

# Treatment of Iron Deficiency Anemia in Adolescent Girls

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**Abstract**—India is the one of the fastest growing youth populations in the world with an estimate 190 millions adolescent in which 22% are girls. As per recent report from UNICEF more than half [59%] of adolescent girls in India. Among adolescents, girls constitute vulnerable groups due to family with limited resources, female child is more likely to be neglected and another added burden is menstrual blood loss and also influences of traditional eating habits and fear of gaining weight. Many adolescent girls are at an increased risk for anemia due to their rapid growth and increasing muscle mass. However, adolescent females with heavy menstrual bleeding are at an even greater risk for anemia. Heavy menstruation in adolescent females not only has negative effects on health-related quality of life and school attendance, but also has major health implications such as iron deficiency anemia. Anemia in the adolescence causes reduced physical and mental capacity and diminished concentration in work and educational performance, and also poses a major threat to future safe motherhood in girls. About 75% of teen girls do not meet the Recommended Dietary Allowance (RDA) for iron, due in part to busy lifestyles, and self-imposed trendy diets. At a time in their lives when young women have an increased need for iron to compensate for menstrual blood loss and increased growth, many young women are preoccupied with body image. To reduce anemia amongst adolescent girls intakes of highly bioavailable forms of iron (supplemental iron and red meat) and fruit, a dietary source of an enhancer of nonheme-iron absorption (vitamin c), promote high iron stores, whereas foods containing phytate (whole grains) decrease these stores. Individual patterns may be important modulators of high iron stores for the deficient.

**Keywords:**-Anemia, adolescence, iron supplement, nonheme-absorption, phytate, recommended dietary allowance.

## 1. INTRODUCTION-

Anemia (from the ancient Greek ἀναιμία, *anaimia*, meaning 'lack of blood') is defined by a decrease in the total amount of hemoglobin or the number of red blood cells. Iron deficiency anemia is a form of anemia due to the lack of sufficient iron to form normal red blood cells. Iron deficiency anemia is typically caused by inadequate intake of iron, chronic blood loss, or a combination of both. Iron deficiency occurs in two main forms: absolute or functional. Absolute iron deficiency arises when total body iron stores are low or exhausted; functional iron deficiency is a disorder in which total body iron stores are normal or increased, but the iron supply to the

bone marrow is inadequate. Absolute and functional deficiencies can coexist. Functional iron deficiency can be present in many acute and chronic inflammatory states, and hepcidin—the master regulator of iron homeostasis—has a key role in pathogenesis. Adolescence has been defined by the World Health Organization as the period of life spanning the ages between 10 to 19 years. This is the formative period of life when the maximum amount of physical, psychological, and behavioral changes take place. This is a vulnerable period in the human life cycle for the development of nutritional anemia, which has been constantly neglected by public health programs. Anemia in the adolescence causes reduced physical and mental capacity and diminished concentration in work and educational performance, and also poses a major threat to future safe motherhood in girls.

## 2. PREVALENCE

Nutritional anemia is one of the India's major public health problems. The prevalence of anemia ranges from 33% to 89% among pregnant women and is more than 60% among adolescent girls. Under the anemia prevention and control program of the government of India, iron and folic acid tablets are distributed to pregnant women, but no such program exists for adolescent girls. Among adolescent girls (n=4,337) from 16 districts, the overall prevalence of anemia (defined as hemoglobin <120g/L) was 90.1% with 7.1% having severe anemia (hemoglobin <70g/L).

## 3. PATHOPHYSIOLOGY

Iron is an essential component of haemoglobin in red blood cells and of myoglobin in muscles, which contain around 60% of total body iron. It is also necessary for the functioning of various cellular mechanisms, including enzymatic processes, DNA synthesis, and mitochondrial energy generation. In adults, the body contains 3–5 g of iron; 20–25 mg is needed daily for production of red blood cells and cellular metabolism. Because dietary intake is limited (1–2 mg per day), other sources are needed for iron homeostasis—eg, recycling of ageing erythrocytes in macrophages, exchange of iron in iron-containing enzymes, and iron stores. About 1–2

mg of iron is lost daily as a result of menstrual bleeding, sweating, skin desquamation, and urinary excretion. Iron is available in two forms: haem and non-haem iron. Iron is complexed as  $\text{Fe}^{2+}$  (ferrous iron) in haemoglobin in the haem form, which is present in animal food sources, such as meat, poultry, and seafood.<sup>30</sup> Non-haem iron ( $\text{Fe}^{3+}$  or ferric iron) is present in the vegetarian diet (black tea, cacao, cereals, dried fruit, etc). Haem iron is estimated to contribute 10–15% of total iron intake in meat-eating populations, but because it is generally better absorbed—with a rate of absorption estimated at 15–35%—than non-haem iron, it can account for more than 40% of total absorbed iron. In iron homeostasis, a small peptide called hepcidin, which is mainly secreted by hepatocytes and was first described in 2001 in mice with iron overload, has a crucial role in the control of iron availability to tissues. Outside the liver, other cell types and organs, such as macrophages, adipocytes, the heart, and the kidneys, can produce hepcidin. In plasma, hepcidin is bound to  $\alpha_2$ -macroglobulin and albumin, and can be cleared via the kidney. The main role of hepcidin is to control surface expression of FPN1 by binding to the protein, which is then internalised and degraded by lysosomes. FPN1 is the only known iron-exporting protein, so after its degradation enterocytes, macrophages, and hepatocytes can no longer export iron, which is sequestered in these cells. High expression of hepcidin decreases plasma iron concentrations; low expression increases concentrations. Hepcidin expression is upregulated by high concentrations of iron in the liver and plasma, inflammation, and physical activity whereas it is downregulated by iron deficiency, erythropoiesis, hypoxia, and endocrine signals (testosterone, estrogen, and growth factors). A new hormone called erythroferrone was identified in 2014. It is produced by erythroblasts in response to erythropoietin, and mediates hepcidin suppression during stress erythropoiesis.

#### 4. TREATMENT OF ANEMIA IN ADOLESCENT GIRLS

About 75% of teen girls do not meet the Recommended Dietary Allowance (RDA) for iron, due in part to busy lifestyles, and self-imposed trendy diets. Due to heavy menstrual bleeding (HMB) and an inadequate diet frequently leads to iron deficiency in young women. In Nationwide Children's Hospital they developed a program for females with Heavy Menstrual Bleeding at the Adolescent, quickly anemia became a very prevalent concern despite the intervention of oral iron therapy. Most of the patients seen in clinic had tried oral iron therapy due to its easy accessibility. And provide age-appropriate education to adolescent females with anemia, and started by first conducting a fairly extensive literature search using key words including anemia, education, prevention, treatment, and therapy for teens and/or adolescents. Iron deficiency anemia is usually treated with iron supplements taken by mouth for several months. Iron is best absorbed when given between meals. Encouraging iron

supplements in mid-morning, between breakfast and lunch or mid-afternoon between lunch and dinner.

#### 5. DIETARY RECOMMENDATION FOR AN IRON DEFICIENT ADOLESCENT GIRLS-

Consume at least three 4 portions of lean red meat per week. For vegetarians an increased consumption of foods with high non-heme iron content along with avoidance of substances that impair the absorption of iron. For a list of iron content in foods The key to preventing iron deficiency is to eat a balanced diet. Eating well rounded meals which include both heme and non-heme iron sources will boost a child's iron stores and help alleviate anemia.

But iron by itself is enough, vitamin is also needs for the body to absorb non-heme iron. Vitamin c will boost the iron get from foods such as legumes, whole grains and vegetables. Substances that impair iron absorption include: coffee, tea, high fiber, calcium and eggs. Substances that improve the absorption of iron include naturally occurring beta carotene (not supplemental) and acidic foods such as tomatoes or oranges. Once iron balance is achieved, continue on a balanced diet such as the following:

Get adequate daily protein from meat, soy or plant food combinations such as beans or lentils with rice. Reduce or eliminate refined sugar from the diet. Small amounts of honey, or molasses are sufficient. Limit the amount of sugar substitutes as well, especially aspartame. Do not avoid complex carbs (whole grains) from the diet. Eat at least 3 portions of complex carbs per day. Eat at least 5 servings per day of fresh or fresh frozen fruits (berries are best choices especially for diabetics) and vegetables. Though the iron in fruits and vegetables is not easily absorbed, these food items are essential for nutrients such as vitamin C, beta carotene and chlorophyll. Fruits and vegetables are a good source of antioxidants, which counter harmful free radical activity. Consume at least two cups of dairy per day such as skim-milk, yogurt (one with active cultures and no sugar).

#### 6. CONCLUSION

Adolescent girls are at a high risk for anemia and malnutrition. Inadequate nutrition during adolescence can have serious consequences throughout the reproductive years of life and beyond. Very often, in India, girls get married and pregnant even before the growth period is over, thus doubling the risk for anemia.

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