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Iron Deficiency Anemia in Children and Adolescents: Epidemiology, Risk Factors, Clinical Manifestations, Diagnosis, Treatment, and Prevention

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Abstract **Review Article**

Iron deficiency anemia (IDA) is the most common nutritional deficiency and the leading cause of anemia worldwide, affecting infants, children, and adolescents disproportionately. IDA remains a significant public health concern because of its high prevalence and its potential impact on growth, neurodevelopment, cognitive function, physical performance, and quality of life. The etiology of pediatric IDA is multifactorial and includes inadequate dietary iron intake, increased iron requirements during periods of rapid growth, chronic blood loss, and impaired intestinal iron absorption. Clinical manifestations vary according to age and disease severity and may range from asymptomatic iron deficiency to fatigue, pallor, impaired cognitive performance, behavioral disturbances, and reduced physical capacity. Diagnosis relies on clinical assessment and laboratory investigations, including complete blood count and iron studies, with serum ferritin serving as the most sensitive indicator of iron deficiency. Oral iron replacement therapy remains the first-line treatment for most children, while intravenous iron is reserved for selected cases. Prevention strategies include optimization of maternal iron status during pregnancy, delayed umbilical cord clamping, appropriate infant feeding practices, iron supplementation for high-risk groups, and routine screening of susceptible populations. Early recognition, timely intervention, and effective preventive measures are essential to reduce the burden of IDA and its long-term consequences in pediatric populations.

Keywords: Iron deficiency anemia; Iron deficiency; Children; Adolescents; Diagnosis; Treatment; Prevention.

INTRODUCTION

Iron deficiency anemia (IDA) is a common form of anemia resulting from insufficient iron availability for erythropoiesis, leading to impaired hemoglobin synthesis [1].

Micronutrient deficiencies are important determinants of child health and contribute significantly to the burden of disease worldwide. Infants and young children are particularly vulnerable because of their rapid growth and development, during which nutritional deficiencies may have long-lasting or even irreversible consequences. Iron deficiency (ID) is one of the most prevalent nutritional deficiencies globally, primarily affecting pregnant women, fetuses, infants, and children. It is also the leading cause of anemia worldwide [2].

Iron deficiency and iron deficiency anemia during early childhood may adversely affect cognitive, behavioral, and physical development, with some effects persisting despite later correction of the deficiency. Among school-aged children, iron deficiency has been associated with impaired cognitive performance, reduced academic achievement, and diminished physical capacity [3].

Several risk factors contribute to the development of IDA in pediatric patients, including increased iron requirements during periods of rapid growth such as infancy, childhood, and adolescence, particularly among adolescent females. Other contributing factors include inadequate dietary iron intake, chronic blood loss, and impaired intestinal iron absorption [2,4].

This review aims to summarize the current evidence regarding epidemiology, risk factors, clinical manifestations, diagnosis, treatment, and prevention of iron deficiency anemia in children and adolescents.

PREVALENCE

Iron deficiency anemia (IDA) is the most common nutritional deficiency disorder worldwide and remains a major public health concern, particularly in developing countries. It is estimated that approximately 30% of the global population is affected by IDA, with the majority of cases occurring in low- and middle-income countries [21]. In the United States, about 9% of children aged 12 to 36 months are iron deficient, and nearly 30% of these children subsequently develop IDA [21]. Globally, the burden of IDA varies substantially according to geographic region and socioeconomic status. The reported prevalence in industrialized countries is approximately 20.1% among children aged 0 to 4 years and 5.9% among those aged 5 to 14 years, whereas corresponding rates in developing countries reach 39% and 48.1%, respectively [22]. In Western countries, two peak periods of increased prevalence have been identified during childhood and adolescence. The first peak occurs between 1 and 3 years of age, with prevalence rates ranging from 2.3% to 15%, while the second occurs during adolescence, affecting 3.5% to 13% of males and 11% to 33% of females [5].

The highest prevalence rates are observed among infants, young children, adolescent girls, and populations living in resource-limited settings, reflecting increased iron requirements, inadequate dietary intake, and socioeconomic disparities [23].

Causes and Risk Factors of Iron Deficiency Anemia

Iron deficiency anemia (IDA) develops when iron availability is insufficient to meet the body's physiological requirements [1]. According to the reviewed literature, the principal causes of iron deficiency in children include inadequate dietary iron intake, increased iron requirements during periods of rapid growth, chronic blood loss, and impaired intestinal iron absorption [6,7].

Several studies consistently identified premature infants, young children, and adolescent females as the populations at greatest risk of developing IDA because their iron requirements often exceed dietary iron intake [4,6]. Premature infants are particularly vulnerable because the majority of fetal iron stores are accumulated during the third trimester of pregnancy. Likewise, rapid growth during infancy and adolescence increases iron demands, placing these groups at elevated risk for iron deficiency. Additional risk factors include prolonged breastfeeding without adequate iron supplementation beyond four months of life [9], the use of low-iron infant formula, early introduction of cow's milk before 12 months of age, excessive cow's milk consumption during early childhood, and diets low in bioavailable iron, such as those with limited red meat intake or adherence to vegetarian or vegan dietary patterns [4].

Chronic or recurrent blood loss is another important contributor to iron deficiency. Common pediatric causes include gastrointestinal bleeding associated with inflammatory bowel disease [4,8], intestinal parasitic infections, and excessive menstrual blood loss in adolescent girls [4]. Furthermore, conditions that impair intestinal iron absorption, such as celiac disease [5,10], atrophic gastritis, gastric surgery, and reduced gastric acidity resulting from the use of acid-suppressive medications such as (antacids, H2 blockers, proton-pump inhibitors) [4,10], may predispose children to iron deficiency despite adequate dietary iron intake [4].

The reviewed studies also highlighted the role of dietary inhibitors of iron absorption. Phytates found in cereals and legumes, polyphenols present in tea and coffee, oxalates contained in certain vegetables, and calcium-rich foods can reduce the bioavailability of non-heme iron. When consumed frequently or alongside iron-rich meals, these dietary factors may contribute to iron deficiency and increase the risk of developing IDA in susceptible pediatric populations [4].

Clinical manifestations

Clinical manifestations of iron deficiency and iron deficiency anemia vary according to the severity and duration of iron depletion and the age of the child [2,6]. Iron deficiency is often symptomatic and may or may not be accompanied by anemia [11]. Mild iron deficiency may be clinically silent, whereas progressive iron deficiency anemia can may cause pallor, fatigue, reduced exercise tolerance, headache, dizziness and lethargy [2,6]. Irritability, reduced concentration, and decreased physical capacity may also occur with iron deficiency [2]. Tachycardia and other cardiovascular manifestations are generally associated with more severe anemia [2,6].

In infants and young children, iron deficiency anemia may present with irritability, poor feeding, fatigue, lethargy and decreased activity [12], although children may remain asymptomatic [13]. Particular attention should be given to neurodevelopmental manifestations, as early-life iron deficiency has been associated with adverse cognitive, motor, and behavioral outcomes [14].

In school-aged children, iron deficiency may manifest with chronic fatigue, reduced physical capacity, impaired concentration [18], learning and memory difficulties [18], irritability, and sleep disturbances [2]. Pica may also occur and can provide an important clinical clue to iron deficiency [2,17].

Among adolescents, fatigue, reduced exercise tolerance, headache, dizziness, and impaired concentration may become more apparent [2]. Pica and restless legs syndrome (RLS) may also occur in association with iron deficiency [2,15]. In adolescent females, heavy menstrual bleeding is an important risk

factor for iron deficiency because of chronic blood loss [16].

On physical examination, pallor of the skin and oral or conjunctival mucosa and tachycardia may be observed, particularly with iron deficiency anemia [2]. Prolonged iron deficiency may also be associated with mucocutaneous manifestations, including angular cheilitis, glossitis, and koilonychia [2].

Diagnosis

Diagnosis of iron deficiency anemia (IDA) begins with a thorough assessment of clinical symptoms, medical history, dietary practices, and physical examination findings. The diagnosis is subsequently confirmed through laboratory investigations demonstrating both anemia and depleted iron stores [19,20].

The initial and most informative laboratory investigation in children with suspected IDA is the complete blood count (CBC). Automated blood cell analysis typically reveals a microcytic, hypochromic anemia characterized by reduced mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH). In addition, red cell distribution width (RDW) is often elevated, reflecting increased variability in erythrocyte size [19]. Hemoglobin (Hb) values should always be interpreted according to age-specific reference ranges. Reduced hemoglobin concentration, red blood cell count, and hematocrit levels below two standard deviations of the normal values for age and sex are consistent with anemia [5]. According to World Health Organization criteria, anemia is defined as a hemoglobin concentration below 11 g/dL in children aged 6-59 months, below 11.5 g/dL in children aged 5-11 years, below 12 g/dL in children aged 12-14 years and females aged ≥15 years, and below 13 g/dL in males aged ≥15 years [6].

Iron studies are essential for confirming iron deficiency and typically demonstrate low serum ferritin, low serum iron, reduced transferrin saturation, and elevated total iron-binding capacity (TIBC) [19,20]. Among these parameters, serum ferritin is considered the most sensitive and specific indicator of iron deficiency. However, its diagnostic value may be limited in the presence of inflammation, infection, or chronic disease because ferritin is an acute-phase reactant [19,20].

Additional investigations may be necessary to identify the underlying cause of iron deficiency, including assessment of dietary iron intake, gastrointestinal blood loss, malabsorption disorders, parasitic infections where endemic, or periods of increased physiological iron demand associated with rapid growth [20].

Treatment

Oral iron replacement therapy (IRT) is considered the first-line treatment for iron deficiency and iron deficiency anemia (IDA) in infants, children, and adolescents [21,22,24]. The recommended dose of elemental iron is 3-6 mg/kg/day administered orally, with a maximum daily dose of 150-200 mg (21). In adolescents, oral iron doses ranging from 65 to 130 mg of elemental iron daily are commonly recommended [22].

To optimize absorption, oral iron should ideally be administered on an empty stomach. Concurrent consumption of calcium-containing foods and beverages, including milk and dairy products, should be avoided because calcium may reduce iron absorption [22]. Gastrointestinal adverse effects such as abdominal discomfort, nausea, constipation, and diarrhea may limit adherence to treatment in some patients [25].

Response to therapy should be monitored through clinical assessment and laboratory evaluation. In patients presenting with severe anemia (hemoglobin <9 g/dL), hemoglobin may be reassessed after approximately two weeks of treatment, with an increase of at least 1 g/dL considered indicative of an adequate response [22]. Reticulocytosis may occur within several days of treatment initiation; however, routine assessment is generally not recommended because the timing of the reticulocyte response varies among patients [22]. Oral iron therapy should be continued for at least three months after normalization of hemoglobin levels to replenish iron stores adequately. Repeat complete blood count and serum ferritin measurement are recommended at the completion of therapy to confirm correction of anemia and restoration of iron reserves [22].

Intravenous iron replacement therapy is reserved for selected patients who are unable to tolerate oral iron, have poor adherence to oral therapy, suffer from malabsorption disorders, inflammatory bowel disease, chronic kidney disease, or iron-refractory iron deficiency anemia (IRIDA), or who fail to respond adequately to oral treatment [22,24].

Packed red blood cell transfusion is rarely required and should be reserved for patients with severe symptomatic anemia, cardiovascular compromise, or profound anemia [21,22].

Current recommendations suggest considering transfusion in children with hemoglobin concentrations below 5 g/dL or in those with significant clinical instability [22]. When indicated, transfusion should be administered cautiously using a restrictive approach, typically 4-5 mL/kg infused over 3-4 hours, followed by iron replacement therapy to correct the underlying deficiency [22].

In adolescent females with iron deficiency secondary to heavy menstrual bleeding, management should include both iron replacement therapy and treatment of the underlying menstrual disorder.

Hormonal therapy may be required to control menstrual blood loss and prevent recurrence of iron deficiency [21].

Prevention

Prevention of iron deficiency and iron deficiency anemia (IDA) is essential to avoid both hematologic and non-hematologic consequences [21]). Preventive measures should begin during pregnancy, as fetal and neonatal iron stores largely depend on maternal iron status. Perinatal iron deficiency has been associated with long-term neurocognitive impairment that may persist despite subsequent iron therapy. Therefore, the World Health Organization (WHO) recommends iron and folic acid supplementation during pregnancy, particularly in populations with a high prevalence of anemia among women of reproductive age [26]. Delayed umbilical cord clamping at birth is another effective strategy, as it increases neonatal iron stores and reduces the risk of iron deficiency during infancy, especially among preterm infants [26].

Optimal infant feeding practices also play a key role in prevention. Exclusive breastfeeding is encouraged, while infants who are not breastfed should receive iron-fortified formula during the first year of life. Cow's milk should be avoided before 12 months of age because of its low iron content and poor iron bioavailability [26,27]. Preterm and low-birth-weight infants require prophylactic oral iron supplementation, with the American Academy of Pediatrics recommending approximately 2 mg/kg/day of elemental iron from one month until one year of age [26,27]. In exclusively breastfed term infants, supplementation with elemental iron at a dose of 1 mg/kg/day is recommended beginning at four months of age and continuing until adequate iron-containing complementary foods are introduced [26,2]. After one year of age, excessive cow's milk consumption should be avoided and limited to less than 20–24 oz (600–720 mL) daily [21,27],as excessive intake may reduce consumption of iron-rich foods and contribute to occult gastrointestinal blood loss through cow's-milk-induced enteropathy [21].

Routine screening is an additional preventive strategy that facilitates early detection and treatment before severe anemia develops. Screening is typically performed using hemoglobin or hematocrit measurements at approximately 12 months of age, with repeat assessment in high-risk children or in those with persistent risk factors for iron deficiency [21,27]. Continued surveillance is recommended when dietary, medical, or socioeconomic risk factors remain present.

CONCLUSION

Iron deficiency anemia remains one of the most common nutritional disorders affecting children and adolescents worldwide. Despite being largely preventable and treatable, it continues to represent a significant public health challenge because of its adverse effects on hematologic status, growth, neurodevelopment, cognitive function, and physical performance. The development of IDA is multifactorial, resulting from inadequate dietary iron intake, increased physiological requirements, chronic blood loss, or impaired iron absorption. Early recognition through careful clinical assessment and appropriate laboratory investigations is essential for prompt diagnosis and management. Oral iron supplementation remains the cornerstone of treatment, while selected patients may require intravenous iron therapy or additional interventions to address underlying causes. Effective prevention, beginning during pregnancy and continuing throughout childhood and adolescence through adequate nutrition, targeted supplementation, and routine screening, is critical to reducing the burden of disease. Improved awareness among healthcare professionals, caregivers, and public health authorities can facilitate early intervention and contribute to better long-term health and developmental outcomes for children and adolescents affected by iron deficiency anemia.

REFERENCES

1. Cappellini MD, Musallam KM, Taher AT. Iron deficiency anaemia revisited. J Intern Med. 2020 Feb;287(2):153-170. doi:10.1111/joim.13004. Epub 2019 Nov12. PMID: 31665543.
2. Chaber R, Helwich E, Lauterbach R, Mastalerz-Migas A, Matysiak M, Peregud-Pogorzelski J, Styczyński J, Szczepański T, Jackowska T. Diagnosis and Treatment of Iron Deficiency and Iron Deficiency Anemia in Children and Adolescents: Recommendations of the Polish Pediatric Society, the Polish Society of Pediatric Oncology and Hematology, the Polish Society of Neonatology, and the Polish Society of Family Medicine. Nutrients. 2024 Oct 25;16(21):3623. doi: 10.3390/nu16213623. PMID: 39519457; PMCID: PMC11547346.
3. Guideline: Daily Iron Supplementation in Infants and Children. Geneva: World Health Organization; 2016. PMID: 27195348.
4. Mantadakis E, Chatzimichael E, Zikidou P. Iron Deficiency Anemia in Children Residing in High and Low-Income Countries: Risk Factors, Prevention, Diagnosis and Therapy. Mediterr J Hematol Infect Dis. 2020 Jul 1;12(1):e2020041. doi: 10.4084/MJHID.2020.041. PMID: 32670519; PMCID: PMC7340216.
5. Talarico V, Giancotti L, Mazza GA, Miniero R, Bertini M. Iron Deficiency Anemia in Celiac Disease. Nutrients. 2021 May 17;13(5):1695. doi: 10.3390/nu13051695. PMID: 34067622; PMCID: PMC8156426.
6. Aksu T, Ünal Ş. Iron Deficiency Anemia in Infancy, Childhood, and Adolescence. Turk Arch Pediatr. 2023 Jul;58(4):358-362. doi:

10.5152/TurkArchPediatr.2023.23049. PMID: 37357449; PMCID: PMC10440944.
7. Baker RD, Greer FR; Committee on Nutrition American Academy of Pediatrics. Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children (0-3 years of age). Pediatrics. 2010 Nov;126(5):1040-50. doi: 10.1542/peds.2010-2576. Epub 2010 Oct 5.PMID: 20923825.
8. El Amrousy D, El-Afify D, Elsawy A, Elsheikh M, Donia A, Nassar M. Lactoferrin for iron-deficiency anemia in children with inflammatory bowel disease: a clinical trial. Pediatr Res. 2022 Sep;92(3):762-766. doi: 10.1038/s41390-022-02136-2. Epub 2022 Jun 9. PMID: 35681097; PMCID: PMC9556315.
9. Reid CG, Graham R. A Case of Rickets and Pediatric Iron Deficiency Anemia in Alabama. Cureus. 2024 May 12;16(5):e60140. doi: 10.7759/cureus.60140. PMID: 38864063; PMCID: PMC11165436.
10. Broekaert IJ, Assa A, Borrelli O, Saccomani MD, Homan M, Martin-de-Carpi J, Mas E, Miele E, Misak Z, Sila S, Thomson M, Tzivinikos C, Dolinsek J. Approach to anaemia in gastrointestinal disease: A position paper by the ESPGHAN Gastroenterology Committee. J Pediatr Gastroenterol Nutr. 2025 Mar;80(3):510-532. doi: 10.1002/jpn3.12454. Epub 2025 Jan 9. PMID: 39783775; PMCID: PMC11874238.
11. Gallagher PG. Anemia in the pediatric patient. Blood. 2022 Aug 11;140(6):571-593. doi: 10.1182/blood.2020006479. PMID: 35213686; PMCID: PMC9373018.
12. Iron needs of babies and children. Paediatr Child Health. 2007 Apr;12(4):333-6. doi: 10.1093/pch/12.4.333. PMID: 19030384; PMCID: PMC2528681.
13. Joo EY, Kim KY, Kim DH, Lee JE, Kim SK. Iron deficiency anemia in infants and toddlers. Blood Res. 2016 Dec;51(4):268-273. doi: 10.5045/br.2016.51.4.268. Epub 2016 Dec 23.PMID: 28090490; PMCID: PMC5234236.
14. Lozoff B, Beard J, Connor J, Barbara F, Georgieff M, Schallert T. Long-lasting neural and behavioral effects of iron deficiency in infancy. Nutr Rev. 2006 May;64(5 Pt 2):S34-43; discussion S72-91. doi: 10.1301/nr.2006.may.s34-s43. PMID: 16770951; PMCID: PMC1540447.
15. Dosman C, Witmans M, Zwaigenbaum L. Iron's role in paediatric restless legs syndrome - a review. Paediatr Child Health. 2012 Apr;17(4):193-7. doi: 10.1093/pch/17.4.193. PMID: 23543250; PMCID: PMC3381661.
16. Screening and Management of Bleeding Disorders in Adolescents With Heavy Menstrual Bleeding: ACOG COMMITTEE OPINION, Number 785. Obstet Gynecol. 2019 Sep;134(3):e71-e83. doi: 10.1097/AOG.0000000000003411. PMID: 31441825.
17. Borgna-Pignatti C, Zanella S. Pica as a manifestation of iron deficiency. Expert Rev Hematol. 2016 Nov;9(11):1075-1080. doi: 10.1080/17474086.2016.1245136. Epub 2016 Oct 19. PMID: 27701928.
18. Gutema BT, Sorrie MB, Megersa ND, Yesera GE, Yeshitila YG, Pauwels NS, De Henauw S, Abbeddou S. Effects of iron supplementation on cognitive development in school-age children: Systematic review and meta-analysis. PLoS One. 2023 Jun 27;18(6):e0287703. doi: 10.1371/journal.pone.0287703. PMID: 37368919; PMCID: PMC10298800
19. Powers JM, Heeney MM, Hord J, Lehmann CU, Abrams SA, Buchanan GR; AAP Section on Hematology-Oncology; AAP Committee on Nutrition; AMERICAN SOCIETY OF PEDIATRIC HEMATOLOGY-ONCOLOGY. Prevention, Screening, Diagnosis, and Treatment of Iron Deficiency and Iron Deficiency Anemia in Infants, Children, and Adolescents: Clinical Report. Pediatrics. 2026 Jul 1;158(1):e2026077414. doi: 10.1542/peds.2026-077414. PMID: 42324084.
20. BMJ Best Practice. *Iron deficiency anemia*[Internet]. London: BMJ Publishing Group; 2026 [cited 2026 Aug 17]. Available from: https://bestpractice.bmj.com/topics/en-us/94
21. Kliegman RM, St Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 20th ed. Philadelphia, PA: Elsevier; 2016.
22. Iolascon A, Andolfo I, Russo R, Sanchez M, Busti F, Swinkels D, Aguilar Martinez P, Bou-Fakhredin R, Muckenthaler MU, Unal S, Porto G, Ganz T, Kattamis A, De Franceschi L, Cappellini MD, Munro MG, Taher A; from EHA-SWG Red Cell and Iron. Recommendations for diagnosis, treatment, and prevention of iron deficiency and iron deficiency anemia. Hemasphere. 2024 Jul 15;8(7):e108. doi: 10.1002/hem3.108. PMID: 39011129; PMCID: PMC11247274.
23. World Health Organization. *Anaemia* [Internet]. Geneva: World Health Organization; 2025 [cited 2026 Aug 18]. Available from: WHO Anaemia Fact Sheet.
24. Aleem A, Alsayegh F, Keshav S, Alfadda A, Alfadhli AA, Al-Jebreen A, Al-Kasim F, Almuhaini A, Al-Zahrani H, Batwa F, Denic S, Jazzar A, Owaidah T, Qari M, Qari Y, Taha M. Consensus Statement by an Expert Panel on the Diagnosis and Management of Iron Deficiency Anemia in the Gulf Cooperation Council Countries. Med Princ Pract. 2020;29(4):371-381. doi: 10.1159/000503707. Epub 2019 Nov 5. PMID: 31698356; PMCID: PMC7445696.
25. Tolkien Z, Stecher L, Mander AP, Pereira DI, Powell JJ. Ferrous sulfate supplementation causes significant gastrointestinal side-effects in adults: a systematic review and meta-analysis. PLoS One. 2015 Feb 20;10(2):e0117383. doi:

10.1371/journal.pone.0117383. PMID: 25700159; PMCID: PMC4336293.

26. Moscheo C, Licciardello M, Samperi P, La Spina M, Di Cataldo A, Russo G. New Insights into Iron Deficiency Anemia in Children: A Practical Review. Metabolites. 2022 Mar 25;12(4):289. doi: 10.3390/metabo12040289. PMID: 35448476; PMCID: PMC9029079.
27. Kazal LA Jr. Prevention of iron deficiency in infants and toddlers. Am Fam Physician. 2002 Oct 1;66(7):1217-24. PMID: 12387433.