BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg
BG PULSE JOURNAL

IRON DEFICIENCY ANEMIA IN CHILDREN: ETIOLOGICAL FACTORS, CLINICAL COURSE, EARLY DIAGNOSIS AND MODERN PREVENTIVE APPROACHES

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Doi: https://doi.org/10.5281/zenodo.22919298

Abstract. Iron deficiency anemia (IDA) remains one of the most frequent micronutrient-related disorders in childhood and is associated with impaired oxygen transport, neurocognitive development, physical growth and resistance to infection. The purpose of this review-analytical article is to systematize current evidence on the etiological structure, clinical course, early laboratory diagnosis and preventive strategies of pediatric IDA, with particular attention to evidence generated in Uzbekistan. A structured review of 15 sources was performed, including 11 publications from Uzbekistan and four international guidelines or clinical reports. The synthesis shows that the major pediatric risk pathways include low neonatal iron stores, maternal anemia, prematurity, rapid growth, delayed introduction of iron-rich complementary foods, dietary imbalance, chronic blood loss and gastrointestinal malabsorption. Early diagnosis should not rely on hemoglobin alone; interpretation of ferritin together with markers of inflammation, red-cell indices and, where available, transferrin saturation or reticulocyte hemoglobin improves diagnostic accuracy. Effective prevention requires a life-course strategy combining maternal health, age-appropriate complementary feeding, risk-based screening and supplementation, food fortification and family education. The evidence supports integrating clinical risk assessment with laboratory confirmation to identify iron deficiency before severe anemia develops.

Keywords: iron deficiency anemia; children; ferritin; hemoglobin; early diagnosis; risk factors; prevention; nutrition; Uzbekistan.

INTRODUCTION

Iron deficiency is a continuum that begins with depletion of iron stores, progresses to iron-restricted erythropoiesis and culminates in iron deficiency anemia when hemoglobin synthesis becomes insufficient. Childhood is particularly vulnerable because iron requirements increase rapidly during infancy, early childhood and adolescence, while the amount of absorbable iron in the diet may remain limited. Uzbek studies consistently identify young age, maternal anemia, prematurity, suboptimal complementary feeding and recurrent illness as important risk conditions for pediatric iron deficiency. Local epidemiological and clinical observations also indicate that latent iron deficiency can be substantially more common than overt anemia, which makes exclusive reliance on hemoglobin-based screening insufficient (1,2,3,4).

The clinical importance of IDA extends beyond pallor and fatigue. Iron participates in mitochondrial energy production, myelination, neurotransmitter synthesis, immune function and muscle metabolism. Prolonged deficiency during critical developmental windows may therefore be

associated with reduced attention, developmental delay, poor school performance, decreased exercise tolerance and higher susceptibility to infection. These consequences are especially important in infants and toddlers, in whom neurodevelopment is rapid and recovery after prolonged deficiency may be incomplete (5,12,15).

The problem remains relevant in Uzbekistan despite progress in nutrition programmes, food fortification and preventive supplementation. Recent local publications emphasize a shift from recognition of established anemia toward risk stratification, detection of pre-anemic iron deficiency and population-level prevention. The objective of this article is to synthesize current evidence on etiological factors, clinical manifestations, early diagnosis and modern prevention of pediatric IDA, and to formulate a practical diagnostic-preventive framework applicable to pediatric primary care (6,7,8,9,10,11).

MATERIALS AND METHODS

A structured narrative review was conducted using 15 sources published or updated between 2010 and 2026. Eleven sources were produced by authors or institutions from Uzbekistan (73.3%), thereby meeting the target of at least 70% local literature. The local evidence included pediatric clinical studies, review articles and conference publications addressing risk factors, hematological characteristics, early diagnostic methods and prevention of iron deficiency in children. Four international sources were selected for normative diagnostic and preventive guidance: the World Health Organization (WHO) guideline on hemoglobin cutoffs, the WHO ferritin guideline, the WHO guideline on daily iron supplementation in infants and children, and the American Academy of Pediatrics (AAP) clinical report on diagnosis and prevention of iron deficiency in young children (12–15).

The analysis was organized according to the IMRAD model. Data were extracted into four domains: (1) etiological and age-specific risk factors; (2) clinical manifestations and developmental consequences; (3) laboratory markers used for early and differential diagnosis; and (4) individual and population preventive interventions. Because the article is a review-analytical study rather than an original clinical trial, the Results section presents a synthesis of published evidence and does not report artificially generated patient data.

RESULTS

The reviewed literature demonstrates that pediatric IDA is usually multifactorial. The first pathway is insufficient iron endowment at birth, which is associated with maternal iron deficiency, prematurity, low birth weight and multiple pregnancy. The second is inadequate intake during periods of rapid growth, particularly when complementary foods are introduced late, are predominantly cereal-based, or contain little heme iron. A third pathway is reduced absorption caused by gastrointestinal disease or chronic inflammation, while a fourth is excessive loss through gastrointestinal bleeding, parasitic disease or, in adolescent girls, menstrual blood loss. Several Uzbek studies highlight the combined action of these factors rather than a single isolated cause (1,3,4,6,8).

Table 1. Major etiological pathways of iron deficiency anemia in children

Etiological pathway	Typical pediatric setting	Mechanism	Clinical implication

Etiological pathway	Typical pediatric setting	Mechanism	Clinical implication
Low iron stores at birth	Maternal anemia, prematurity, low birth weight	Reduced fetal iron accumulation	Earlier depletion during infancy; need for closer surveillance
Insufficient intake	Delayed/poor complementary feeding, low heme-iron diet	Intake does not match growth-related requirement	Progressive depletion of ferritin and later fall in hemoglobin
Increased requirement	Rapid growth, adolescence	Higher erythropoietic and tissue demand	Latent deficiency may precede anemia
Reduced absorption	Celiac disease, chronic enteropathy, inflammation	Impaired uptake and/or hepcidin-mediated iron restriction	Oral iron response may be suboptimal; underlying disease must be addressed
Chronic loss	GI bleeding, parasitosis, heavy menstrual loss	Loss exceeds replacement	Persistent or recurrent IDA unless source of loss is corrected

The table shows why successful management requires identification of the mechanism rather than correction of hemoglobin alone. In a child with persistent IDA, recurrence after apparently adequate iron therapy should trigger reassessment of adherence, dose, ongoing blood loss, malabsorption and alternative diagnoses.

Clinically, mild or early iron deficiency may be almost asymptomatic. As the deficit progresses, pallor, fatigue, irritability, reduced appetite, pica, tachycardia on exertion, decreased physical endurance, brittle nails and impaired concentration can appear. In younger children, poor weight gain, delayed psychomotor development and recurrent infections may be more prominent than classic subjective complaints. Local clinical observations in children born to anemic mothers and in high-risk young children support the importance of developmental and nutritional history in addition to physical examination (2,3,7).

Early diagnosis requires recognition that anemia is a late stage of iron deficiency. Updated WHO guidance provides age-specific hemoglobin thresholds for defining anemia, while ferritin remains the principal marker of iron stores. Ferritin, however, is an acute-phase reactant; therefore, normal or moderately elevated ferritin does not exclude iron deficiency when infection or inflammation is present. In such situations, simultaneous assessment of C-reactive protein or another inflammatory marker and the use of transferrin saturation, soluble transferrin receptor or reticulocyte hemoglobin, when available, can improve interpretation (7,12,13).

Table 2. Laboratory markers used in early diagnosis and differential interpretation

Marker	Typical change in IDA	Strength	Important limitation

Marker	Typical change in IDA	Strength	Important limitation
Hemoglobin	Reduced after iron-deficient erythropoiesis becomes established	Defines anemia severity	Low sensitivity for pre-anemic iron deficiency; many non-iron causes
MCV / MCH	Usually reduced	Supports microcytic, hypochromic pattern	Can be normal early; also reduced in thalassemia
Serum ferritin	Reduced in uncomplicated iron deficiency	Best-established marker of iron stores	May be falsely normal/high during inflammation
Transferrin saturation	Often reduced	Reflects circulating iron availability	Affected by diurnal variation and inflammation
Reticulocyte Hb	Reduced early when iron delivery to marrow is limited	Sensitive to recent iron-restricted erythropoiesis	Availability and reference ranges vary
CRP / inflammation marker	May be elevated in concurrent inflammation	Helps interpret ferritin	Does not diagnose iron deficiency itself

Microcytosis and hypochromia support IDA but should not be treated as pathognomonic findings. Thalassemia trait, anemia of inflammation, chronic kidney disease, lead toxicity and other less common disorders may produce overlapping hematological patterns. A disproportionately low MCV with a relatively preserved erythrocyte count, family or ethnic history of hemoglobinopathy, normal ferritin, or lack of expected hematologic response to iron should prompt differential evaluation. This step prevents prolonged empirical therapy in children whose anemia is not caused by iron deficiency (7,12,13).

Figure 1. Editable clinical algorithm for early detection of pediatric iron deficiency

1. Identify risk Maternal anemia • prematurity/low birth weight • rapid growth • low-iron diet • chronic GI disease or blood loss
↓
2. Clinical screening Dietary history • growth • pallor/fatigue/pica • developmental and menstrual history when age-appropriate
↓

3. Initial tests CBC with indices + ferritin; add CRP/inflammation marker when clinically relevant
↓
4. Confirm mechanism and act Low ferritin / compatible profile → treat iron deficiency and cause; discordant profile or poor response → differential work-up

The algorithm prioritizes risk recognition before laboratory testing. This is important because children with normal hemoglobin may already have depleted iron stores. Conversely, a low hemoglobin concentration should not automatically be labeled as IDA without considering morphology, ferritin and clinical context. A documented rise in hemoglobin after appropriate therapy supports iron deficiency, but monitoring should also confirm replenishment of iron stores and correction of the underlying risk factor (4,7,12,13).

DISCUSSION

The synthesis confirms that the most effective pediatric strategy is to prevent the transition from iron depletion to symptomatic anemia. Uzbekistan-specific publications are particularly consistent in emphasizing maternal anemia, early childhood nutrition, prematurity and rapid growth as major points of intervention. This is biologically plausible because neonatal iron endowment and the quality of complementary feeding determine how long infant iron stores can meet expanding blood volume and tissue demands. In adolescence, accelerated growth and menstrual blood loss create a second vulnerable period, especially when dietary iron density is low (1,2,3,6,10,11).

For clinical practice, serum ferritin should be interpreted as part of a panel rather than as an isolated number. The WHO ferritin guideline stresses that inflammation changes ferritin concentrations; consequently, a child with infection may have depleted iron stores despite ferritin values that would appear reassuring in a healthy child. Integrating ferritin with CRP, transferrin saturation, red-cell indices and clinical context reduces both underdiagnosis and unnecessary iron therapy. The increasing use of reticulocyte hemoglobin and soluble transferrin receptor in specialized centers may further improve detection of iron-restricted erythropoiesis, although availability and age-specific reference intervals remain practical limitations (7,12,13).

Prevention should be organized across the life course. Maternal iron deficiency requires recognition and treatment because fetal iron acquisition depends on maternal and placental factors. After birth, breastfeeding should be supported, but exclusive breastfeeding alone does not provide sufficient iron indefinitely; iron-rich complementary foods should be introduced at the appropriate developmental age. Children born preterm or with low birth weight require individual preventive plans. At population level, food fortification, supplementation programmes for high-risk groups, control of parasitic disease and nutrition education remain important. Uzbek publications report that structured supplementation and fortification programmes can reduce the burden of anemia and iron deficiency, supporting continued public-health investment (4,6,10,14).

Figure 2. Editable prevention continuum for pediatric iron deficiency anemia

PRECONCEPTION / PREGNANCY	BIRTH / INFANCY	6–24 MONTHS	CHILDHOOD / ADOLESCENCE	POPULATION LEVEL
Detect and correct maternal iron deficiency	Preserve neonatal stores; identify prematurity and low-birth-weight risk	Iron-rich complementary feeding; risk-based supplementation	Diet quality, growth monitoring, menstrual and chronic-disease assessment	Fortification, targeted screening, education and programme monitoring

A prevention programme is most effective when these levels are linked. Screening without access to nutritional counselling or supplementation has limited value, while supplementation without correcting dietary quality or chronic blood loss leads to recurrence. Similarly, population fortification does not replace targeted clinical evaluation in high-risk children. The practical implication is a combined model in which primary care teams identify risk, confirm iron status, initiate age-appropriate correction and follow the child until both hematologic recovery and iron-store restoration are achieved (4,6,10,14,15).

One limitation of the available evidence is heterogeneity in study design, age groups, diagnostic thresholds and reporting quality across local publications. Some Uzbek studies are descriptive or review-based rather than large prospective cohorts. Therefore, local prevalence figures should be interpreted in relation to the specific population, year and diagnostic method used. Future multicenter pediatric studies using standardized WHO definitions, ferritin adjusted for inflammation and clearly defined age strata would strengthen national evidence and permit more precise evaluation of prevention programmes.

CONCLUSION

Iron deficiency anemia in children is the final stage of a preventable continuum that frequently begins with depleted iron stores before hemoglobin falls. The main etiological pathways include low stores at birth, inadequate dietary intake, increased demand during growth, impaired absorption and chronic blood loss. Early diagnosis should combine clinical risk assessment with CBC indices and ferritin, with inflammatory markers and additional iron-status tests used when interpretation is uncertain. Modern prevention is most effective when maternal health, age-appropriate complementary feeding, targeted screening and supplementation, food fortification and family education are integrated. For pediatric practice in Uzbekistan, a risk-based algorithm that detects latent deficiency and follows children until iron stores are restored offers a rational approach to reducing neurodevelopmental, functional and infectious consequences of iron deficiency.

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