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Iron Deficiency Anemia Diagnosed in Female Teenagers

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Abstract

Iron deficiency anemia (IDA) is a common, worldwide problem with a prevalence of 9% in toddlers, 9-11% in adolescent girls, and less than 1% in teenager boys. Iron deficiency occurs in both developing and developed countries, making it the most common nutritional deficiency worldwide. In response to this epidemic, the World Health Organization's goal is to reduce anemia in women of reproductive age by 50% in addition to other targeted pediatric initiatives by 2025. IDA is most commonly seen in the following pediatric populations: infants fed cow's milk, toddlers fed large volumes of cow's milk, and menstruating teenage girls not receiving supplemental iron. The second peak of IDA seen in teenagers is primarily due to rapid growth often combined with poor dietary intake of iron. These contributors may be compounded by menstrual blood loss in adolescent females. Iron is an important component in the formation of hemoglobin, the protein found in red blood cells necessary for oxygen transport. Anemia develops as iron stores are depleted. Pediatric patients may deplete iron stores, without presenting with an anemia, which is consistent with iron deficiency. Because of this, hemoglobin alone is not an accurate indicator of iron deficiency anemia. With less iron available, erythropoiesis is affected. A low serum ferritin (in the absence of any concurrent inflammatory condition) is specific for iron deficiency and, coupled with low hemoglobin levels and abnormal red blood cell indices (low mean corpuscular volume [MCV], low mean corpuscular hemoglobin concentration [MCHC], and high red cell distribution width [RDW]), supports a diagnosis of iron deficiency anemia.

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INTRODUCTION

Iron Deficiency Anemia Diagnosed in Female Teenagers

Iron deficiency anemia (IDA) is a common, worldwide problem with a prevalence of 9% in toddlers, 9-11% in adolescent girls, and less than 1% in teenager boys [1]. Iron deficiency occurs in both developing and developed countries, making it the most common nutritional deficiency worldwide [2]. In response to this epidemic, the World Health Organization's goal is to reduce anemia in women of reproductive age by 50% in addition to other targeted pediatric initiatives by 2025 [3]. IDA is most commonly seen in the following pediatric populations: infants fed cow's milk, toddlers fed large volumes of cow's milk, and menstruating teenage girls not receiving supplemental iron [1]. The second peak of IDA seen in teenagers is primarily due to rapid growth often combined with poor dietary intake of iron [4]. These contributors may be compounded by menstrual blood loss in adolescent females [4].

Iron is an important component in the formation of hemoglobin, the protein found in red blood cells necessary for oxygen transport. Anemia develops as iron stores are depleted. Pediatric patients may deplete iron stores, without presenting with an anemia, which is consistent with iron deficiency. Because of this, hemoglobin alone is not an accurate indicator of iron deficiency anemia. With less iron available, erythropoiesis is affected. A low serum ferritin (in the absence of any concurrent inflammatory condition) is specific for iron deficiency and, coupled with low hemoglobin levels and abnormal red blood cell indices (low mean corpuscular volume [MCV], low mean corpuscular hemoglobin concentration [MCHC], and high red cell distribution width [RDW]), supports a diagnosis of iron deficiency anemia [5].

We present a 12 year-old African American female, previously healthy, who presented to the hospital for admission with hemoglobin of 4.8 and ferritin of 5.1. Despite being an active cheerleader, she had experienced intermittent headaches and dizziness for the prior year. Her coach reported that she, oftentimes, had to rest during practice due to dizziness. Of note, her menarche started at 11 years of age with subsequent heavy, irregular menstrual cycles. She was, previously, started on an iron supplement, but was not compliant with administration. She was admitted and received two units of packed RBCs. Her hemoglobin appropriately increased to 7.2. She was started on



an optimal dose of ferrous sulfate (325mg TID) and discharged to home in stable condition. She was seen in our outpatient clinic for follow-up. She reported 100% compliance with her iron replacement which was reflected in her hemoglobin of 12 and ferritin of 19.9. She was discharged from our care.

Our patient represents a classic case of iron deficiency anemia in the teenage female population. Adolescents with iron deficiency anemia initially present to their primary care physicians. Referral to a hematologist may follow, especially for the management and treatment of severe or refractory iron deficiency anemia. Physical exam findings include pallor, bruising, weakness, and fatigue. Vital signs abnormalities, such as tachycardia, hypotension, and hypoxia, may be present in severe cases of anemia. IDA may also lead to cardiac and central nervous system abnormalities due to poor oxygen transport to vital organs. IDA is associated with apathy, irritability, and poor concentration. Children and adolescents with IDA are at greater risk of developing cognitive deficits and poor school performance.

In an otherwise healthy pediatric patient, a therapeutic trial of oral iron supplementation is the best diagnostic evaluation for iron deficiency anemia. Serial re-assessments will serve to document compliance and improvement in symptoms and lab abnormalities. Therapeutic dosing is crucial in order to elicit the most optimal response. Ferrous iron should be prescribed in terms of elemental iron content. In pediatric patients, ideal iron dosing is 3-6mg/kg/day by mouth divided BID-TID (or 100-200mg of elemental iron in older children) [5]. Parental iron replacement is only recommended for certain populations including those with intolerance to oral iron, need for rapid replacement, poor intestinal absorption, and need for erythropoietin [x]. With patient compliance, laboratory values show a positive response within days to weeks. Reticulocytosis occurs within 5-10 days after initiation of iron therapy followed by rise in hemoglobin over the next weeks to months. If, by two weeks, there is no improvement in hemoglobin levels, then a more thorough investigation should occur. Duration of treatment should last 2-3 months after hemoglobin and other indices have normalized [5]. In a female teenager who is menstruating heavily with evidence of iron deficiency anemia, in addition to ferrous sulfate replacement, it would behoove the patient to see a pediatric gynecologist regarding hormonal therapy for menstrual bleeding control. Iron therapy can lead to gastrointestinal discomfort, constipation, and bloating which, oftentimes, make patients self-discontinue.

Certainly, if improvement is not noted after proper

compliance with iron therapy and/or other clinically relevant information is present, other differential diagnoses should be considered. Thalassemia, either a quantitative or qualitative deficiency in hemoglobin synthesis, commonly mimics iron deficiency anemia with both being characterized by microcytic anemias. Family history should be investigated and hemoglobin variants considered during the initial evaluation. In addition, lead poisoning and copper deficiency result in similar red blood cell index abnormalities and should be considered in the appropriate situation. Inflammatory disease states such as rheumatologic disease and inflammatory bowel disease, especially in the teenage population, could be emerging and confound the laboratory picture. However, with inflammatory states, even though the hemoglobin may be low, the ferritin level is, generally, elevated. In situations where hemoglobin is not improving with iron replacement and/or there is a strong indication for other disease conditions (family or individual history of chronic disease), further evaluation, most often by a subspecialist, is warranted. Lastly, if there are other cell counts that are abnormal in addition to hemoglobin, a bone marrow evaluation should be considered to evaluate for infiltrating malignancies and/or bone marrow failure syndromes.

In conclusion, iron deficiency anemia is a common pediatric problem that is completely treatable if adequately targeted. Universal screening is somewhat controversial, but most pediatricians practice screening of all infants between 9 and 15 months of age and in symptomatic teenagers (especially females). If diagnosed with iron deficiency anemia, adequate dosing of ferrous iron is crucial for optimal response. In addition, routine iron replacement should be considered in heavily menstruating teenage females. Other, more serious entities should be investigated in the absence of a robust response to iron replacement in a compliant patient.

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