ISSN: 2582-4686 SJIF 2021-3.261, 2022-2.889, 2023-5.384, 2024-6.875 ResearchBib IF: 9.948 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-5, ISSUE-2

DEFICIENCY ANEMIAS IN EARLY CHILDHOOD: IRON DEFICIENCY ANEMIA

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Abstract. The article presents a literature review of modern trends in the diagnosis and treatment of iron deficiency anemia in children. The forms of iron deficiency are determined, the causes of development and criteria for laboratory diagnostics are given, a modern view on the choice of drugs for the treatment of children with iron deficiency anemia.

Keywords: children, iron deficiency anemia, IDA, iron preparations.

INTRODUCTION

Anemia is a pathological condition characterized by a decrease in the hemoglobin (Hb) content in a unit of blood volume [1]. According to WHO, anemia is extremely widespread: in certain age periods in developing countries, it is detected in almost half of the population. Iron deficiency (ID) ranks first among the 38 most common human diseases - more than 3 billion people on Earth suffer from it [2]. The risk of developing ID, both latent (LD) and manifest (iron deficiency anemia - IDA), is highest in children (especially in the first two years of life) and women of reproductive age [3]. Iron deficiency anemia accounts for 90% of all childhood anemias.

MATERIALS AND METHODS

IDA is a polyetiological disease, the occurrence of which is associated with ID in the body due to a violation of its intake, absorption or increased losses, characterized by microcytosis and hypochromic anemia. LDA is an acquired condition in which there is latent (hidden) ID, a decrease in iron reserves in the body and its insufficient content in tissues (sideropenia, hyposiderosis), but there is no anemia yet. Headings assigned to various iron deficiency disorders (IDD) in the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) [4]:

- *IDA E61.1*
- *IDA D50*
- *Chronic posthemorrhagic anemia D50.0*
- *Sideropenic dysphagia D50.1*
- *Other IDA D50.8*
- *Unspecified IDA D50.9*
- *Anemia complicating pregnancy, childbirth, and the postpartum period O99.0*

Iron is an essential microelement, is part of the structure of proteins, and is involved in the work of enzymatic systems that ensure systemic and cellular aerobic metabolism, as well as oxidation-reduction homeostasis of the body. Iron, being a structural component of a number of enzymes, is involved in the transport of electrons (cytochromes, iron-sulfur proteins), oxygen (myoglobin, hemoglobin), as well as in oxidation-reduction reactions (oxidases, hydrolases, superoxide

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dismutase). Disruption of the functioning of iron-containing proteins, present in all organs and tissues, leads to changes in a number of vital processes [2].

RESULTS AND DISCUSSION

It is known that iron is not only a component of various heme proteins, which are necessary for the normal implementation of oxidation-reduction reactions, but also a cofactor of some enzymes. Trivalent iron from food with the help of copper-dependent ferroreductase on the membrane of enterocytes under the influence of vitamin C is reduced to divalent iron and through manganese-dependent proteins-transporters of divalent metals (DMT proteins) enters the enterocyte, from where through the protein ferroportin on the basal membrane it passes into the blood, where with the help of copper-dependent ferroxidases it is oxidized to a trivalent state in order to bind with the transport protein - transferrin [2]. This complex (transferrin - iron) interacts with specific receptors on the membrane of erythroid cells of the bone marrow, then penetrates the cells, where iron is transferred to the mitochondria and included in protoporphyrin, participating in the formation of heme. Normally, transferrin is saturated with iron by about 30%. Transferrin freed from iron can participate in the transfer of iron several times. Iron is stored in a depot as part of the proteins ferritin and hemosiderin. Iron can also enter the depot during the natural destruction of erythrocytes. With a normal iron balance in the body, an equilibrium is established between the ferritin content in the plasma and the depot. The level of ferritin in the blood reflects the amount of deposited iron. An iron deficiency develops in the erythron system, which leads to a decrease in the production of erythrocytes and reticulocytes and a decrease in hemoglobin in them. Thus, ineffective erythropoiesis may develop. Then, depletion of iron reserves in the depot occurs - the content of ferritin and hemosiderin decreases. Hormonal factors play an important role in the development of IDA, especially in adolescence, since androgens stimulate erythropoiesis, absorption and utilization of iron, hemoglobin synthesis, and estrogens inhibit these processes.

The main causes of IDA in children are:

• insufficient intake of trace elements with food (alimentary iron deficiency);
• increased need of the body for iron due to the rapid growth rate of the child, excessive weight gain;
• decreased absorption of trace elements;
• iron losses from the body exceeding physiological ones.

Depending on the time of exposure, the following causes of IDA in children are distinguished [3]:

1. Antenatal:

• impaired uteroplacental circulation;
• fetoplacental bleeding;
• feto-fetal transfusion syndrome (in multiple pregnancies);
• intrauterine melena;
• prematurity;
• profound and prolonged iron deficiency in the pregnant woman.

2. Intranatal:

• fetoplacental transfusion;
• incorrect ligation of the umbilical cord;
• bleeding due to traumatic obstetric interventions, abnormal development of the placenta or umbilical vessels.

Postnatal:

• insufficient intake with food;

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• increased iron requirements with high growth rates;
• increased iron losses due to bleeding;
• impaired iron intake with malabsorption;
• impaired iron metabolism due to hormonal changes during puberty.

The development of ID in the body has a clear staged nature. There are sequentially developing stages of IDS:

• LD, characterized by a decrease in iron stores in the depot and the onset of iron deficiency erythropoiesis;
• IDA, characterized by a combination of sideropenic and anemic syndromes.

Most authors believe that laboratory research methods are of primary importance in the diagnosis of IDA, but clinical data, which are a combination of two syndromes - sideropenic and anemic, also play a certain role [4]. The following symptoms are characteristic of sideropenic syndrome:

• dystrophic changes in the skin and appendages, mucous membranes;
• perversion of taste and smell;
• muscle pain;
• muscle hypotonia;
• changes in the nervous system in the form of delayed psychomotor development and impaired cognitive functions.

CONCLUSION

If all the above points are taken into account, then in the absence of an effect from iron therapy, one should think about iron-refractory irondeficiency anemia (IRIDA), which has been recently described in the literature. IRIDA is a disease inherited in an autosomal recessive manner, its cause is the presence of various mutations in the TMPRSS6 gene, which causes an increase in the concentration of hepcidin, which inhibits the absorption of iron from the intestine and the release of iron from macrophages. IRIDA is characterized by the absence of a response to therapy with oral iron preparations, a partial response to treatment with parenteral iron preparations, a chronic course of the disease and the presence of familial cases.

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