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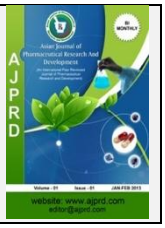

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Review Article

Iron Deficiency and Iron Deficiency Anemia in Women: A Review

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A B S T R A C T

Iron deficiency (ID) affects more than 20% of women during their reproductive years, making it the most prevalent micronutrient deficiency in the world. Hepcidin is a peptide hormone that is mostly made by the liver and regulates iron absorption. For ID and iron deficiency anemia (IDA) to be successfully treated with oral preparations, parenteral iron, or blood transfusions, an understanding of iron metabolism is essential. Oral preparations can cause gastrointestinal adverse effects and have varying iron contents. When there are problems with oral iron compliance or tolerance, comorbidities that could impair absorption, or persistent iron losses that surpass absorptive capability, parenteral iron is recommended. When quick iron replenishment is necessary to avoid physiological decompensation, or when administered prior to non-deferrable surgery, it might also be the best choice. In our role as gynecologists, we address women's heavy menstrual bleeding (HMB) and presume that primary care physicians are attending to the related ID/IDA. It is now our responsibility to take the lead in concurrently diagnosing, treating, and controlling ID/IDA and HMB. This dual management will greatly enhance their standard of living. We will provide a summary of the role iron plays in cellular processes, explain how to diagnose ID/IDA, and assist doctors in selecting from a variety of therapy options in this review.

Key words: Hepcidin, Deficiency, Anemia, Hemoglobin

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INTRODUCTION:

Iron deficiency poses serious public health issues and is the most prevalent nutritional issue in the world. A disproportionate number of women and children suffer from iron deficiency (ID) and iron deficiency anemia (IDA). According to WHO estimates, more than 2,109 people, or over one-third of the global population, suffer from anemia, primarily as a result of iron deficiency[1, 2].

In addition to the iron loss in menstrual blood, premenopausal women are susceptible to ID because they frequently consume inadequate amounts of iron from their diet and may engage in restricted eating habits in an attempt to reduce their body weight. Men should consume 8 mg of iron per day, while women of reproductive age should consume 18 mg[3].

The lack of iron Anemia occurs when the body's iron storage, iron intake, and iron loss are not balanced enough to maintain erythrocyte formation. Although it seldom results in death, iron deficiency anemia has a major negative influence on human health. Although doctors usually ignore this illness, it is easily diagnosed and treated in the modern world. On the other hand, it is a medical condition that primarily impacts people in developing nations[4].

Iron is a micronutrient that is essential for hemoglobin production and enzyme function. IDA is particularly high among pregnant women, children under five,

adolescent girls, and women due to their higher nutritional requirements for iron[5].

Anemia is defined as hemoglobin below two standard deviations of the mean for the age and gender of the patient. Iron is an essential component of the hemoglobin molecule. The most common cause of anemia worldwide is iron deficiency, which results in microcytic and hypochromic red cells on the peripheral smear. Several causes of iron deficiency vary based on age, gender, and socioeconomic status. The patient often will have nonspecific complaints such as fatigue and dyspnea on exertion. Treatment is a reversal of the underlying condition as well as iron supplementation. Iron supplementation is most often oral, but certain cases may require intravenous iron. Patients with iron-deficient anemia have been found to have a longer hospital stay, along with a higher number of adverse events[6].

Iron Deficiency/Iron Deficiency Anemia In Women Across Their Various Stages Of Life

Unfortunately, IDA's substantial consequences on quality of life, morbidity, mortality, and physical and cognitive capabilities are overlooked, leading to the misconception that ID and IDA are benign illnesses[7].

Reproductive Age

According to recent statistics, 40–50% of European nonpregnant women have inadequate iron storage, despite the fact that ID is most prevalent in low-income nations.The frequency of IDA is approximately ten times higher in women than in men of the same age, which is well recognized. Regular blood loss during menstruation, which is frequently linked to inadequate iron consumption, is the main cause of this discrepancy. Due to their increased iron needs for rapid growth and menstrual blood loss, teenage girls are especially susceptible to this illness. Additionally, a number of diseases, including endometriosis, adenomyosis, endometrial hyperplasia, and chronic gynecologic bleeding brought on by uterine fibroids, can influence a woman's propensity for ID. Other reasons of IDA in women include frequent blood donation, intestinal malabsorption issues, and benign and malignant GI abnormalities[8].

Heavy Menstrual Bleeding

Any blood loss that consistently above 80 milliliters every menstrual cycle is considered heavy menstrual bleeding. A definition that demands blood loss be quantified, however, is only helpful for research projects and precise measurements of menstrual blood flow. According to the UK-based National Institute for Health and Care Excellence (NICE), menstrual blood loss that is consistently excessive and "affects the physical, social, emotional, or material quality of life of the patient" should also be considered for the diagnosis of HMB[9].

Approximately 18–38% of women of reproductive age are thought to have HMB, and its incidence may rise as women get closer to menopause. However, there is a lot of variation in how HMB is reported, and it is probably underdiagnosed[9].

Pregnant Women

Despite the brief reprieve from iron losses during menstruation, the physiological iron demand peaks during pregnancy (about 1,000–1,200 mg with an average weight of 55 kg). This amount includes around 350 mg linked to placental and fetal growth, 500 mg linked to red cell mass expansion, and 250 mg linked to blood loss during delivery. Iron requirements fluctuate during pregnancy and show an increasing tendency; in fact, the first trimester has a lower iron requirement (0.8 mg/day) and the third trimester has a significantly larger demand (3.0–7.5 mg/day). About 40% of women have little or no iron stores at the start of pregnancy, and up to 90% have iron reserves of less than 500 mg, which is not enough to satisfy the increased needs. Remarkably, pregnant women are rarely tested for ID unless they are anemic, and when other iron parameters are not included in screening laboratory testing, low Hb concentration alone may miss up to 55% of pregnant women with ID[10].

Etiology

- Iron deficiency (ID) can be brought on by a number of pathologic, physiological, environmental, and hereditary causes (Fig. 1). Most importantly, different patient categories (elderly, women, and children) may have etiologies that differ greatly or often coexist[11].
- Anemias induced by nutritional inadequacies are a major worldwide concern. For instance, anemias can be brought on by deficiencies in iron, folic acid, and vitamin B12[12].

- The cause of iron-deficiency anemia varies based on age, gender, and socioeconomic status. Iron deficiency may result from insufficient iron intake, decreased absorption, or blood loss. Iron-deficiency anemia is most often from blood loss, especially in older patients. It may also be seen with low dietary intake, increased systemic requirements for iron such as in pregnancy, and decreased iron absorption such as in celiac disease. In neonates, breastfeeding is protective against iron deficiency due to the higher bioavailability of iron in breast milk compared to cow's milk; iron deficiency anemia is the most common form of anemia in young children on cow's milk. In developing countries, a parasitic infestation is also a significant cause of iron-deficiency anemia. Dietary sources of iron are green vegetables, red meat, and iron-fortified milk formulas[13].

Epidemiology

The World Health Organization estimated worldwide prevalence of anemia to be 42% in children, 29% in non-pregnant women, and 38% in pregnant women in 2011. In 2013, iron deficiency (ID) was identified as the predominant cause of anemia among the 1.93 billion anemic people (27% of the world's population) globally, making iron deficiency anemia (IDA) a major global health issue. The people most at risk are women and children, regardless of socioeconomic status or geography. In 2017, the Global Burden of Diseases Study reported that dietary ID remains the fourth and twelth leading cause of years lived with disability in women and men, respectively. Yet, the true prevalence and clinical impact of ID remains difficult to capture. Population-based studies are limited, functional iron deficiency (FID) is common, and the prevalence of ID without anemia remains uncertain[14].

Prevalence of Anemia

Hemoglobin levels and blood samples were analyzed to detect anemia in children and women. The study does not contain severe cases.64% of male children had normal anemia, 18% had mild anemia, and 18% had substantial anemia, according to Figure 1. While 56% of female children had normal hemoglobin, 44% had moderate anemia. The frequency in females was 14.7% for moderate anemia, 20.6% for light anemia, and 64.7% for normal anemia. There was a clear gender difference in anemia, as evidenced by the higher hemoglobin levels in female children compared to male infants.

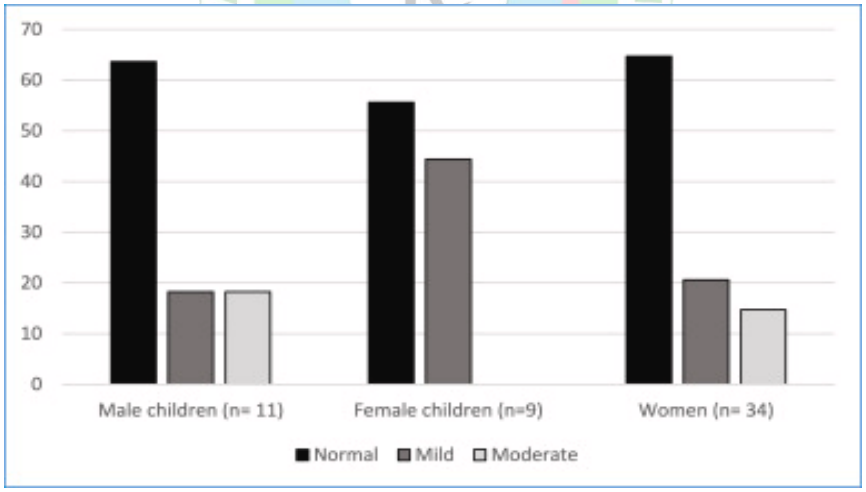

Figure 1: Prevalence of anemia in children and women[15]

Pathophysiology

Iron is a necessary element that is mostly regulated by intestine absorption, iron recycling, and dietary intake. There are two types of dietary iron: haem and non-haem. Haemoglobin (Hb) and myoglobin, which are found in meat, poultry, and fish, are the sources of easily absorbed haem iron. Although it is less readily absorbed, non-haem iron is primarily present in plant foods. Plant-based compounds like phytate, oxalate, polyphenols, and tannin, as well as some medications like proton pump inhibitors, reduce the absorption of non-haem iron. On the other hand, ascorbic acid, citrate, and gastric acid promote the absorption of iron. About 5 to 15 mg of elemental iron and 1 to 5 mg of hemo iron are consumed daily in a healthy diet, but only 1-2 mg are eventually absorbed into the intestine, primarily in the duodenum and proximal jejunum. Information on the pathways involved in iron absorption is shown in Figure 2[16].

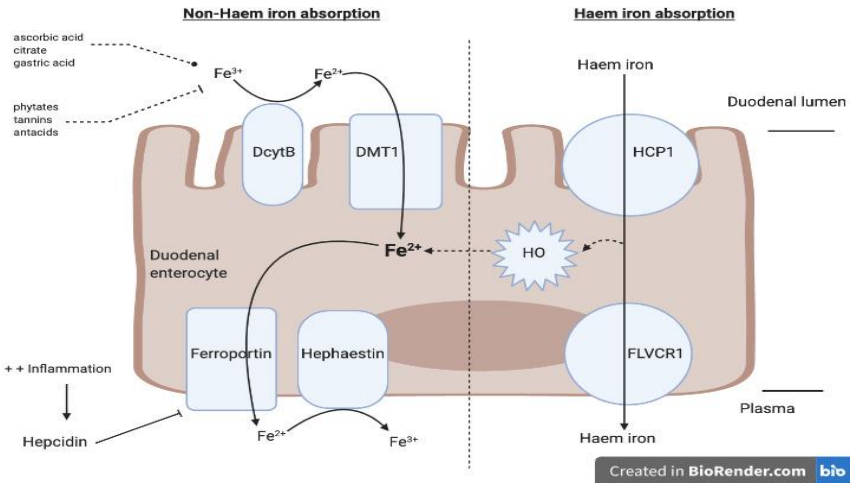

Figure 2: Information on the pathways involved in iron absorption

The two different iron absorption pathways- Non-haem absorption pathway (left): insoluble ferric iron (Fe3+) is reduced to absorbable ferrous iron (Fe2+), which is carried out by the enzyme duodenal cytochrome B (DcytB). The divalent metal transporter 1 (DMT1) imports Fe2+ across the apical surface and into the cell, which can then be either stored as ferritin or exported into circulation through ferroportin. Prior to exiting the enterocyte, Fe2+ must be oxidised back to Fe3+ by hephaestin or ceruloplasmin. Haem absorption pathway (right): the haem carrier protein (HCP1) transports haem iron directly into the enterocyte. Once inside the enterocyte, haem iron can either be released into plasma via the haem exporter FLVCR1 or be converted back into Fe2+ via the haem oxidase (HO) enzyme. The ferroportin receptor then releases Fe2+ into the plasma. Hepcidin, a hepatic peptide hormone, controls ferroportin, the sole iron exporter, by promoting its endocytosis. Hepcidin production and circulation are regulated by plasma iron concentration and iron stores. Hepcidin is increased in the presence of inflammation, which then promotes the degradation of ferroportin and subsequently impairs the exportation of cellular iron into plasma[17].

Clinical Characteristics

The majority of patients with mild IDA are asymptomatic. Patients may exhibit vague symptoms including exhaustion, pallor, and dyspnea with exertion as their anemia worsens. Tachycardia, conjunctival and widespread pallor, koilonychia, glossitis, stomatitis, and other symptoms suggestive of heart failure may be found during a physical examination. There may also be behavioral abnormalities such restless legs syndrome and pica, which is the desire and intake of nonfood objects[18,19]. Occasionally, patients with GI causes of IDA may report "alarm" symptoms such altered stool quality, epigastric pain, altered bowel habits, weight loss, early satiety, and decreased appetite. IDA-related esophageal webs make to the Plummer-Vinson syndrome.

Diagnosis:

A Hb level of less than 13.0 g/dL in male adults, less than 12.0 g/dL in non-pregnant female adults, and less than 11.0 g/dL in pregnant women is considered anemia by the World Health Organization. Care must be exercised, especially when interpreting borderline readings, as Hb levels can differ by age and ethnicity. Additionally, baseline hemoglobin levels may be higher among smokers and residents of higher elevations 49, 50 and endurance sports activity may change hemoglobin levels 51.

The hypochromic and usually microcytic iron deficiency anemia is distinguished from macrocytic anemia by the mean corpuscular Hb and mean corpuscular volume. The use of thiopurine drugs (such as azathioprine in IBD) or deficiencies of several nutrients (such as malabsorption) can cause iron deficiency anemia and macrocytosis together[20].

Table 1: Diagnostic criteria for iron deficiency anaemia

Serum markers	Diagnosis for IDA
Haemoglobin	< 130 g/L males < 120 g/L females < 110 g/L in pregnancy
Ferritin	< 30 ug/L if no inflammation < 100 ug/L if inflammation
Transferrin	Raised
Total iron binding capacity	Raised
Iron	Reduced
Transferrin saturations	< 20%
Mean corpuscular volume	Low

The breakdown of IDA diagnostic criteria is shown in Table 1. Because a considerable quantity of iron must be lost before the Hb levels start to drop, it is important to remember that iron insufficiency should not be ruled out in the presence of a normal Hb. Therefore, mild iron deficiency without anemia is indicated by a low mean corpuscular hemoglobin with a normal hemoglobin or an increase in red cell distribution width[21].

Management

Restoring iron reserves and bringing hemoglobin levels back to normal should be the goals of treatment for patients with IDA. Pregnancy outcomes, morbidity, prognosis, and quality of life have all been demonstrated to improve as a result[22]. Three methods are available for iron replenishment: packed red cell transfusion, parenteral oral, and oral iron. Every approach has advantages and disadvantages, which will be covered in more detail below.

Treatment:

Treatment of iron deficiency should begin with dietary replacement (i.e., fortified cereals and breads, red meat, beans, green leafy vegetables), but when diet alone is inadequate to restore iron stores and Hb to normal levels, or when anemia is severe, treatment with exogenous iron supplements should be implemented. Treatment is dependent on the urgency of the situation and the patient's presenting symptoms. If serum hemoglobin is < 8 g/dl and the patient is symptomatic with shortness of breath, extreme fatigue, or signs of myocardial ischemia, then an immediate blood transfusion is warranted. When the patient is asymptomatic and the hemoglobin level is within an acceptable range, treatment should begin with oral iron[23].

Oral Iron:

Oral iron is a convenient, affordable, and safe first-line treatment for IDA. Because elemental iron is inexpensive and has a high bioavailability, ferrous sulfate and ferrous gluconate are the two recommended oral forms of iron. Since iron is better absorbed in an acidic environment, ferrous salts should be taken with orange juice to maximize absorption. Ascorbic acid also lessens the oxidation of ferrous iron to ferric iron. Tea, coffee, carbonated drinks that include phosphate, and drugs that prevent the production of stomach acid (such as proton pump inhibitors, H2 blockers, and antacids) are among the foods that decrease the absorption of iron. Three to four times a day, 300 mg tablets of ferrous sulfate (60 mg elemental iron) and 320 mg tablets of ferrous gluconate (36 mg elemental iron) are recommended. Constipation, nausea, and epigastric discomfort are common side effects of oral iron supplements that are dose-related. Up to 20% of patients may experience adverse symptoms, which could hinder their compliance. More than 90% of ingested iron is not absorbed by the duodenum, which can result in erosions and enteric siderosis because it can only absorb 10–20 mg of iron per day. Lower dosages, like 15 mg of elemental iron per day, can nevertheless result in successful iron replacement. Iron may not be released in the duodenum, where it is mainly absorbed, hence enteric-coated iron pills are less effective but better tolerated. Oral iron therapy aims to restore iron reserves in about 3–4 months by inducing reticulocytosis within days and increasing serum hemoglobin by 1-2 g/dl every 2 weeks. Given the

aforementioned factors, if negative effects occur, lowering the dosage of oral iron preparations should be attempted; if reticulocytes or RDW rise within 4 weeks, the dosage is most likely sufficient.

INTRAVENOUS IRON

Indications

Parenteral iron therapy is recommended when oral iron therapy is ineffective. Parenteral iron treatment is indicated for the following GI diseases: (1) increased iron needs brought on by long-term hemodialysis or uncontrollable bleeding; (2) iron deficiency brought on by a gastrointestinal disorder (such as celiac disease, atrophic gastritis, or gastric bypass); (3) IBD with poor absorption, intolerance, and inefficient erythropoiesis (also mentioned below); (4) severe anemia and a reluctance to accept transfusions; (5) severe anemia and a reluctance to accept transfusions; (6) Iron stores must be quickly restored (e.g., pre-operative) However, others claim that following a gluten-free diet for six to twelve months can cure celiac disease and (7) potentially in restless legs and associated conditions. It should be noted that the US FDA currently only permits parenteral iron sucrose therapy for patients with renal failure who are receiving concomitant epoeitin therapy and are either undergoing dialysis or pre-dialysis.

Preparations

Iron gluconate, iron sucrose, iron dextran, and ferric carboxymaltose are the forms of intravenous iron preparations that are available. Ferric gluconate (Ferrlecit) works well for both IDA patients without renal illness and IDA patients receiving hemodialysis. Those who experience hypersensitive reactions to ferric gluconate may be administered iron sucrose (Venofer). Iron sucrose dosages are reduced for hemodialysis patients. Additionally, iron sucrose seems to be a safe and efficient substitute medication that can help pregnant and postpartum women with IDA quickly replenish their iron stores. Iron dextran (INFeD, DexFerrum) has a higher molecular weight than ferric gluconate and iron sucrose, and it releases iron more slowly so that it can be bound by transferrin and supply the bone marrow. Due to these characteristics, it has traditionally had the benefit of being able to be given in large doses (200–500 mg), meeting the patient's whole iron needs with a single administration (total-dose infusion), which lowers costs and increases patient compliance. However, in addition to side effects like fever, myalgia, arthralgia, hypotension, nausea, and vomiting, a lower test dose is currently advised because of reports of severe anaphylactic reactions. Iron dextran is therefore seldom used since it is discouraged in adults and contraindicated in children. Total-dose or high-dose iron sucrose infusions, which are similarly efficacious and linked to less toxicities, have recently been proposed as a potential substitute for total-dose iron dextran delivery.

Ferric carboxymaltose (Ferinject R) is a new intravenous iron preparation that can be given at infusion rates significantly greater than those of iron sucrose, and in high single doses (up to 1,000 mg iron per week). Iron deficiency anemia patients with IBD are among the various patient categories for which phase III trials have provided evidence of its safety and effectiveness to date[23].

CONCLUSION

IDA is still a significant and prevalent condition. Enhancing access to contemporary IV iron therapies, improving the availability of appropriate oral formulations, raising clinician and public awareness of the issue, and conducting prospective studies of the epidemiology, investigation, and treatment of IDA are all important ways to lessen the burden of the disease and help develop best practice guidelines[24].

Iron is essential to the body's metabolic processes. The elevated iron need linked to typical menstrual loss is mainly unmet, and ID and IDA are extremely common worldwide. Since ID and IDA are more likely to occur in women with HMB, we must carefully assess and look into our patients for the likelihood of these illnesses and, if necessary, start iron therapy. Additionally, it's critical to properly manage anemia and its underlying cause[25].

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