Bio-Science Research Bulletin
http://www.bpasjournals.com

Review Article

Print version ISSN 0970 0889
Online version ISSN 2320 3161
DOI: 10.5958/2320-3161.2020.00007.3
Vol. 36 No. 1, January-June 2020: P.50-54

Nutritional Approach for the Treatment of Anemia and Iron Deficiency Anemia

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Received on 25.02.2020
Accepted on 13.05.2020

ABSTRACT

Around the globe every year around ten million lives are lost due to hunger and undernutrition. Multitudes of people are impinged on by "hidden hunger," a lack of essential vitamins and minerals, which is known as micronutrient deficiencies. Worldwide till date iron deficiency remains as the top-ranking source of anemia. The consequences of poor nutrition are not always prominent to notice. Infants, young children and women of child- bearing age are those at greatest risk of nutritional anemia besides the men. Over 30% of the world's population – are anemic with about 1 billion suffering from iron deficiency anemia (Gleason G.). The etiology of ID and IDA is complex and has a prolonged imbalance between dietary intake, absorption and body needs which in the long run leads to iron deficiency anemia. However this article focuses on the nutritional approach for the balance of iron absorption in the body thereby reducing the occurrence of ID and IDA.

KEYWORDS: Anemia, Iron deficiency anemia, Dietary Iron, Absorption, Nutrition.

I. INTRODUCTION

The issue of iron is global. Anemia is a widespread public health issue related with an elevated threat of morbidity and mortality in the young and the old, in men and women and very specially among the pregnant women and young children (Banjari, 2018). Among the numerous factors, both nutritional (such as vitamin and mineral deficiencies) and non-nutritional (such as infection and hemoglobinopathies) contribute to the onset of anemia. The iron deficiency (ID) continues to be the primary reason for the cause of iron deficiency anemia (IDA).

This review reconsiders iron deficiency and its anemia in light of nutrition and its contribution and examines causes, and treatment options in terms of nutrition

II. DEFINITION

Iron deficiency occur when the reticuloendothelial system (RER) iron storage is run down or in other words iron deficiency can be defined as a state of iron-poor erythropoiesis wherein the increased demand is met with an inadequate mobilization of iron from stores, even after treatment with erythropoiesis stimulating agents (Camaschella C, 2015). When the red cell number, blood hemoglobin concentration and hematocrit fall below the normal range, iron deficiency anemia develops (Means R. T., 2019).

III.a IMPORTANCE OF IRON

Iron is an essential constituent of virtually all living cells, and undoubtedly it is an important requisite for all human cells. The interconversion between two thermodynamically stable oxidation states of iron, the ferric form (Fe^{3+}) and the ferrous form (Fe^{2+}), under physiological conditions makes it superlatively suited to the catalysis of biochemical reactions. All biologic functions require iron; therefore iron plays a very pivotal role in aerobic respiration, production of energy, DNA synthesis, and proliferation of cell (Hentze MW, 2010). Even the final step of hemoglobin formation demands Iron; the incorporation of iron into protoporphyrin is assisted by ferrochelatase which results in the formation of hemoglobin (Cornah J.E, 2002). Therefore, the body makes insufficient quantity of hemoglobin in red blood cells whenever there is a deficit in iron levels, which in turn will hinder the delivery of oxygen to various organs and tissue throughout the body. As a result microcytic anemia (small red blood cells and low hemoglobin levels) occurs which is also referred to as iron deficiency anemia (IDA), the most widespread outcome of ID.? For any alteration to be observed in hemoglobin levels, the iron stores of the body must be depleted first. A large number of enzymes for their biological functions depend on iron. These distinguishing features make iron important, useful and beneficial for all living systems also mean that the metal is able to catalyze reactions leading to the production of toxic oxygen radicals, particularly when it is present in excess. To handle with this dual character or property of iron, cells and the body as a whole has developed, advanced, progressive and complicated features for regulating iron influx and efflux. However it is extremely essential to supply sufficient iron to cells for all their metabolic requirements (Vulpe, 2010). The human body has evolved over the years in such a way that it conserve iron by a number of ways, including the recycling of iron after the breakdown of red cells and the withholding of iron in the absence of an excretion mechanism. Nevertheless excess amount of iron can be toxic, therefore maximum amount of iron absorption is limited to 1 to 2 mg daily, and macrophages that phagocytise senescent erythrocytes provide most of the iron needed daily (about 25 mg per day) by recycling. Hepcidin hormone controls the latter two mechanisms, thereby maintaining the total-body iron within the normal range, avoiding both i.e iron deficiency and excess (Camaschella C, 2015).

III.b HEPCIDIN AND ITS ROLE IN IRON ABSORPTION

Iron acquisition in the circulation is regulated by hepcidin (Hentze M.W, 2010). Initially hepcidin was identified as liver--enhanced antimicrobial peptide--1. Actually hepcidin is a 25 amino acid peptide hormone synthesized by the liver and a component of the innate immune system. It is the major regulator of normal iron balance and plays a significant role in the pathogenesis of disorders of iron metabolism (Robert T. Means, 2019).

The duodenal enterocytes iron absorption (final step of transport) as well as macrophage recycling iron (efflux of iron, mostly spleen and bone marrow) are controlled by the hepcidin homeostatic control mechanism. Hepcidin also act as an acute- phase reactant that has a major role in regulating the fluctuations in plasma iron level which occur due to absorptive enterocytes and macrophages by binding to and inducing the degradation of ferroportin, which exports iron from the cell (Nemeth E.Tuttle MS, 2004).

In duodenum Ferrous iron is absorbed which is then transported by Divalent metal transporter -1 into the enteric mucosal epithelial cell (enterocytes). Within the cell Ferrous iron is enzymatically converted to ferric form, which then enters the circulation with the help of ferroportin. Once they are freed from enterocytes, ferric irons are shipped to other cells bound to transferrin. In the cells they remain in the stored form or for metabolism. Transferrin receptor (TfR)-1 is responsible for its uptake into the cell while export of iron from other cell is ferroportin dependent (Means R. T., 2019).

Taken into consideration enterocytes, macrophages and hepatocytes, ferroportin is the only known ligand for hepcidin (T, 2011, pp. 4425-33). The internalization and degradation of iron occur due to the binding of hepcidin to ferroportin (Means RT, 2004). Thus it can be concluded that iron will be retained in the cell in the presence of increased hepcidin level, while a decreased level of hepcidin will

cause the movement of iron into the circulation (Robert T. Means, 2019, pp. 85-92). However liver hepcidin expression escalate in response to elevated circulating and tissue levels of iron (its physiologic signal) but the liver hepcidin level also rises in response to a systemic inflammation or infection (Besarab A, 2018).

IV. NUTRITION AND ANEMIA

The most commonly recognized nutritional deficiency is Iron deficiency (ID). (Clark, 2008) Anemia which is defined as a low concentration of hemoglobin (Hb) in blood can occur due to multiple reason. There are several factors, sickness, disorders and malfunctioning which are responsible to cause anemia. The diseases which results in blood loss, presence of hookworms, intestinal infections leading to internal bleeding and blood loss and malfunction in the manufacture Hb, for that matter even non steroidal anti-inflammatory drugs (NSAIDS) and anticoagulants can cause iron deficiency anemia due to blood loss, in addition to these another major role is played by nutrition which results in anemia and iron deficiency anemia. Some vitamins like vitamin B_{12}, folic acid and riboflavin have some bearing on the making of Hb but the most significant nutritional factor remains to be iron deficiency, which also happens to be the most repeatedly happening micronutrient deficiency in both developed and less developed countries (Gleason G, 2007). Iron bioavailability is reduced by a cereal based diet because they usually possess phylates. Phylates sequester iron into a poor absorbable complex. IDA can also be resulted due to a strict vegan or vegetarian diet. Ascorbic acid or vitamin C enhances the absorption of iron. Regular blood donors are also at a high risk of iron deficiency anemia. Malabsorption can also result in anemia (Besarab A, 2018).

V. NUTRITIONAL APPROACH IN THE TREATMENT OF IRON DEFICIENCY AND IRON--DEFICIENCY ANEMIA

Particular measures to address iron deficiency anemia are among the most cost- effective public health interventions.

Three basic elements which are prevalent for the treatment of iron deficiency anemia are (a.) correction of anemia, (b.) sated of iron stores and (c.) detecting of the origin of iron deficiency. The last two elements can be applied to individuals who are iron deficient but are not anemic. Iron deficiency can be prevented effectively through nutritional approach. There occurs a variation in the bioavailability of iron in the food that is the amount of iron which can be absorbed in duodenum (Boulpaep E.L, 2006). The dietary intake reference set according to US Institute of Medicine (www.iom.edu), adult men and women over 50 years of age has the dietary reference intake of 8 mg/ day and younger adult women has the dietary intake reference of 18mg/day. Moreover the presence of ID and IDA increases the capacity of the gut for the readily absorption of dietary iron (Adamson, 2008).

A well balanced food goes hand-in-hand with iron rich foods. A slight modification in the diet like a dietary modifications such as the addition of ascorbic acid to the diet can improve the absorption of dietary iron (Diaz M., et al 2003). Iron to be absorbed in the stomach acidity is important. Nonheme iron absorbtion is restricted to duodenum (Boulpaep E.L, 2006). The ferric form of the nonheme iron (Fe^{3+}) requires a very low pH i.e, it requires an acidic environment in the stomach to form complex salts with anions, while the ferrous (Fe^{2+}) form of the nonheme is soluble even to pH 8 (Lynch, 1997).

Cereals and its products can be considered as an important food source to increase the iron stores of the body (Thompson, 2007). Various studies have proved that plant source also is a good dietary source of iron (Black M.M. *et al.* , 2011).

There is no doubt that meat is a rich source of dietary iron, however it has to be brought to the notice that red meat is actually the real source of iron and not the white meat like chicken (Banjari I *et al.*, 2013). Nevertheless there is a definite influence on iron absorption by the meat factor effect as well as amino acids with sulphur (Reddy M.B, 2007).

It is also found that alcohol has a significant effect on the absorption of ferric iron but do not contribute in the absorption of ferrous iron. This property is attributed due to the enhanced secretion of gastric acids. However effect of wine differs in this area (Hallberg L, 2000).

Vitamin A has an antagonistic effect on polyphenols (present in tea and coffee) and phylates (present in cereals) thereby making the absorption of iron available in duodenum at pH 6 by forming a complex. For that matter beta-carotene and other carotenoids such as lycopene, lutein and zeaxanthin are found to have a stronger effect than vitamin A (I.Banjari, 2012).

Food that act as inhibitors for iron absorption- Not all food support for iron absorption or are rather loaded with iron. There are certain diets which act as iron inhibitors and does not support iron absorption. Phylates which is an important constituent of cereals are found to have inhibitory effect on the absorption of dietary iron; therefore they need to be taken with special care (Kristensen M.B., 2005). Consumption of tea and coffee reduces iron absorption due their polyphenol property. It was found that tea had a higher inhibiting effect of around 75-80% for cca 200 ml while coffee had a lower reduction level of 60% for cca 150 ml. (Hurrell R.F, 1999). Interestingly consumption of calcium higher than 600mg/day was also found to have a negative impact on the absorption of iron (Lynch, 2000).

However it is also a matter of fact that nutritional approach alone is not a competent approach to rectify iron deficient state (Gracia O.P, 2003). Hence cannot depend only on nutrition rather also have to depend on pharmacological iron replacement like oral iron replacement, intravenous iron replacement to combat iron deficiency and iron deficiency anemia.

AUTHOR'S ACKNOWLEDGMENT

The authors are grateful to S.P.C Government College, Ajmer and Sophia Girls' College, (Autonomous), Ajmer for all the help and support provided to carry out the research.

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