosis, sepsis, osteomyelitis, HIV/AIDS); autoimmune diseases (rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel diseases - ulcerative colitis, Crohn's disease); diabetes; chronic liver diseases (hepatitis, cirrhosis), kidneys; malignant neoplasms [2]. The key mediator of ACB is hepcidin, a humoral regulator of iron metabolism. Inflammation causes macrophages to produce a pro-inflammatory cytokine, interleukin 6, which stimulates the production of hepcidin by hepatocytes. This protein, in turn, blocks the absorption of iron in the small intestine and the release of iron from the depot, promoting its accumulation in macrophages, which leads to a decrease in serum iron concentration and the development of anemia [3].

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 a poor response to therapy with iron

supplements in IDA is due to the presence of a mutation in the TMPRSS6 gene, which leads to excessive production of hepcidin, which blocks the absorption of iron in the intestine and prevents its release from macrophages [5]. This is why anemia in such patients cannot be treated with either oral or intravenous iron supplements.

CONCLUSION

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.

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