THE PHYSIOLOGY, EPIDEMIOLOGY, CLINICAL MANIFESTATIONS, LABORATORY DIAGNOSIS, AND MANAGEMENT OF IRON DEFICIENCY AND IRON DEFICIENCY ANAEMIA IN CHILDREN.

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Abstract. *The purpose of this review article is to explain the physiology, epidemiology, aetiology, clinical effects, laboratory diagnosis, and management of iron deficiency and iron deficiency anaemia in children.*

Keywords*: Iron; iron deficiency; iron deficiency anaemia; children; treatment*

INTRODUCTION

One of the most prevalent nutritional disorders in the world is iron deficiency. The proper operation of key processes and reactions involving electron transport depends on a sufficient supply of iron. Polycythaemia and iron consumption are brought on by hypoxia and the subsequent erythrocytosis that follows. The propensity to bleed due to thrombocytopenia and anomalies in the haemostatic system can also result in iron deficiency.

Iron depletion, iron deficiency, and iron deficiency anaemia are the three stages that occur when the body's iron stores are diminished. The body gradually reduces its iron stores when iron depletion occurs because the body needs more iron than is consumed. A low serum ferritin content indicates a decrease in iron reserves. Iron deficiency is characterised by low stored iron, low absorption of iron to replenish normal bodily losses, and low levels of serum ferritin, mean corpuscular haemoglobin (MCH), and mean corpuscular volume (MCV). Iron deficiency anaemia is the final and most severe stage, which is typified by lower iron levels in red blood cells (RBCs), low mean MCV, low mean MCH, low haemoglobin (Hb) levels, and decreased serum ferritin. It's critical to distinguish between "anaemia" and "iron deficiency." Acute inflammatory conditions, chronic inflammations, genetic polymorphisms, and sickle cell disease states can all affect the sensitivity and specificity of several laboratory tests used to detect iron deficiency, including MCV, serum ferritin, and transferrin saturation (Tfsat). As a result, these tests have limited utility. To determine the iron status of a population, the World Health Organisation (WHO) advised combining a number of laboratory tests.

Methods. Search engines such as PubMed, Medline, Web of Science, Psych Info and CINAHL, Google, JSTOR ARCHIVES, EBSCO HOST, Ohio LINK, DOABOOK, Access to Research for Development and Innovation (ARDI), and Health Inter Network Access to Research Initiative (HINARI) were used. Iron deficiency in children, iron deficiency anaemia in children, and latent iron deficiency in children were among the keywords that were utilised. Textbooks, journals, periodicals, newspapers, policy documents, scholarly papers, conference papers, and online resources such as dictionaries, encyclopaedias, abstracts, and reviews were among the items used. The purpose of this review article is to

provide an overview of the physiology, epidemiology, aetiology, clinical manifestations, laboratory diagnosis, and management of iron deficiency and iron deficiency anaemia in children.

Iron physiology. One cation required for the production of blood is iron. It is also a component of haemoglobin and is required for a number of cytochrome and enzyme processes. It is dispersed both in storage pools and as an active metabolite. In humans, iron is efficiently recycled from aged red blood cells. Only 10% of the iron in food is absorbed by the small intestine. Iron must be consumed daily to replace the iron lost through intestinal and skin cell desquamation and to support children's growth. During times of strong growth, such as infancy and adolescence, the need for iron is greatest. How well iron is absorbed depends on its shape when consumed. When compared to the non-haem condition, it is more readily absorbed in the haem form. The non-haem form needs to be reduced to the ferrous state so that the stomach acids can release it from food binders. Additionally, foods like calcium, tea tannins, and vegetable fibre phytates in grains and pulses decrease the absorption of non-haem iron. Iron absorption is promoted by certain other foods, such as vitamin C. The liver produces a binding protein called transferring, which is responsible for moving the absorbed iron. The iron level has an impact on the liver's production of transferrin. Iron deficiency causes an increase in synthesis, while chronic illness states cause a decrease. Hemostasin or ferritin are the two forms of iron storage. During erythropoiesis, it is utilised. Ferritin is soluble and widely accessible; it is stored in bone marrow, serum, RBCs, hepatocytes, and macrophages (spleen). The total amount of ferritin stored in the body is equal to the amount in circulation.

There are three primary stages to the reduction of bodily iron: iron depletion, which is the initial stage, is followed by iron deficiency without anaemia.

The metabolism of iron. Iron has a crucial role in almost every living cell, including human cells. Under healthy settings, iron is ideally suited for the catalysis of a variety of biological activities due to its capacity to transition between two thermodynamically stable oxidation states: the ferric (Fe3+) form and the ferrous (Fe2+) form. Iron is necessary for the activity of a large number of enzymes. These include nitrogen fixation, oxygen activation, storage, and electron transfer; deoxyribonucleotide synthesis from ribonucleoside diphosphates; and detoxification of activated oxygen species. It is a crucial nutrient involved in energy metabolism, immunological response, and brain development. Iron is firmly attached to proteins (transferrin) during the processes of absorption and distribution, leaving very little free intracellular iron. Controlling intracellular iron is crucial since even tiny amounts of "free iron" can seriously harm many cellular components, such as membranes and DNA.

Iron deficiency stages. Iron deficient erythropoiesis and iron depletion are the two phases of iron shortage without anaemia. A person with iron deficiency has no iron stocks to draw from in case their body need extra iron since iron depletion lowers stored iron levels without affecting necessary iron levels. Iron-deficient erythropoiesis occurs when stored iron is depleted and transport iron is further reduced. The amount of iron that is absorbed is insufficient to make up for the iron that is lost or needed for development and bodily

functions. The reduction of iron (storage and transport) results in underproduction of iron-containing molecules that are necessary for function, such as haemoglobin, in iron deficiency anaemia, the most severe form of iron deficiency.

Iron deficiency's clinical consequences. Additionally, research has shown that even after iron replenishment, early-life iron insufficiency might have long-lasting cognitive impacts. In a long-term study, Lozoff et al. examined the possibility that newborns with iron deficiency would be "functionally isolated." He contrasted the conduct of 52 Costa Rican infants with iron deficiency, who were between the ages of 12 and 24 months, with that of a control group that had a higher iron status. In addition to testing them using the same motor and mental protocols, the researcher watched them while they played freely. Babies with iron deficiency made less attempts at test objects, were less playful, reluctant and wary, and paid less attention to instructions. The authors' theory that newborns with iron deficiency interact with their surroundings less is supported by this finding. Over a ten-year period, the authors also found that children with ID had poor socioemotional function and persistent cognitive impairment.

Iron deficiency has been linked to decreased work ability. Iron-containing cytochromes, iron-sulfur proteins, and electron transport proteins are necessary for the oxidative generation of energy in the muscles' mitochondria during prolonged muscular activity.

Handling iron deficiency. Oral iron salts, usually over-the-counter ferrous sulphate, are used to treat iron deficiency because they are reasonably priced and well absorbed. The elemental iron is used to determine dosages; children are given 3–6 mg/kg daily, whereas adolescents are given 60 mg/dose. If the iron deficit is dietary, the response to iron therapy is usually quick. Intramuscular iron injections are typically inappropriate; if oral iron is not tolerated, parenteral form is used. If hypovolemia or severe cardiovascular impairment are present, erythrocyte transfusion is recommended. If the anaemia is corrected quickly, cardiac dilatation may occur.

After a month of treatment, the haemoglobin level should be checked again. The diagnosis of iron deficient anaemia is confirmed by a rise of 1 g/dL or more. If Hb doesn't improve, more laboratory tests, such as MCV, RDW, and serum ferritin, should be performed to assess the anaemia further and look for potential causes of blood loss. After Hb has recovered to normal, iron therapy should be continued for another two to three months. After stopping iron therapy, haemoglobin levels should be checked again around six months later. According to current standards, individuals with iron deficiency who are cyanotic should be treated cautiously and with continuous monitoring of their haemoglobin levels, and unnecessary venesection should be avoided.

Conclusion. Iron deficiency and iron deficiency anemia remain significant public health concerns, especially among children, who are particularly vulnerable due to their rapid growth and developmental needs. Early identification and intervention are crucial to prevent the long-term cognitive and physical repercussions associated with these conditions. A comprehensive approach, combining dietary management, appropriate supplementation, and regular monitoring, is essential for effective treatment. By improving awareness and

understanding of iron metabolism and deficiency stages, healthcare providers can enhance the quality of care for affected children, ultimately promoting better health outcomes and development. Addressing iron deficiency proactively can lead to substantial improvements in children's overall well-being and quality of life.

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