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Salahaddin University-Erbil

Iron and anemia

Research project

Submitted to the Chemistry department-College of education – Salahaddin University in partial fulfillment of the requirements for the degree of (BSc.) in chemistry

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May -2022

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Acknowledgments

At the first. I would like to express my thanks to God forgive a chance and opportunity for reach this level and preparing research project. I would like to thanks salahaddin university, which is always helpful for everything. And I want to thanks a bunch for my parents who are always supporting and helping me. I would like to thanks my dear supervisor Mrs. Lutfia Hassan for her invaluable instructions along with constant guidance and encouragement.

Finally, I want to thank all of those people who, once upon a time, were my teachers and who helped me begin this long journey.

CERTIFICATE:

This research project has been written under my supervision and has been submitted for the award of the BSc. degree in Chemistry with my approval as a supervisor.

Name: Lutfia Hassan

Date: / /2022

Signature

A handwritten signature in black ink, appearing to be 'Lutfia Hassan', written over a horizontal line.

Abstract

In our bloodstream, there are plenty of red blood cells (RBC) , which function as an important oxygen carrier in our bodies. Each RBC consists of millions of hemoglobin (Hb), which is made up from globin and iron. If any deficiency or malfunction of any globin, it will lead to anemia as indicated in low Hb level while iron deficiency anemia (IDA) is anemic due to the lacking of iron as indicated in low Hb and ferritin levels. Iron deficiency anemia is the most common cause of anemia worldwide and results from inadequate iron supply for erythropoiesis. Iron deficiency is most prevalent during periods of rapid body growth in infancy and again at puberty. Insufficient intake accounts for most cases. The clinical manifestations of iron deficiency anemia can be subtle, but irreversible delayed psychomotor development may occur if the anemia is severe and prolonged.

Key word: Iron , Deficiency , Anemia

1- Introduction

Iron is a metal, an element of group VIII of the periodic table (Fig.1). It is a lustrous, ductile, malleable, silver-grey in colour. It is the tenth most abundant element in the universe. It is found in a major amount at the core of the Earth in a molten form (Pascual, et al., 2012)..

The electron configuration of iron is ([Ar] 3d⁶ 4s²), The body of an adult human contains about 4 grams (0.005% body weight) of iron, mostly in **hemoglobin** and **myoglobin**. These two proteins play essential roles in vertebrate metabolism, respectively oxygen transport by blood and oxygen storage in muscles (Kumar, et al., 2015). To maintain the necessary levels, human iron metabolism requires a minimum of iron in the diet. Iron is also the metal at the active site of many important redox enzymes dealing with cellular respiration and oxidation and reduction in plants and animals (Kumar, et al., 2015).

Table (1): show some information about iron metal.

Atomic Number	26
Atomic Symbol	Fe
Atomic Weight	55.845
Electron Configuration	([Ar] 3d ⁶ 4s ²)

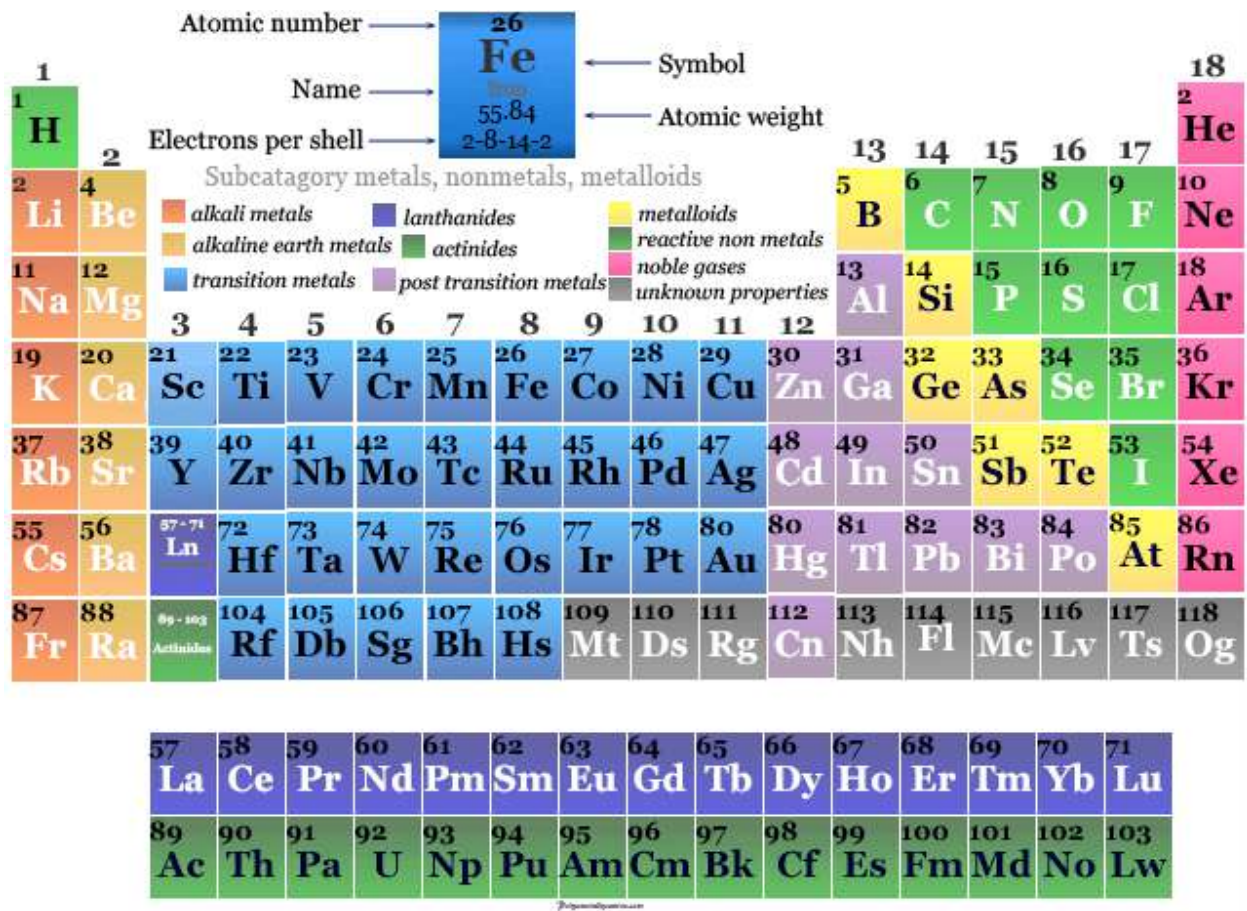


Figure (1): shows iron in the periodic table

1.1 History of Iron

Iron objects have been found in Egypt dating from around 3500 BC. They contain about 7.5% nickel, which indicates that they were of meteoric origin. The ancient Hittites of Asia Minor, today's Turkey, were the first to smelt iron from its ores around 1500 BC and this new, stronger, metal gave them economic and political power. The Iron Age had begun. (W. M. Haynes, et al 2015) Some kinds of iron were clearly superior to others depending on its carbon content, although this was not appreciated. Some iron ore contained vanadium producing so-called Damascus steel, (swordss, Kaye & Laby 2014). The first person to explain the various types of iron was René Antoine Ferchault de Réaumur who wrote a book on the subject in 1722 (J. S. Coursey, D. J. Schwab 2016). This explained how steel, wrought iron, and cast iron, were to be distinguished by the amount of charcoal (carbon) they contained. The Industrial Revolution which began that same century relied extensively on this metal. (T. L. Cottrell 1954)

1.2 Iron absorption

Iron is an essential element of various metabolic processes in humans, including DNA synthesis, electron transport, and oxygen transport. Unlike other minerals, iron levels in the human body are controlled only by absorption. (Andrews NC. 2000). Additionally, approximately 2.2% of total body iron is found in the so-called labile pool, a poorly defined and reactive pool of iron that forms reactive oxygen species via the Fenton Reaction. (Banerjee, D., Flanagan1986,)

The average adult stores about 1 to 3 grams of iron in his or her body. An exquisite balance between dietary uptake and loss maintains this balance. About 1 mg of iron is lost each day through the sloughing of cells from the skin and mucosal surfaces, including the lining of the gastrointestinal tract (Cook et al., 1986) and (Gibson et al., 1988). Gastric acid lowers the pH in the proximal duodenum, enhancing the solubility and uptake of ferric iron (Table 1). When gastric acid production is impaired (for instance by acid pump inhibitors such as the drug, prilosec, iron absorption is reduced substantially. (Bezwoda, W. R., MacPhail,1986) and (Bothwell, T. H etal,1982). Iron absorption occurs predominantly in the duodenum and upper jejunum (Muir and Hopfer, 1985) (Figure 1). The mechanism of iron transport from the gut into the bloodstream remains a mystery despite intensive investigation and a few tantalizing hints. (Beutler, E., and Buttenweiser, E,1960)

Table 2. Factors That Influence Iron Absorption	
Physical State (bioavailability)	heme > Fe ²⁺ > Fe ³⁺
Inhibitors	phytates, tannins, soil clay, laundry starch, iron overload, antacids
Competitors	lead, cobalt, strontium, manganese, zinc
Facilitators	ascorbate, citrate, amino acids, iron deficiency

Iron enters the stomach from the esophagus. Iron is oxidized to the Fe^{3+} state no matter its original form when taken in orally. Gastric acidity, as well as solubilizing agents such as ascorbate, prevent precipitation of the normally insoluble Fe^{3+} . Intestinal mucosal cells in the duodenum and upper jejunum absorb the iron. The iron is coupled to transferrin (Tf) in the circulation which delivers it to the cells of the body. Phytates, tannins and antacids block iron absorption.

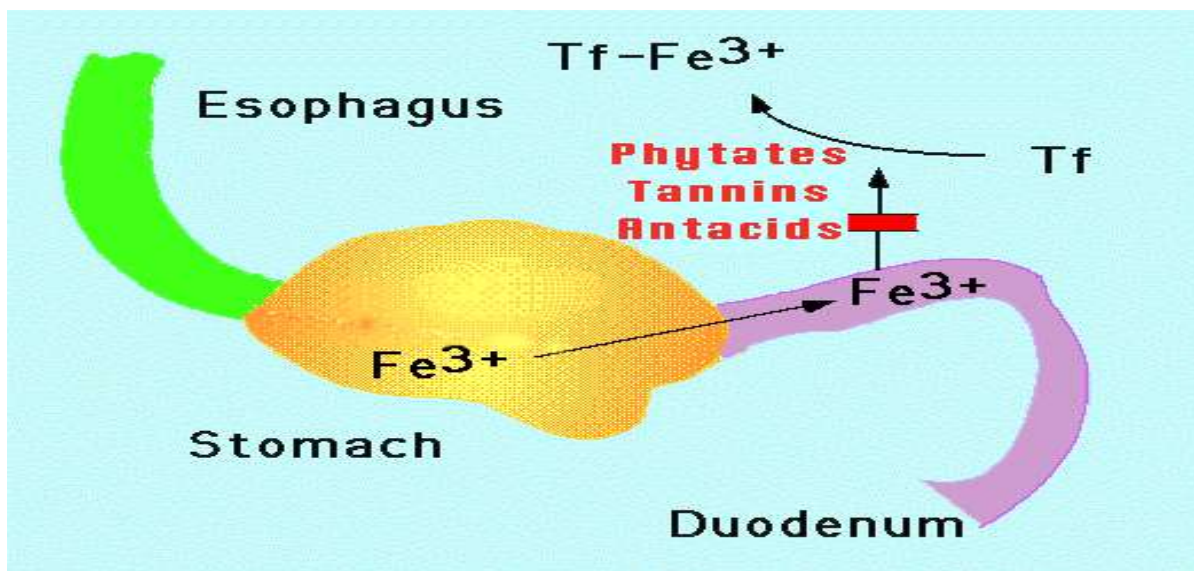


Figure (2): show Iron absorption

1.3 Iron Homeostasis Mechanism

In circulation, iron exists in two forms: heme and non-heme iron. The majority of non-heme iron is bound to transferrin, which is capable of binding two molecules of ferric iron. Adults have approximately 3 mg of circulating non-heme iron, only saturating 30% of transferrin binding sites. However, transferrin cannot cross the tight junctions of the blood-brain barrier (BBB); therefore, cells that comprise the BBB must import iron and transfer it into neural tissue. Iron-laden transferrin binds to the transferrin receptor (TfR) on the cell surface, and this complex is internalized into endosomes (Sipe and Murphy, 1991). At low pH, iron is released and exported by endosomes for use, storage in ferritin, a multisubunit protein consisting of H- and L-chains (Aisen et al., 2001) or export. Upon iron delivery, transferrin is recycled to the surface (Hunt and Davis, 1992).

Ferroportin exports ferrous iron that is not utilized or stored in the cell (Abboud and Haile, 2000; Donovan et al., 2000; McKie et al., 2000), but exported iron must be oxidized to be accepted by transferrin in the circulation. Oxidation of iron is accomplished by the ferroxidases such as ceruloplasmin, hephaestin (Heph), amyloid precursor protein (APP) or zyklopen (Chen et al., 2010). These enzymes are crucial for iron export and when disrupted result in cellular iron accumulation and degeneration. Ceruloplasmin, a copper-binding protein, contains over 95% of copper found in plasma. In Alzheimer's disease, iron export by APP is inhibited by elevated extracellular Zn²⁺ dissociating from β -Amyloid. The APP domain responsible for ferroxidase activity has been debated (Ebrahimi et al., 2012). In 2010, zyklopen was identified as another multicopper ferroxidase and plays a role in placental iron transport. (Abboud, S., and Haile, D. J. 2000). Immunostaining of zyklopen identified its expression in the brain, kidney, testes and retina but not in the liver or intestine (Chen et al., 2010). Our unpublished data also suggests that zyklopen is expressed in brain and neural retina, but not RPE cells.

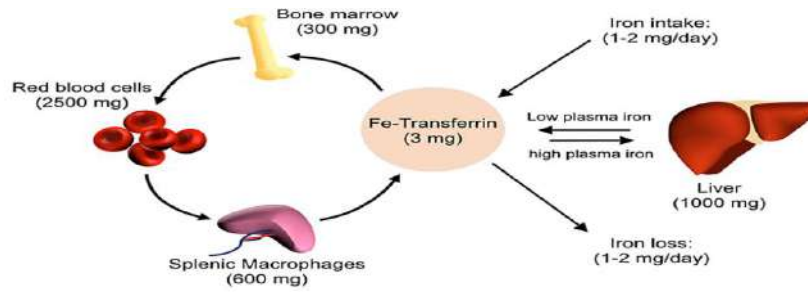


Figure (3): show hemostasis mechanism

1.4 Metabolism

Iron is transported by a globulin of the serum to and from the various tissues of the body to satisfy their metabolism. Surplus iron carried by this iron-binding protein is deposited chiefly in the liver. Storage iron may be increased in two ways. The first mechanism results from the inability of the body to excrete significant amounts of iron. Because of this, any decrease in circulating red cell iron (any anemia other than blood loss or iron deficiency anemia) is accompanied by a shift of iron to the tissue compartment. The total amount of body iron remains constant and is merely redistributed. (Lozoff B .2011)

This is to be contrasted with the absolute increase in body iron and enlarged iron stores which follow excessive iron absorption or parenteral iron administration. Enlarged iron stores in either instance may be evaluated by examination of sternal marrow or determination of the serum iron and saturation of the iron-binding protein. In states of iron excess, differences in initial distribution are observed, depending on the route of administration and type of iron compound employed. Iron absorbed from the gastrointestinal tract and soluble iron salts injected in small amounts is transported by the iron-binding protein of the serum and stored predominantly in the liver. (Peirano PD Algarín 2009)

The normal iron circuit includes the uptake of iron from transferrin by developing erythroblasts, the incorporation of iron into heme, red blood cell (RBC) production, RBC survival, and RBC senescence in the spleen with a return of iron to the bone marrow via transferrin (Figure 1).

Uptake of iron from the diet is necessary to replace the small amount of iron normally lost daily via the sloughing off of intestinal mucosal cells and menstrual blood loss in women of reproductive age. (CR Chamorro R et al. 2009)

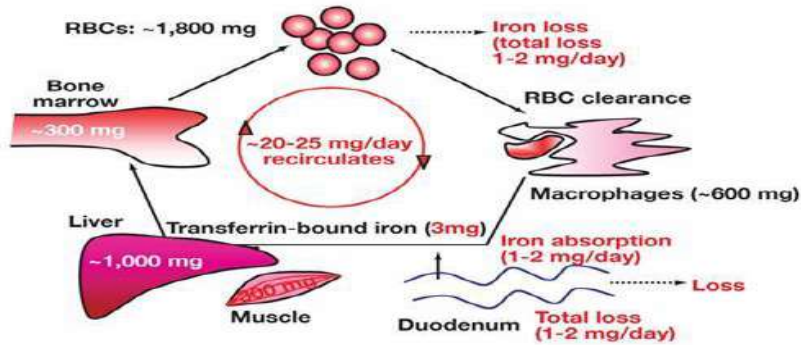


Figure (4): show Metabolism of iron

1.5 Function of iron in human body

Iron helps oxygenate the blood

Iron boosts the immune system

Iron helps convert blood sugar to energy

Iron aids cognitive function

Iron supports healthy skin, hair and nails

(Braun, L and Cohen, M; 2007)

1.6 What is hemoglobin

Hemoglobin is the protein molecule in red blood cell that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs.

Hemoglobin is made up of four protein molecules (globulin chains) that are connected together. The normal adult hemoglobin (abbreviated Hgb or Hb) molecule contains two alpha-globulin chains and two beta-globulin chains. In fetuses and infants, beta chains are not common and the hemoglobin molecule is made up of two alpha chains and two gamma chains.

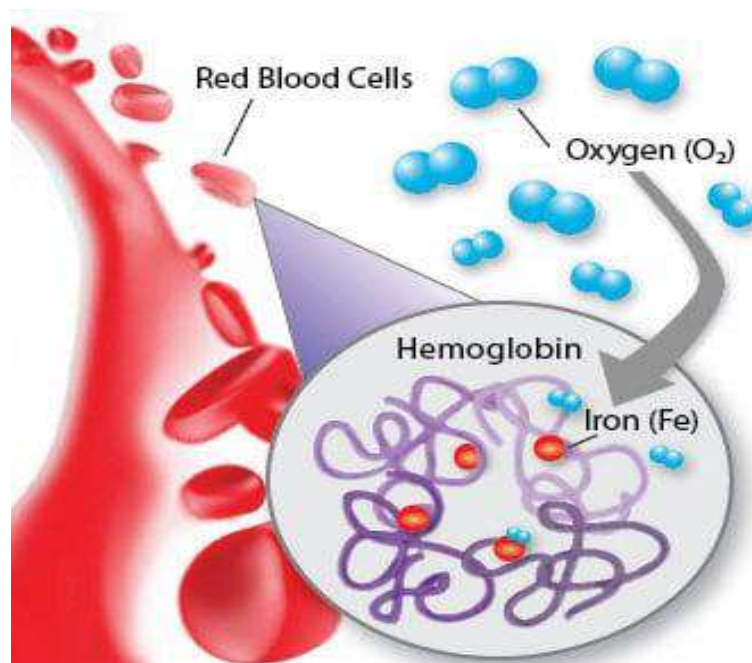


Figure (5): show hemoglobin.

As then-infant grows, the gamma chains are gradually replaced by beta chains, forming the adult hemoglobin structure.

Each globulin chain contains an important iron-containing porphyrin compound termed heme. Embedded within the heme compound is an iron atom that is vital in transporting oxygen and carbon dioxide in our blood.

The iron contained in hemoglobin is also responsible for the red color of blood.

Hemoglobin also plays an important role in maintaining the shape of the red blood cells.(Charles Patrick Davis,MD,PhD,2006)

1.7 What are normal hemoglobin values?

The hemoglobin level is expressed as the amount of hemoglobin in grams (gm) per deciliter (dL) of whole blood, a deciliter being 100 milliliters.

The normal ranges for hemoglobin depend on the age and, beginning in adolescence, the gender of the person. The normal ranges are:

1-Newborns: 17 to 22 gm/dL

2-One (1) week of age: 15 to 20 gm/dL

3-One (1) month of age: 11 to 15 gm/dL

4-Children: 11 to 13 gm/dL

5-Adult males: 14 to 18 gm/dL

6-Adult women: 12 to 16 gm/dL

7-Men after middle age: 12.4 to 14.9 gm/dL

8-Women after middle age: 11.7 to 13.8 gm/dL

(Goldman L, Schafer A 2016)

1.8 The role of Iron in health and disease

Iron is a mineral that combines with protein to form hemoglobin, the red substance in blood that carries oxygen to the body's cells. Iron helps prevent nutritional anemia and increase resistance to infection. Hemochromatosis is a disease in which too much iron builds up in the body, causing iron overload. iron overload (hemochromatosis) is almost always the result of an inherited abnormality of the regulation of iron transport that affects hepcidin or its receptor ferroportin. (Brissot, P 2008). If your body doesn't have enough hemoglobin, your tissues and muscles won't get enough oxygen to be able to work effectively. This leads to a condition called anemia (Gavin V. De Walle and Mary J. Brown, updated 2022).

1.9 Iron Requirement Per Day

Age	Male	Female	Pregnancy	Lactation
Birth to 6 months	0.27 mg*	0.27 mg*		
7–12 months	11 mg	11 mg		
1–3 years	7 mg	7 mg		
4–8 years	10 mg	10 mg		
9–13 years	8 mg	8 mg		
14–18 years	11 mg	15 mg	27 mg	10 mg
19–50 years	8 mg	18 mg	27 mg	9 mg
50+	8 mg	8 mg		

Table (6): Shows iron ranges per day.

1.10 Sources of Iron

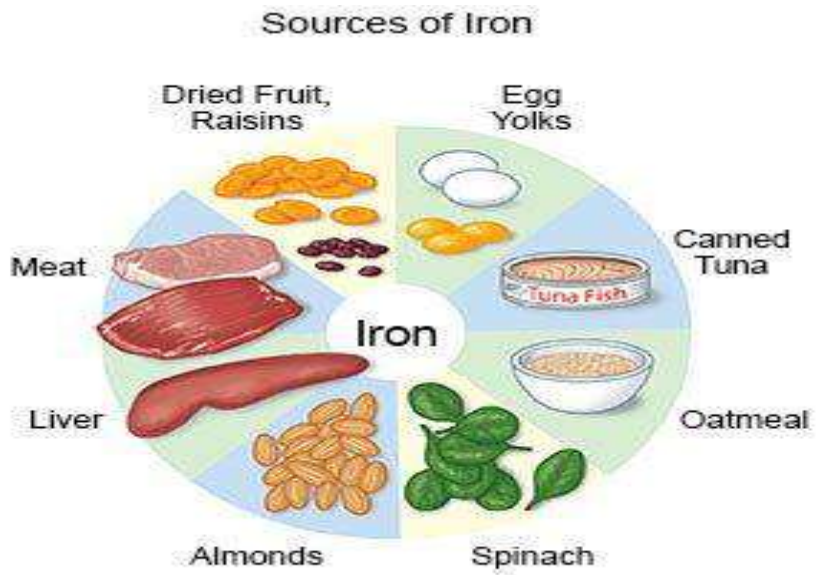


Figure (7): Shows sources of iron.

Iron-deficiency anemia

Iron deficiency is defined as a decrease in the total amount of iron in the body. Iron deficiency anemia occurs when iron deficiency is severe enough to interfere with the erythropoiesis process resulting in anemia symptoms to arise. This happens when the balance between iron intake, iron storage, and iron loss from the body cannot meet erythrocyte production. (Sudoyo AW, Setiyohadi B 2017)

Anemia is the most common medical problem encountered in daily practice. Anemia by functional defined as a decrease in the number of erythrocyte masses so thus it cannot fulfil its function to carry an adequate amount of oxygen to peripheral tissue.¹ Anemia is not a disease, but a part of the symptoms of an underlying illness, whether it was acquired or inherited.² Anemia is a significant health problem especially in developing countries that have a high prevalence. (Alli N, Vaughan2017). There are three leading causes of anemia including iron deficiency anemia, hemoglobinopathy, and malaria. (Naghavi M, et al.2010)

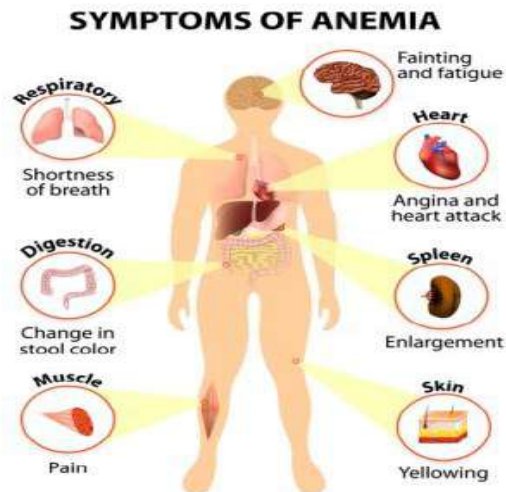


Figure (8): shows Symptoms of anemia.

1.12 Different types of anemia have different causes. They include:

- **Iron deficiency anemia.** This most common type of anemia is caused by a shortage of iron in your body. Your bone marrow needs iron to make hemoglobin. Without adequate iron, your body can't produce enough hemoglobin for red blood cells.
- **Vitamin deficiency anemia.** Besides iron, your body needs folate and vitamin B-12 to produce enough healthy red blood cells. A diet lacking in these and other key nutrients can cause decreased red blood cell production.
- **Anemia of inflammation.** Certain diseases — such as cancer, HIV/AIDS, rheumatoid arthritis, kidney disease, Crohn's disease and other acute or chronic inflammatory diseases — can interfere with the production of red blood cells.
- **Aplastic anemia.** This rare, life-threatening anemia occurs when your body doesn't produce enough red blood cells. Causes of aplastic anemia include infections, certain medicines, autoimmune diseases and exposure to toxic chemicals.
- **Anemia associated with bone marrow disease.** A variety of diseases, such as leukemia and myelofibrosis, can cause anemia by affecting blood production in your bone marrow. The effects of these types of cancer and cancer-like disorders vary from mild to life-threatening.
- **Hemolytic anemia.** This group of anemia develops when red blood cells are destroyed faster than bone marrow can replace them. Certain blood diseases increase red blood cell destruction. You can inherit a hemolytic anemia, or you can develop it later in life.
- **Sickle cell anemia.** This inherited and sometimes serious condition is a hemolytic anemia. It's caused by a defective form of hemoglobin that forces red blood cells to assume an abnormal crescent (sickle) shape. These irregular blood cells die prematurely, resulting in a chronic shortage of red blood cells. (Elghetany MT, Schexneider KI, 2017)

1.13 Factors of anemia

- A diet lacking in certain vitamins and minerals
- Menstruation
- Pregnancy
- Chronic conditions
- Family history.
- Other factors.
- Age

(Elghetany MT, Schexneider KI, 2017)

1.14 Treatment of iron deficiency anemia

- **Iron supplements**, also called iron pills or oral iron, help increase the iron in your body. This is the most common treatment for iron-deficiency anemia.
- **Intravenous or IV iron** is sometimes used to put iron into your body through one of your veins. This helps increase iron levels in your blood.
- **Medicines** help your bone marrow make more red blood cells.
- **Blood transfusions** quickly increase the amount of red blood cells and iron in your blood. They may be used to treat serious iron-deficiency anemia.
- **Surgery** may be needed to stop internal bleeding.

(Chiang, W.C etal..2002)

2. Estimation of iron

A simple, rapid, and sensitive method for the determination of iron in serum or plasma is described. The procedure is carried out at room temperature with 2 ml. of serum or plasma, or with 1 ml. if high values are expected; it can be applied to turbid or jaundiced samples, whether previously frozen or not. An ethanolic solution of 4: 7-diphenyl-1: 10-phenanthroline is used to produce a colored iron complex, the optical density of which can be measured in any suitable photometer, using either 10- or 20-mm. fused glass cuvettes or matched tubes of 1.1 cm. internal diameter. (Niedzielski, M. Zielinska-Dawidziak, 2014)

THE ESTIMATION OF IRON BY STANDARD POTASSIUM BICHROMATE SOLUTION (Penny's Method).

Apparatus, Reagents. —For the preparation of the standard solution pure $K_2Cr_2O_7$ is required, also the iron wire as before, E. $SnCl_2$, 2/5 E. HCl and E. $K_6Fe_2C_{12}N_{12}$, freshly prepared and containing no ferrocyanide. A white porcelain plate is used for the indicator tests. For analysis the student may take another sample of the ferric sulphate previously used.

Method, Reactions. —If a solution of $K_2Cr_2O_7$ be added to a solution of a ferrous salt in presence of a strong free acid, oxidation takes place according to the equation,



The end point of this reaction is determined by an “external” indicator. A drop of the solution is brought in contact on a white porcelain plate with a drop of $K_6Fe_2C_{12}N_{12}$ freshly prepared and containing no $K_4FeC_6N_6$. A rich blue results if the oxidation has not proceeded far. On adding

to the solution more $K_2Cr_2O_7$ and testing a drop after each addition, the blue changes to a turbid greenish blue, to a grey, and finally to a brown. When the greenish blue has just disappeared, the reaction is complete.

Preparation of the Standard Solution N/10 $K_2Cr_2O_7$. As this salt yields 3 atoms of oxygen, which are equivalent to 6 atoms of hydrogen, the normal solution will contain $78 + 104.8 + 112/6 = 49.13$ gms. per litre, therefore a solution will contain 4.913 gms. per litre. Weigh out this quantity of the salt; dissolve it in distilled water, and make up to 1 litre at $16^\circ C$. One c.c. of this solution should be equal to .0056 gm. of iron.

3. CONCLUSION:

IDA remains the most commonly encountered health problems in developing countries. This disorder can interfere with cognitive functions and the ability to carry out daily activities. Complete history taking, physical examination, and laboratory test need to be conducted thoroughly to be able to establish the diagnosis of IDA as well as to determine its cause. Male and female patients who have anemia should be given diagnostic evaluation in the form of endoscopy to find out the reason according to gastrointestinal tract disorders. The management of IDA is focused on to increase the hemoglobin levels, red blood cells levels, iron storage, and comorbid conditions. Currently used therapies include oral iron therapy which is the first-line therapy, intravenous iron therapy, and blood transfusion.

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