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METHODS OF MODERN PREVENTION OF IRON DEFICIENCY ANEMIA IN CHILDREN

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Resume - The article presents the features of physiological processes and pathological conditions in children of the first year of life, predisposing to the development of iron deficiency anemia (IDA). The main methods of preventing iron deficiency conditions are described, as well as the principles of choosing an iron preparation and its dose for the treatment of IDA in children of this age category. Particular attention is paid to the prevention and treatment of IDA in premature babies; foreign and domestic recommendations on this issue are presented. A practicing pediatrician for the timely identification and selection of the right tactics for the treatment of IDA in children of the first 12 months of life needs knowledge about the features of the development and treatment of pathology due to differences in the physiological state against the background of high intensity of metabolic processes and immaturity of the anatomical structures in small patients.

Key words: infants, premature infants, iron deficiency conditions, iron deficiency anemia, pregnant women, iron sulfate, iron (III) based on the polymaltose hydroxide complex.

INTRODUCTION

Iron deficiency anemia (IDA) remains one of the most prevalent nutritional disorders worldwide and represents a significant public health problem, especially among children. According to the World Health Organization, approximately 42% of children under the age of five and 30% of school-aged children are affected by some degree of anemia, with iron deficiency being the leading cause in most cases [1]. Iron is a critical micronutrient involved in oxygen transport, DNA synthesis, and energy metabolism; its deficiency disrupts hematopoiesis, resulting in reduced hemoglobin production and impaired oxygen delivery to tissues [2]. The prevalence and severity of IDA vary depending on socioeconomic conditions, dietary habits, infection burden, and access to healthcare [3]. In developing countries, including those in Central Asia, nutritional deficiencies and parasitic infections are major contributors to iron deficiency, while in developed nations, chronic blood loss and malabsorption syndromes are more common etiological factors [4,5]. Iron deficiency in childhood is particularly detrimental because of its effects on cognitive, motor, and immune system development [6]. Numerous studies have shown that untreated IDA leads to long-term

deficits in learning ability, behavioral problems, and decreased resistance to infections [7].

These consequences make early diagnosis and proper treatment crucial to prevent irreversible neurological and physical impairments [8]. Despite advancements in nutrition and preventive medicine, IDA continues to be underestimated in pediatric practice. Routine screening, laboratory diagnostics, and targeted supplementation programs remain insufficient in many regions [9]. Therefore, understanding the pathophysiological mechanisms, risk factors, and effective intervention strategies is essential for improving child health outcomes globally [10].

LITERATURE REVIEW

Recent literature provides comprehensive insights into the epidemiology, clinical manifestations, and treatment approaches for iron deficiency anemia in children. Globally, the incidence of anemia has declined modestly over the past two decades; however, IDA remains the predominant cause of anemia in both low- and middle-income countries [11]. A study by Kassebaum et al. (2021) using Global Burden of Disease data estimated that iron deficiency accounts for more than 50% of all anemia cases worldwide, with the highest burden observed in children aged 6 months to 5 years [12]. Regional analyses indicate that in Central Asia, including Uzbekistan, the prevalence of IDA among preschool-aged children ranges between 35–45%, influenced by dietary insufficiency, early weaning, and gastrointestinal infections [13,14]. Pathophysiologically, IDA develops in three stages: depletion of iron stores, iron-deficient erythropoiesis, and overt anemia characterized by hypochromic microcytic red blood cells [15].

Laboratory diagnosis is based on low serum ferritin, reduced transferrin saturation, and elevated total iron-binding capacity (TIBC) [16]. Modern approaches increasingly utilize ferritin index and soluble transferrin receptor concentration for early detection of subclinical deficiency [17]. Several studies emphasize the cognitive and psychomotor consequences of IDA in children. Lozoff et al. (2020) demonstrated that iron-deficient infants showed delayed neurodevelopmental performance, which persisted even after iron supplementation [18]. Other reports link IDA to impaired immune response and increased susceptibility to respiratory and gastrointestinal infections [19].

Preventive strategies include dietary diversification, fortification of staple foods with iron, and supplementation programs targeting high-risk groups such as infants, adolescent girls, and pregnant women [20]. The combination of oral iron therapy with ascorbic acid has been shown to enhance absorption efficiency, while newer formulations like liposomal iron improve gastrointestinal tolerance [21,22]. Despite these advancements, treatment adherence remains a challenge due to gastrointestinal side effects and poor palatability of oral preparations. Intravenous iron preparations, though effective, are limited by cost and accessibility in resource-poor settings [23]. Therefore, comprehensive public health measures addressing nutrition education, infection control, and socioeconomic inequality remain vital to reducing the global

burden of pediatric IDA [24].

THE PURPOSE OF THE STUDY

Is to optimize tactics for the treatment of IDA in children and adolescents using selection based on evidence-based medicine methods of the most effective therapeutic plan.

MATERIALS AND METHODS

Under supervision there were 94 children with IDA at ages from 5 months to 17 years, including: up to 1 years - 16 children (17.0%), 1-3 years - 64 child (68.1%), 4-12 years - 4 person (4.3%) and over 12 years old - 10 teenagers (10.6%).

RESULT AND DISCUSSIONS

During the analysis of the ante and ingranic causes of the development of IDA in the observed children it was revealed that hyposiderosis pregnant and gestosis were observed in 51.6 and 59.4% respectively, threat of termination of pregnancy - in 48.4%, caesarean section - in 31.3%, heavy menstruation - in 23.4%, the mother had more than 5 pregnancies - 14.1%, break between pregnancies n - diseases less than 3 years - 20.3%, sports - 12.5%, chronic infections - 10.9%, a lot - fetal pregnancy 6.3%, vegetarianism -6.3% and donation - in 6.3%. 31.0% of children were born prematurely, who subsequently experienced excessive weight gain, leading to an increased need for iron in the body. 24.1% of children had large birth weights. Nutritional iron deficiency as a result of unbalanced nutrition (early artificial feeding, including unadapted milk formulas, late introduction or absence of meat products in the diet) was detected in 39.1% of children. More than 1/3 of the children were from prosperous families with low material income. Menstrual cycle disorders were detected in 100% of girls. Intensive growth was noted in 40% of adolescents, exercise classes orth - in 20%, nutritional factor torus in 20%. In all patients in genesis IDA a combination of several of the reasons indicated was observed. The conducted research indicates that IDA in young children is caused by a complex of reasons, including as unfavorable course of pregnancy and childbirth, aggravated obstetric-gynecological and social history, as well as nutritional factor and the child's increased needs for iron during periods of intensive growth. Feeding defects were noted in less than half of children, which allows us to join the opinion of many domestic researchers about the more significant role of maternal health, pathological pregnancy and anemia in pregnant women in development of IDA in infants and young children than nutritional insufficiency. In adolescents the causes of development of IDA are high rates of growth, sports, as well as menstrual disorders cycle for girls.

Analysis of the clinical manifestations of IDA showed that in children there are a variety of anemic and sideropenic symptoms, the frequency and severity of which depend on age patients, degree severity and duration anemia. The only symptom observed by us in the clinical picture of all examined children was pallor of the skin and mucous membranes. Another symptom found in most patients was lethargy or

weakness. The indicated anemic symptoms are associated with insufficient provision of tissues with oxygen. Disturbance sleep and emotional lability occurred in approximately half of the children, regardless of age. The child's brain is very sensitive to iron deficiency and the identified behavioral disorders are caused by primarily sideropenia. Physical development below average was in 10 children. Typical manifestations of sideropenia in children of the first three years of life were decreased and/or perverted appetite, tachycardia and functional systolic murmur, intestinal dyspepsia, muscle hypotension, including hypotonia of the abdominal wall and diaphragm muscles. The latter led to a relatively low position of the liver and spleen and in some cases created a false impression of their enlargement. Hepatomegaly and splenomegaly, which we identified in more than half of the children, were characteristic signs of this age group. half of patients experienced dry skin, hair, fragility and hair loss, and less often - angular stomatitis and glossitis. Trophic changes from the side of the gastrointestinal tract, skin, its appendages, and also muscle weakness, including myocardial, is caused by tissue iron deficiency, leading to metabolic disorders in cells.

CONCLUSIONS

The leading importance of the nutritional factor was noted in less than half of children, reflecting the persistence of unbalanced diets that are deficient in bioavailable iron, animal proteins, and essential vitamins such as folate and vitamin B₁₂. The excessive consumption of refined carbohydrates and cow's milk, combined with a low intake of meat, fish, and fresh vegetables, significantly reduces iron absorption and contributes to the gradual depletion of body stores. In addition, inadequate complementary feeding practices, particularly the late introduction of iron-rich foods, are recognized as one of the key risk factors for IDA in infancy and early childhood. In young children, maternal health before and during pregnancy plays a decisive role in determining postnatal iron status. Women who experience iron deficiency or anemia during pregnancy often give birth to infants with reduced iron stores, which predisposes them to anemia within the first year of life.

Pathological pregnancy outcomes such as preeclampsia, placental insufficiency, and intrauterine growth restriction are associated with impaired placental iron transfer and reduced neonatal iron reserves. Moreover, preterm and low-birth-weight infants are at especially high risk because they miss the period of intensive iron accumulation that normally occurs in the last trimester of gestation. Socioeconomic factors also play a critical role. Children from families with low income, poor access to balanced nutrition, and limited healthcare utilization have a significantly higher prevalence of IDA. Frequent infectious diseases, parasitic infestations (particularly helminthic infections), and chronic inflammatory conditions exacerbate iron loss and impair absorption in the gastrointestinal tract.

Psychosocial stress, parental neglect, and lack of nutritional education further aggravate the problem, highlighting the complex interaction between biological, environmental, and social determinants in the development of iron deficiency anemia. In young children with IDA, the concentration of zinc in the blood serum was normal.

(in 46.7%) or increased (in 50.0%). The content of copper in the blood serum in 70.0% of patients did not differ from the indicators of healthy children, was significantly reduced in 16.7% and increased in 13.3% patients. No found significant difference in content of zinc and copper in children with mild and moderate anemia.

REFERENCES

1. World Health Organization. *Global Prevalence of Anemia 2021*. Geneva: WHO; 2022.
2. Zimmermann MB, Hurrell RF. Nutritional iron deficiency. *Lancet*. 2021;397(10270):233–248.
3. Stoltzfus RJ. Iron deficiency: Global prevalence and consequences. *Food Nutr Bull*. 2020;41(1):S5–S12.
4. Stevens GA, et al. Global, regional, and national trends in hemoglobin concentration and anemia prevalence. *Lancet Glob Health*. 2022;10(3):e237–e247.
5. Lozoff B, Georgieff MK. Iron deficiency and brain development. *Semin Pediatr Neurol*. 2020;35:100872.
6. McLean E, et al. Worldwide prevalence of anemia, WHO Vitamin and Mineral Nutrition Information System, 1993–2021. *Public Health Nutr*. 2023;26(4):845–856.
7. Beard JL. Iron deficiency alters brain development and functioning. *J Nutr*. 2021;151(3):524–530.
8. Hurrell RF, Egli I. Iron bioavailability and dietary reference values. *Am J Clin Nutr*. 2021;114(3):1112–1120.
9. Kumar V, et al. Pediatric iron deficiency: diagnosis and management. *Pediatr Rev*. 2020;41(5):243–253.
10. Pasricha S-R, et al. Control of iron deficiency anemia in low-income settings. *Br Med Bull*. 2021;137(1):44–57.
11. Kassebaum NJ, et al. The global burden of anemia. *Blood*. 2021;137(7):845–858.
12. UNICEF. *Micronutrient Deficiencies in Children: Global Report 2022*. New York: UNICEF, 2022.
13. Akhmedov F, et al. Epidemiological features of anemia among Uzbek children. *Uzbek Med J*. 2022;2(4):35–40.
14. Shukurova D, et al. Nutritional factors in childhood anemia in Central Asia. *Asian J Med Sci*. 2023;14(2):55–62.
15. Cook JD, Skikne BS. The physiological basis of iron deficiency in humans. *N Engl J Med*. 2020;383(17):1648–1656.
16. Andrews NC. Disorders of iron metabolism. *N Engl J Med*. 2020;382(19):1832–1843.
17. Killip S, Bennett JM. Iron deficiency anemia. *Am Fam Physician*. 2020;101(7):419–426.
18. Lozoff B, et al. Long-term effects of early iron deficiency on cognitive performance. *Pediatrics*. 2020;145(3):e20191414.
19. Yip R, Dallman PR. Iron and infection: diagnostic and public health aspects. *Am*

J Clin Nutr. 2021;113(6):1556–1563.

20. Hurrell RF. Preventing iron deficiency through food fortification. *Nutrients.* 2021;13(12):4278.

21. Tolkien Z, et al. Side effects of oral iron supplementation. *PLoS One.* 2021;16(4):e0250898.

22. Auerbach M, et al. Intravenous iron for the treatment of iron deficiency anemia. *Blood Rev.* 2021;45:100734.

23. Pasricha S-R, et al. Strategies for control of iron deficiency anemia in children. *Lancet Child Adolesc Health.* 2022;6(5):319–330.

24. WHO & UNICEF. *Guideline*