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Iron Deficiency Anemia in Young Children: Prevalence, Early Diagnosis and Prevention

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Abstract Background. Iron deficiency is the most common micronutrient deficiency in the world and the leading cause of anemia in early childhood. In 2019, 40% of children aged 6–59 months worldwide — about 269 million — were anemic.

Aim. To summarize current evidence on the prevalence, early diagnosis and prevention of iron deficiency anemia (IDA) in children under five years of age, with particular reference to Uzbekistan.

Methods. A narrative review of WHO guidelines, Cochrane systematic reviews, national survey data and clinical studies published in 2006–2026 (PubMed, Scopus, Google Scholar).

Results. According to the Uzbekistan Nutrition Survey 2017, anemia affected about 15% of children aged 6–59 months and one in four children under two years; about three-quarters of anemia cases were due to iron deficiency, and more than half of under-fives were iron deficient. In 2024 WHO lowered the hemoglobin cut-off for children aged 6–23 months to 105 g/L. Serum ferritin < 12 µg/L (< 30 µg/L with inflammation) remains the WHO criterion of iron deficiency, but a physiologically based threshold of about 22 µg/L suggests that current cut-offs underestimate the problem. Delayed cord clamping, exclusive breastfeeding followed by iron-rich complementary feeding, targeted iron supplementation and food fortification are the most effective preventive measures.

Conclusion. Iron deficiency without anemia is common in young children and may impair neurodevelopment. Screening should therefore combine hemoglobin with ferritin and an inflammation marker, and prevention should start before birth and continue through the first two years of life.

Keywords: iron deficiency anemia; children under five; ferritin; hemoglobin thresholds; prevention; delayed cord clamping; Uzbekistan.

Introduction

Iron is essential for hemoglobin synthesis, oxygen transport and energy metabolism, as well as for brain development, including myelination, dendritogenesis and dopamine neurotransmission [1, 2]. Requirements per kilogram of body weight are highest in the first two years of life, when growth is fastest. About 80% of the newborn's iron is accumulated during the third trimester of pregnancy. A healthy term infant therefore has enough iron for roughly the first four months, whereas preterm infants and those born to anemic or diabetic mothers start life with reduced stores [3].

Anemia remains a major global health problem. In 2019, 40% of children aged 6–59 months were anemic, compared with 48% in 2000, and progress is too slow to meet the World Health Assembly targets [4]. In the same year, anemia caused 50 million years of healthy life lost to disability, with dietary iron deficiency as the leading cause [5].

Anemia is a late sign of iron deficiency. Tissue iron depletion, including in the developing brain, begins well before hemoglobin falls, and its neurodevelopmental effects may not be fully reversible

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with later treatment [1, 3]. Early detection of iron deficiency and its prevention are therefore more important than treatment of established anemia.

The aim of this review is to summarize current evidence on the prevalence, early diagnosis and prevention of IDA in children under five years of age, with particular reference to Uzbekistan.

Materials and methods

A narrative review of literature published between 2006 and 2026 was carried out in PubMed/MEDLINE, Scopus, Google Scholar, the Cochrane Library and the WHO Institutional Repository (IRIS). The search terms were “iron deficiency”, “iron deficiency anemia”, “infants”, “young children”, “ferritin”, “hemoglobin threshold”, “cord clamping”, “iron supplementation” and “Uzbekistan”, in various combinations.

Priority was given to WHO guidelines, systematic reviews and meta-analyses, randomized controlled trials, large longitudinal cohorts and nationally representative surveys covering children aged 0–59 months. National data were taken from the Uzbekistan Nutrition Survey 2017 and the WHO Global Health Observatory.

Prevalence

Globally, 40% (95% UI 36–44%) of children aged 6–59 months were anemic in 2019, which corresponds to about 269 million children [4, 5]. The burden is concentrated in the WHO African and South-East Asian Regions, which together account for about 103 million anemic children, and in 11 countries childhood anemia exceeds 70% [4, 5]. Iron deficiency is found in roughly a quarter to a half of anemic children; its share is lower in populations with a heavy burden of infection [4].

In Uzbekistan, the most detailed data come from the nationally representative Uzbekistan Nutrition Survey (UNS) 2017, conducted by the Ministry of Health with UNICEF in 3,874 households across all 14 regions [6]. Anemia was found in about 15% of children aged 6–59 months and in one in four children under two years of age; prevalence peaked at 6–11 months and declined with age [6]. About three-quarters of anemia cases were caused by iron deficiency, and more than half of all children under five were iron deficient — most of them without anemia [6]. The WHO modelled estimate for 2019 is higher, at 21.9% [7].

The survey also documented dietary risk factors: only 50.7% of infants under six months were exclusively breastfed, and only 30.3% of children aged 6–23 months reached minimum dietary diversity [6]. In Uzbekistan, therefore, the main problem is not overt anemia but a large hidden pool of iron deficiency in the first two years of life.

Etiology, risk factors and consequences

Iron deficiency in young children results from a mismatch between rapidly growing requirements and limited intake or stores. The main risk factors are:

- **Perinatal:** prematurity, low birth weight, maternal anemia, maternal diabetes or hypertension with intrauterine growth restriction, and early umbilical cord clamping [3].
- **Dietary:** exclusive breastfeeding beyond six months without iron-rich complementary foods (human milk is low in iron), introduction of whole cow’s milk before 12 months, excessive milk intake in toddlers, and low dietary diversity with predominantly plant-based (non-heme) iron sources [3].
- **Increased losses or impaired absorption:** intestinal helminths, coeliac disease, possibly Helicobacter pylori infection, and chronic inflammation [8].
- **Social:** low family income and low maternal education, which limit access to iron-rich foods and health care [3].

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Clinically, mild iron deficiency is usually silent. As it progresses, pallor, fatigue, irritability, poor appetite and pica appear, and severe anemia may lead to tachycardia and heart failure.

The most serious consequences concern the brain. Iron deficiency during brain development disturbs neurometabolism, myelination and dopamine function, especially in the hippocampus and striatum [1, 2]. In a Costa Rican cohort followed from infancy to 19 years, children with chronic iron deficiency in infancy scored 8–9 points lower on cognitive tests than peers with good iron status, despite iron treatment; in low-income families the gap widened over time [9]. At 19 years, the same cohort showed persistent deficits in executive function and recognition memory [10].

Early diagnosis

Iron deficiency develops in three successive stages, and only the last one is detected by hemoglobin:

1. **Iron depletion** — storage iron falls (low serum ferritin), while hemoglobin and red cell indices remain normal.

2. **Iron-deficient erythropoiesis** — transferrin saturation and reticulocyte hemoglobin content (CHr) decrease, soluble transferrin receptor and zinc protoporphyrin increase; hemoglobin is still normal.

3. **Iron deficiency anemia** — hemoglobin falls below the age-specific cut-off, with microcytosis (low MCV), hypochromia (low MCH) and increased red cell distribution width (RDW).

In 2024 WHO revised its hemoglobin thresholds and lowered the cut-off for children aged 6–23 months from 110 to 105 g/L [11]. The change is based on a pooled analysis of healthy reference populations, in which the 5th centile of hemoglobin was 104.4 g/L at 6–23 months and 110.2 g/L at 24–59 months [12]. A 2026 pooled analysis from eight countries found that these cut-offs lie in the middle-to-upper range of values that discriminate functional and developmental outcomes [13].

Serum ferritin is the recommended marker of iron stores. WHO defines iron deficiency in children under five as ferritin < 12 µg/L, or < 30 µg/L in the presence of infection or inflammation, and advises measuring C-reactive protein (CRP) and α_1-acid glycoprotein (AGP) at the same time [8]. However, a 2025 multinational study derived a physiologically based ferritin threshold of 22.1 µg/L (95% CI 20.8–23.4) for children aged 6–59 months, implying that the WHO cut-off substantially underestimates iron deficiency [14].

Indicator	6–23 months	24–59 months
Anemia, hemoglobin (g/L)	< 105	< 110
Mild anemia (g/L)	95–104	100–109
Moderate anemia (g/L)	70–94	70–99
Severe anemia (g/L)	< 70	< 70
Iron deficiency, ferritin, no inflammation (µg/L)	< 12	< 12
Iron deficiency, ferritin, with inflammation (µg/L)	< 30	< 30

Table 1. WHO diagnostic thresholds for anemia and iron deficiency in children under five [8, 11].

In practice, the American Academy of Pediatrics recommends universal hemoglobin screening at about 12 months of age together with a risk-factor assessment, supplemented by ferritin with CRP or by CHr [3]. In Central Asia, where beta-thalassemia carriage is common, microcytosis with a normal or raised red cell count (Mentzer index MCV/RBC < 13) should prompt hemoglobin electrophoresis rather than empirical iron therapy.

Prevention and management

Prevention should begin at birth and continue through the first two years of life, combining individual clinical measures with population-level programmes (Table 2).

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Age	Measure	Details
Birth	Delayed umbilical cord clamping	Clamp no earlier than 1 minute after birth in term infants [15, 16]
0–6 months	Exclusive breastfeeding	Breastfed term infants: 1 mg/kg/day of iron from 4 months until iron-rich foods are introduced; breastfed preterm infants: 2 mg/kg/day from 1 to 12 months [3]
6–23 months	Iron-rich complementary feeding	Required intake 11 mg/day at 6–12 months; no whole cow's milk before 12 months [3]
6–23 months	Point-of-use fortification	Iron-containing micronutrient powders where anemia prevalence is ≥ 20% [17]
6–59 months	Daily iron supplementation	10–12.5 mg (6–23 months) or 30 mg (24–59 months) for 3 consecutive months a year where anemia prevalence is ≥ 40% [18]
1–3 years	Balanced diet, limited milk	Required intake 7 mg/day [3]
All ages	Mass fortification	Iron-fortified wheat flour [19]

Table 2. Main measures for preventing iron deficiency in children under five.

Delayed cord clamping. In a Cochrane review, term infants with early cord clamping were more than twice as likely to be iron deficient at 3–6 months (RR 2.65; 95% CI 1.04–6.73; 5 trials, 1,152 infants) [15]. In a Swedish randomized trial of 400 term infants, clamping after at least 180 seconds raised ferritin at 4 months by 45% [16]. The only trade-off is a slightly greater need for phototherapy for jaundice [15].

Feeding. After six months of exclusive breastfeeding, complementary foods should include meat, liver, legumes or iron-fortified cereals, ideally with a source of vitamin C. Giving tea to young children with meals, a common practice in Central Asia, should be discouraged because polyphenols inhibit non-heme iron absorption.

Supplementation and fortification. WHO recommends universal daily iron supplementation only where anemia affects at least 40% of children [18], whereas micronutrient powders are recommended from a prevalence of 20% [17]. With about one in four children under two anemic, Uzbekistan falls into the second category, so micronutrient powders and targeted supplementation of high-risk infants are more appropriate than universal supplementation. A national wheat flour fortification programme has operated since 2005, but its evaluation showed that greater impact requires wider coverage of adequately fortified flour and a more bioavailable iron compound [19].

Treatment. Suspected IDA is treated with oral ferrous sulfate at 3 mg/kg/day of elemental iron, given between meals. A hemoglobin rise of at least 10 g/L after 4 weeks confirms the diagnosis; treatment then continues for two more months to replenish stores, followed by a repeat blood count [20]. In a randomized trial of 80 children aged 9–48 months, once-daily ferrous sulfate for 12 weeks raised mean hemoglobin from 79 to 119 g/L, 10 g/L more than iron polysaccharide complex [21]. Lack of response calls for checking adherence and excluding ongoing blood loss, malabsorption and thalassemia trait. Iron preparations must be kept out of children's reach because accidental overdose is dangerous.

Conclusion

Iron deficiency remains the leading cause of anemia in children under five. In Uzbekistan, more than half of young children are iron deficient, although only about 15% are anemic. Because anemia is

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a late marker and early iron deficiency may permanently affect neurodevelopment, diagnosis should not rely on hemoglobin alone. Ferritin, together with CRP and ideally AGP, should be measured in infants with risk factors and in every anemic child, and hemoglobin should be interpreted using the 2024 WHO cut-offs.

The most effective preventive package combines delayed cord clamping, exclusive breastfeeding to six months followed by iron-rich complementary foods, avoidance of cow's milk and tea in infancy, iron supplementation of preterm and low-birth-weight infants, home fortification with micronutrient powders and better-targeted flour fortification. New national studies are needed to update prevalence data collected in 2017 and to validate ferritin thresholds in Uzbek children.

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