

## A Review on Iron Deficiency Anemia

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### ABSTRACT

Iron deficiency and the resulting anemia occur worldwide. Determining that iron deficiency is the sole factor or contributor to anemia is critical to initiating appropriate treatment. Signs and symptoms of iron deficiency are subtle and nonspecific. If there is no inflammation, a TSAT < 15% and a ferritin < 30 ng/ml indicate an iron deficiency. Inflammation changes these parameters by affecting transferrin and ferritin. Myeloid erythroblast inhibits liver-derived hepcidin via erythroferron to optimize iron mobilization and absorption when iron is required for Hb synthesis. Availability of iron at the level of the bone marrow is best assessed by the reticulocyte hemoglobin content or the percent of hypochromic cells. Treatment of iron deficiency must correct the underlying cause. Oral therapy, even at doses <100 mg/day are effective and produce less side effects than higher doses. IV iron should be reserved for iron-refractory iron-deficient anemia and that accompanying chronic inflammation. The role of iron in congestive heart failure and in some myopathic states is still being evaluated.

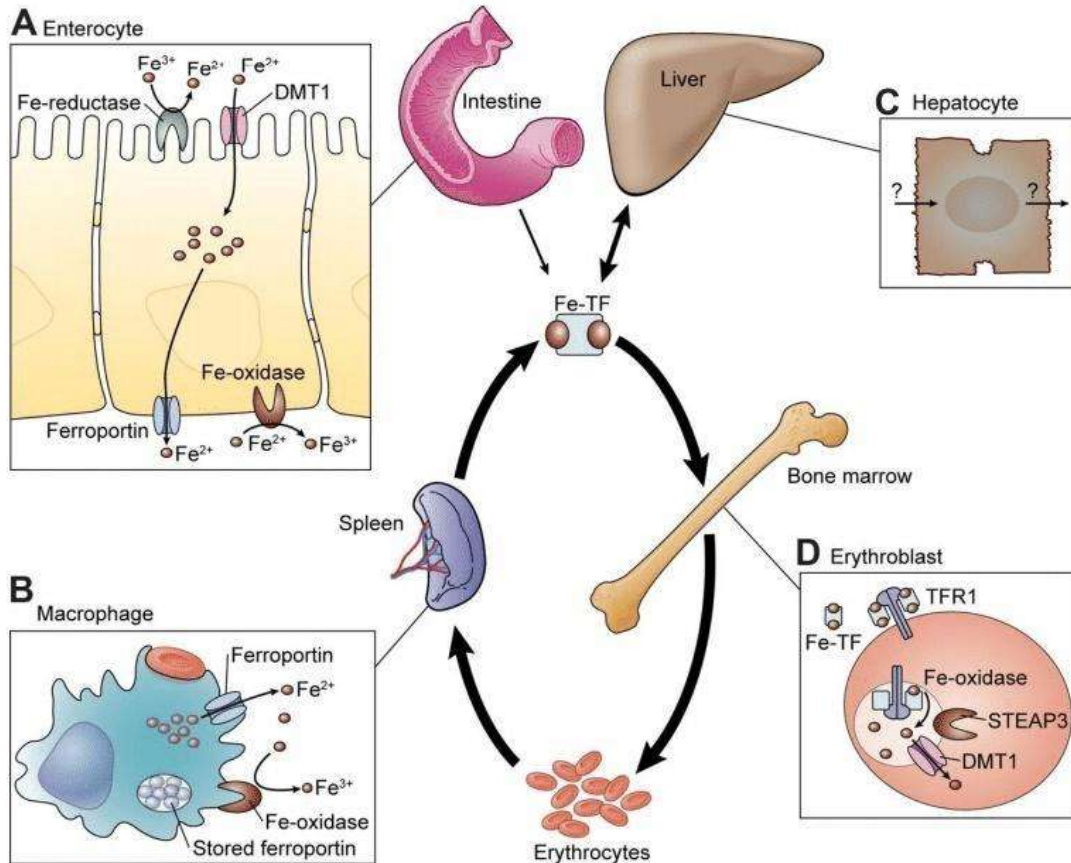
**KEYWORDS-** Iron, Anemia, iron balance; iron deficiency; iron deficiency anemia; iron supplementation; laboratory testing; pregnancy.

### I. INTRODUCTION

Iron makes up 5% of the earth's crust. Its redox states make iron useful for developing

biological processes. More and more biomolecules that bind or incorporate iron are being cataloged based on their structural similarities. Four general categories of proteins include iron :

(1) mononuclear iron proteins (e.g. superoxide dismutase ), (2) diiron carboxylate proteins (e.g. ribonucleotide reductase , ferritin), (3) iron-sulfur proteins (e.g.B. aconitase) and (4) heme proteins (e.g. hemoglobin). Of these four categories, the first three protein groups are detected in the lowest amounts but are functionally important. Hemoglobin is the most abundant iron-containing protein in humans. More than half of the iron contained in the entire organism is contained in hemoglobin. Based on the localization of hemoglobin in erythrocytes, it can be concluded that anemia is a feature of iron deficiency. Despite the abundance of iron on Earth, iron deficiency is extremely common in humans and is the most common cause of anemia worldwide. To better understand iron deficiency anemia, you need to pay attention to the concepts of iron supply and demand in red blood cell production. Iron requirements associated with erythropoiesis arise from three variables: tissue oxygenation, erythrocyte turnover, and erythrocyte loss due to bleeding. Tissue oxygen demand and erythrocyte production generally remain stable throughout adulthood in the absence of bleeding, disease, or reduced physical activity. Therefore, iron homeostasis



**Fig-Iron Homeostasis IN Humans**

In figure- 20 mg of iron is recycled daily between circulating transferrin (Fe- Tf) and erythrocytes. This recycling pathway is supported by (A) intestinal iron absorption, (B) erythrophagocytosis, (C) iron accumulation in the liver, and (D) incorporation of iron into hemoglobin.

Abnormalities in iron homeostasis and iron metabolism have also been suggested to be involved in RLS (Connor, 2008). MRI studies have shown that in the brains of RLS patients, there is less iron in the substantia nigra, an iron-enriched area of the brain, compared to controls (Allen et al., 2001). Low ferritin levels or high transferrin levels in serum or cerebrospinal fluid indicate systemic iron deficiency. Decreased ferritin levels were observed in RLS patients compared to controls (Earley et al., 2000; Mizuno et al., 2005). In addition, intravenous iron administration has resulted in symptom improvement in some patients with RLS (Bhandal and Russell, 2006; Earley et al., 2005). These results suggest iron deficiency in the

brain, which may play a key role in the pathogenesis of RLS. Iron also appears to regulate the function of the dopamine system.

**MAIN CAUSES OF IRON DEFICIENCY ANEMIA**

**BLOOD LOSS-**

Presslackfrailtyhappens when your body doesn't have sufficientpress to create hemoglobin. Hemoglobin is the portion of ruddy blood cells that gives blood its ruddycolor and empowers the ruddy blood cells to carry oxygenated blood all through your body. If you aren't expendingssufficientpress, or in the event that you're losing as well much press, your body can't deliversufficient hemoglobin, and pressinsufficiencyfrailty will inevitably develop. Causes of presslackiron deficiency include: Blood misfortune. Blood contains pressinsideruddy blood cells.Soin case you lose blood, you lose a fewpress.

**AN INABILITY TO ABSORB IRON-**

Ladies with overwhelming periods are at chance of pressinsufficiencyiron deficiencysince they lose blood amidmonthly cycle. Moderate, unremitting blood misfortuneinside the body — such as from a peptic ulcer, a hiatal hernia, a colon polyp or colorectal cancer — can cause pressinsufficiencyfrailty. Gastrointestinal dying can result from normalutilize of a few over-the-counter torment relievers, particularly aspirin. A need of press in your count calories.Your body frequently gets press from the nourishments you eat. In the event that you expendas wellsmallpress, over time your body can ended uppressinsufficient. Cases of iron-rich nourishmentsincorporate meat, eggs, verdant green vegetables and iron-fortified nourishments.

**PREGNANCY-**

For legitimatedevelopment and advancement, newborn children and children requirepress from their diets, too. An failure to retainpress.Press from nourishment is retained into your circulation system in your littledigestive system. An intestinal clutter, such as celiac illness, which influences your intestine's capacity to assimilatesupplements from processednourishment, can lead to pressinsufficiencyfrailty. On the off chance thatportion of your littledigestive system has been bypassed or evacuated surgically, that willinfluence your capacity to retainpress and other nutrients. Pregnancy. Without press supplementation, presslackfrailtyhappens in numerous pregnant women because their press stores ought to serve their claimexpanded blood volume as well as be a source of hemoglobin

**DIAGNOSIS –****Differential Diagnosis**

The differential diagnosis of iron deficiency anemia include:

- Lead poisoning
- Microcytic anemia
- Anemia of chronic disease
- Hemoglobin CC disease
- Hemoglobin DD disease
- Autoimmune hemolytic anemia
- Hemoglobin S-beta thalassemia

**Blood tests**

The complete blood count is one of the most common blood tests. It's often done as part of a routine checkup. This test measures many different

parts of your blood, including red blood cells, white blood cells, and platelets.

- **Red blood cell levels** that are higher or lower than normal could be a sign of anemia. Red blood cells carry oxygen from your lungs to the rest of your body.
- **Hemoglobin levels** that are higher or lower than normal may be a sign of anemia. Hemoglobin is an iron-rich protein in red blood cells that carries oxygen.
- **Hematocrit levels** that are too low may be a sign of anemia. Hematocrit is a measure of how much space red blood cells take up in your blood.
- **Mean corpuscular volume (MCV) levels** that are higher or lower than normal may be a sign of anemia. MCV is a measure of the average size of your red blood cells.

**Herbs and Supplements for anemia**

- Astragalus.
- Copper.
- Dong Quai.
- Iron.
- Spirulina.
- Stinging Nettle.
- Vitamin B12 (Cobalamin)
- Vitamin B2 (Riboflavin)

**Oral iron therapy and its limitations**

Traditionally hemodynamically stable patientswith iron deficiency anemia resultant fromchronic blood loss from the gut are prescribedoral iron therapy. The two categories of iron sup-plements are those containing the ferrous form ofiron and those containing the ferric form of iron.The most widely used iron supplements are thosethat contain the ferrous form of iron given that itis the better absorbed of the two. The three commonly administered types of ferrous iron supplements: ferrous fumarate, ferrous sulfate, andferrous gluconate, which differ in the amount ofelemental iron (the form of iron in the supple-ment that is available for absorption by thebody), and contain 33%, 20%, and 12% iron,respectively (NIH, 2010). Recent studies havesuggested that these iron preparations are essen-tially equivalent in terms of bioavailability[Harrington et al. 2011; Navas-Carretero et al.2007; Lysionek et al. 2003]. The recommendeddaily dose of treatment by the Centers forDisease Control and Prevention (CDC) rangesfrom 150 mg/day to 180 mg/day of

elemental iron administered in divided doses two to threetimes a day [CDC, 1998].

#### Adverse effects

As previously discussed, the common adverse effects of oral iron are well known among healthcare professionals and patients. The potential adverse effects of intravenous iron have more recently been publicised as they become further researched and understood. The rare adverse effect of hypersensitivity reactions has been known for some time and have dictated specialised protocols and training for healthcare professionals routinely administering intravenous iron.

A less commonly recognised adverse effect is that of extravasation of intravenous iron that can cause long-lasting tattoo-like skin discolouration preceded by skin irritation and pain at the injection site. Though this adverse effect is considered to be rare (occurring at a rate of approximately 1.6%), the skin staining can last for several months after the initial infusion despite pharmacological interventions to resolve the reaction.

## II. CONCLUSION

IDA is a major public health problem. Coordinated efforts should be made to control anaemia. Recognised risk factors should be considered in prevention and control strategies of IDA. Although predisposing factors for anaemia were documented, large scale studies should be done to identify specific aetiologies and root causes of anaemia among the groups by assessing micronutrients (serum iron, folate, and vit-b12 levels).

#### Data Availability

\*e data used to support the findings of this paper are upon request to the corresponding author. Conflicts of Interest \*e authors declare that there are no conflicts of interest regarding the publication of this paper.

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