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REVIEW ARTICLE / ПРЕГЛЕД ЛИТЕРАТУРЕ

The basis of prevention of iron deficiency anemia during childhood and adolescence

Nedeljko Radlović[1], Zoran Leković[2,3], Jelena Radlović[4], Marija Mladenović[5]

[1]Serbian Medical Society, Academy of Medical Sciences, Belgrade, Serbia;
[2]University Children's Hospital, Belgrade, Serbia;
[3]University of Belgrade, Faculty of Medicine, Belgrade, Serbia;
[4]Railways of Serbia Institute for Health Care of Workers, Belgrade, Serbia;
[5]Singidunum University, Faculty of Health, Legal, and Business Studies, Valjevo, Serbia

SUMMARY
Anemia is a common and etiologically heterogeneous health problem both during the period of growth and development and in other phases of life. It is most often caused by a deficiency of iron, primarily due to inadequate nutrition, and less often as a consequence of various diseases. Particularly risk groups for the occurrence of anemia due to iron deficiency are children in the stages of rapid growth and development, i.e., in the first years after birth and during puberty. In accordance with the fact that it is better to prevent than to treat, in this article are given basic guidelines related to the prevention of this type of anemia in children and adolescents.
Keywords: iron deficiency anemia; children; adolescents; prevention

INTRODUCTION

Anemia is often and etiologically very heterogeneous pathological condition followed by hemoglobin (Hb) level in the blood below the lower reference value for the appropriate period of life (Table 1) [1]. It is most often caused by a deficiency of iron, primarily due to inadequate nutrition, and less often due to its malabsorption or loss through acute or chronic bleeding [2–10]. In addition, high iron loss from the body occurs in diseases accompanied by transferrin and ferritin exudation, such as extensive burns, exudative gastroenteropathy, nephrotic syndrome, and extensive exfoliative dermatitis [11, 12]. As the fetus creates significant iron reserves during the last trimester intended for adequate growth and development in the first 4–6 months after birth, it is clear that premature birth or intrauterine growth restriction (IUGR) will result in its deficiency [13, 14]. Also, an important etiological factors of iron deficiency and iron deficiency anemia during infancy are fetal–maternal hemorrhage, twin-to-twin transfusion and premature umbilical cord clamping during birth [10, 15, 16].

Table 1. Lower blood Hb reference value in children and adolescents [1]

Age	Hb (g/L)
0–30 days	15.0
1–23 months	10.5
2–9 years	11.5
Boys 10–17 years	12.5
Girls 10–17 years	12.0

It is estimated that 25–33% of the human population have anemia, 50% of which are due to iron deficiency [8]. According to the World Health Organization, the latest prevalence estimates of anemia in 2016 were 41.7% in children and 32.8% in women of reproductive age, being most prevalent in low- and middle-income countries [14].

In this paper are presented the key facts related to metabolism and physiological needs in iron, as well as basic guidelines for the prevention of its deficit during a period of growth and development.

IRON METABOLISM

Iron is a microelement essential for many processes in the human body. As a constituent of Hb and myoglobin it allows the transport of oxygen, and as part of cytochrome enzymes, catalase and peroxidase oxidative-reductive reactions. In addition, it is an indispensable factor in the synthesis of deoxyribonucleic acid, myelin, serotonin, noradrenaline, dopamine and other compounds [17]. Bearing in mind these facts, it is clear that iron is essential for all cells of the body, and that as a prooxidant, both inside and outside them, except at the time of transfer from one compound to another, is never free [17, 18].

Iron metabolism is a very complex process that involves its intake, intestinal absorption, blood flow, import into cells, incorporation into appropriate functional compounds and deposition [17]. Since iron excess has a toxic effect

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Correspondence to:
Nedeljko RADLOVIĆ
Serbian Medical Society
Džordža Vašingtona 19
110000 Belgrade
Serbia
n.radlovic@beotel.net

and limited mechanisms of excretion from the body, the regulation of its homeostasis is primarily performed at the level of the mature intestinal epithelium of the small intestine, i.e., enterocytes [17]. The main role in this process is the hepatic polypeptide hormone hepcidin, which controls not only intestinal iron absorption but also its mobilization from the depot and transplacental transport [8, 17, 19, 20].

Iron absorption is performed in the proximal part of the small intestine, of which 85% in the duodenum [17]. Two main types of iron in foods, heme and non-heme, use different mechanisms of absorption [8, 17]. Heme iron, present in the meat, is transferred into the enterocyte via a specific membrane transport protein (heme carrier protein 1, HCP 1), after which it is released by proto-porphyrin IX under the action of hem-oxygenase-1 [17]. The degree of absorption of these mechanisms on iron in the state of high demands of the organism, and of the left disorders of digestion and absorption reaches 20–30%, and even up to 50% [8, 18]. The only food ingredient that compromises the absorption of heme iron is calcium [21]. Non-hem iron, present in other foods, has a significantly lower utilization rate, which in healthy, depending on the type of food and other factors, is only 1–10%, on average 4–5% [18]. Stimulating effects on intestinal absorption of non-hem iron are factors that favor its solubility (hydrochloric acid, gastric juice, citric, lactic and other organic acids) or reduction (enterocytic ferric reductase DcytB, vitamin C), and inhibitory compounds that make it insoluble (phosphates, phosphoproteins, phytates, oxalates, polyphenols, tannins, fibers, casein) or in competition with its absorption (copper, zinc, manganese, nickel, cobalt, lead, cadmium) [8, 18]. In addition, amino acids, extensive protein hydrolysates and beta-carotene have a beneficial effect on the absorption of non-hem iron, while calcium has an unfavorable effect [18]. Non-hem iron absorption is, after previous reduction, performed via a divalent metal transporter 1 (DMT1) located on the apical membrane of enterocytes [17, 19, 22]. Apart from iron, copper, zinc and other divalent metals are transmitted via DMT1, which explains the interdependence of their degree of absorption and content in the body. Iron present in ferritin of animal and vegetable foods is a separate form of absorption [23]. Also, breast milk lactoferrin-bound iron is absorbed through specific receptors on the apical membrane of enterocytes [24]. Upon passage into the enterocyte, the iron in conjunction with an as yet unidentified protein reaches its basolateral membrane where it is oxidized and via ferroportin 1 is exported and transferred to transferrin [17]. Oxidation of iron, which is a prerequisite for its exit from the cell and transfer to transferrin, is performed by hephaestin, enterocytic ferro-oxidase added to ferroportin 1 [25]. Through transferrin the iron reaches all cells of the body which via its transferrin receptors 1 uptake and transfer it to the cytoplasm via DMT1 [16, 17]. Excess of iron in enterocytes with apoferritin builds ferritin [19]. The import of iron into enterocytes and its binding to apoferritin are controlled by specific intracellular iron regulatory proteins (IRPs) [18, 19]. The same mechanism controls the homeostasis of iron in other cells of the body [26]. Consistent with its physiological role, its largest acceptors are the precursors of the red blood cell, as well as the cells that deposit it, i.e., hepatocytes and macrophages of spleen, liver and bone marrow. Given the limited transfer capacity, as well as short lifespan of enterocytes, which desquamate after 3–5 days, iron fraction which escapes the transport is lost, which represents a physiological barrier of its excess in the state of excessive oral intake [25].

As stated, the control of iron homeostasis is a very complex process whose main carriers are enterocytes and hepatocytes, or their regulatory proteins [8, 19, 27]. Intracellular IRPs play a key role in controlling the import of iron into enterocytes, and in its export hepcidin [20, 26]. The physiological inducer of IRPs is high iron content in enterocyte, and hepcidin in circulation and hepatocyte [27, 28]. By their effect IRPs interrupt the expression of DMT1 and ferri-reductase and stimulate ferritin synthesis, while hepcidin reacts with ferroportin 1 and leads to its internalization and degradation [27, 28]. By the same mechanism as in enterocyte, hepcidin regulates iron efflux from hepatocytes, macrophages and placental cells [27, 28]. The physiological significance of the activation of IRPs and hepcidin is contained in the protection of the body against excessive iron intake, while in the state of iron deficiency or hypoxia, the activity of these factors disappears [27, 28]. Sex hormones (testosterone, estrogens), growth hormone, and hypoxia-induced erythroblastic polypeptide erythroferrone have an important role in suppressing hepcidin expression [28, 29]. Being the reactant of the first stage of inflammation, hepcidin, by blocking the intestinal resorption of iron and its mobilization from the depot, is responsible for the occurrence of anemia in chronic infectious and other inflammatory diseases, as well as in some malignancies [8, 27, 30, 31, 32]. In addition, dysregulation of hepcidin is the pathogenetic basis of hereditary hemochromatosis, which in the first type is due to autosomal recessive defect in its expression, in the second as a result of insufficient stimulation of its secretion and in the third due to the absence of its effect caused by an autosomal dominant defect of ferroportin [27].

DIETARY REQUIREMENTS FOR IRON OF CHILDREN AND ADOLESCENTS

Dietary requirements for iron of children and adolescents are dependent on the rate of growth and the degree of physiological losses (Table 2) [33]. In girls with menstruation onset, the necessary dietary intake of iron is significantly higher compared to male peers. The needs in iron of infants born at term are low because within the first six months it is mainly provided from prenatally acquired stocks [33].

Table 3 shows the iron content of some of the basic foods [10, 34]. Within the standard diet, except in the first year after birth, the participation of heme and non-heme iron in meeting physiological needs is approximately equal [33]. Hem iron is present in meat, while non-hem iron is found in other foods. Due to its rich content and high

Table 2. Recommended dietary intake for iron in childhood and adolescence [33]

Age	Male	Females
0–6 months	0.27 mg*	0.27 mg*
7–12 months	11 mg	11 mg
1–3 years	7 mg	7 mg
4–8 years	10 mg	10 mg
9–13 years	8 mg	8 mg
14–18 years	11 mg	15 mg

Adequate intake*

Table 3. Iron content in certain types of food [10, 34]

Type of food	mg/100 g
Human milk	0.3–0.5
Cow's milk	0.3–0.6
Infant formula	4–12
Chicken meat	0.6–2
Turkey meat	0.8–2
Beef meat	3–3.1
Lamb meat	1.2–1.9
Pork meat	0.9–2.3
Sardines	2.2–3.6
Chicken liver	7.4
Chicken egg yolk	7.2
Beans and peas	2
Spinach	1.5–1.7
Cabbage	0.7
Potatoes	0.3
Whole-wheat flour	3.9
White wheat flour	2
Hazelnut	3
Walnut	2
Apple	0.1

utilization rate, meat is the best source of iron [35, 36]. Many other foods are abundant in iron, such as egg yolks, liver, various types of vegetables and nuts, but in a non-heme form, and are inferior to meat in this respect. Due to its low content, despite its extremely high absorption rate (about 50%), breast milk is not a rich source of iron [36]. Cow's, goat's and sheep's milk, cereals and fruits, due to their low concentration and/or scarce absorption, are a poor source of iron [10, 36].

Food grade iron is used to cover physiological losses and build up reserves, and during growth and development to incorporate into new body structures. About 65–75% of total body iron in an adult is found in erythrocytes, 10% in muscle, and rest in other tissues and depots [8]. On circulating iron accounts for only 0.1% of its total content in the body [37].

Iron from decayed and lysed erythrocytes and other cells is conserved and recycled [15, 33]. It is lost from the body in the desquamation of skin and mucous epithelium, and minimally through urine and sweat [33]. Hence, the degree of physiological iron loss is primarily proportional to body surface [33]. In addition, women in the generative period have an additional loss of iron by menstrual bleeding, and their needs are much greater than men [33]. This is also true for pregnant women, as a significant part of the iron ingested is directed towards increasing its erythrocyte mass, building the placenta and the needs of the fetus [33].

PREVENTION OF IRON DEFICIENCY IN CHILDREN AND ADOLESCENTS

Optimal nutrition is the basis of iron deficiency prevention. Exceptions are infants born preterm, with IUGR and as twins who also require iron supplementation. An important factor in the prevention of early infantile anemia is delayed cord clamping until 1–3 minutes after birth, which provides placental transfusion and iron-rich blood flow to the newborn [16, 36].

Optimal nutrition of the infant is based on breast milk and/or infant formula, as well as timely initiation and optimal intake of complementary foods. Although relatively poor in iron (0.3–0.5 mg / 100 ml), due to its high absorption rate breast milk to a term infant during the first 4–6 months covers the need for it [33, 36]. Adequate alternatives to breast milk deficiency are infant formulas [38]. Infant formulas contain 0.3–1.3 mg of elemental iron per 100 kcal [39]. The iron content in infant formulas is much higher compared to breast milk because its absorption rate is so much lower [36]. Because prenatally acquired trace element reserves are depleted during the first 4–6 months after birth, cows' milk formulas with a higher iron content (1.1–1.9 mg/100 kcal) are recommended for infants aged 6–12 months [40]. Due to the even lower utilization rate, the recommended iron content in formulas based on soy protein isolates intended for infants at that age is much higher (1.3–2.5 mg/100 kcal) [40]. According to current recommendations, complementary foods are introduced to a child aged 4–6 months [41, 42]. There are several reasons for this, and one of them is to cover the need for iron. Therefore, infants who only consume breast milk at 4–6 months of age are advised daily intake of appropriate iron preparation at a dose of 1 mg/kg [38]. The same is true for infants in the second half who are not eating enough meat and vegetables. However, children born pre-term or with IUGR, as their prenatally acquired iron reserves are extremely scarce, drug prophylaxis of sideropenic anemia begins at the end of the first month and continues until the end of the first year or until the optimum intake of non-milk food, especially meat [38]. The recommended daily dose of elemental iron to this infant on the breast milk diet, depending on the degree of prematurity and IUGR, is 2–3 mg / kg, and if fed with the appropriate cow's milk formula 1 mg/kg [36, 38, 43]. The iron content of standard cow's milk formulas intended to feed preterm infants by the age of 40 postconception varies from 1.5 to 2.5 mg/100 kcal [44]. At present, there is no clearly defined attitude regarding the introduction of complementary feeding in preterm infants. According to the experience of different groups of authors, its initiation to these children is generally delayed in proportion to the degree of prematurity, i.e., for as many as were born earlier [45, 46]. Ordinarily cow's milk and milk of other mammals, for a number of reasons, including poor iron content (about 0.5 mg/L) and low utilization rate

(5–10%), are not advised to the child in the first year [38]. In order to identify and correct sideropenic anemia in a timely manner, according to the American Academy of Pediatrics, routine blood counts should be performed for every 9–12-month-old child, and preterm, malnourished or otherwise suspected of iron deficiency and earlier [38].

Consistent with growth rate, stage of development and nutritional requirements, feeding the child after the first year is based on non-milk foods, including foods with rich and highly utilized iron content, such as meat, legumes and other types of vegetables [38]. However, in order to meet the need for calcium as well as other qualities, milk and dairy products remain a mandatory part of the child's menu. The daily amount of milk should be limited at the age of 1–3 years to 500 ml, from four to eight years to 600 ml and from nine to 18 years to 700 ml [36, 47]. This applies primarily to cows, goats and sheep's milk, which are poor sources of iron. Therefore, children aged 12–36 months instead of plain milk recommended follow-up formula, which, among other benefits, contains a significant amount of iron (1–3 mg/100 kcal) [48]. After the first year of life iron deficiency most commonly affects children aged 1–5 years and adolescents, especially girls starting menstruation [36, 49]. The iron deficiency of healthy children of these age groups is based on excessive intake of milk, dairy products and cereals at the expense of meat and vegetables [8, 38]. In line with the above facts, children and adolescents who do not eat at least two or three iron-rich foods every day do not provide adequate iron needs and require appropriate supplementation [50].

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Основа превенције анемије узроковане недостатком гвожђа током детињства и адолесценције

Недељко Радловић[1], Зоран Лековић[2,3], Јелена Радловић[4], Марија Младеновић[5]

[1]Српско лекарско друштво, Академија медицинских наука, Београд, Србија;
[2]Универзитетска дечја клиника, Београд, Србија;
[3]Универзитет у Београду, Медицински факултет, Београд, Србија;
[4]Завод за здравствену заштиту радника „Железнице Србије", Београд, Србија;
[5]Универзитет „Сингидунум", Факултет здравствених, правних и пословних студија, Ваљево, Србија

САЖЕТАК

Анемија је чест и етиолошки хетероген здравствени проблем како у периоду раста и развоја, тако и у осталим фазама живота. Најчешће је узрокована недостатком гвожђа, пре свега због неадекватне исхране, а ређе као последица различитих болести. Посебно ризичне групе за појаву анемије услед недостатка гвожђа су деца у фазама брзог раста и развоја, тј. у првим годинама после рођења и током пубертета. У складу са чињеницом да је боље спречити него лечити, у овом чланку су дате основне смернице везане за превенцију ове врсте анемије код деце и адолесцената.

Кључне речи: анемија због недостатка гвожђа; деца; адолесценти; превенција