Water and minerals metabolism

304 Biochem

By

Prof. ENTSAR ALI SAAD

2020

Iron (Fe)

- **Absorption, transport and storage:**
- **Absorption:**
  - Ordinary diet contain 10-20 mg of iron, < 10% of them is absorbed into mucosal cells of the doudenum&proximal jejnum
  - **Heme iron** is absorbed as it is then broken down and iron released within the intestinal mucosal cells
  - **Nonheme iron** is absorbed in ferrous state
  - **Ferric ion** is reduced to ferrous ion before absorption
  - **Reducing substances** in the diet like vitamin C **enhance** iron absorption
  - While **substances that decrease the solubility of iron** in the intestine (such as phosphate, oxalate, phytic acid, fatty acids) and **increased pH inhibit** iron absorption

Iron (Fe)

- **Absorption, transport and storage:**
- **Transport and storage:**
  - Inside the intestinal mucosal cells there is a **specific protein carrier** combines with **Fe2+**and transfer it across the cell membrane
  - The **Fe2+**is oxidized to **Fe3+**
  - Inside the cell the intracellular carrier protein molecule delivers ferric to mitochondria and then ferric is distributed to **apoferritin** (apoferritin acts as a ferroxidase and oxidizes **Fe2+** to **Fe3+**) to form **ferritin** or to **apotransferrin** to form **transferrin** depending on the state of iron metabolism in the body
  - **Ceruloplasmin** (copper-binding plasma protein) acts as a ferroxidase that convert **Fe2+**to **Fe3+** and helps incorporation of iron into transferrin
  - **Ferritin** is the chief storage form of iron . It is present in intestinal mucosal cells, liver, kidney, spleen, and bone marrow. In order to release iron from ferritin iron must be reduced from **Fe3+** to **Fe2+**
  - **Transferrin** is a plasma protein for the transport of iron to bone marrow
  - Under normal conditions, all iron bound to transferrin is taken up rapidly by the bone marrow for hemoglobin synthesis.

Iron (Fe)

- **Iron deficiency**
- **<u>Iron deficiency anemias</u>**

Causes:

1. Inadequate intake
2. Inadequate absorption e.g. gastrointestinal disturbances such as diarrhea, steatorrhea (fatty feces) , after gastrictomy or intestinal resection
3. Excessive blood loss

Symptoms:

Anemia: weakness, fatigue, headaches; impaired mental and physical work performance; impaired immunity; pale skin, nailbeds, and mucous membranes; concave nails; chills; pica

In iron deficiency anemias:

1. Plasma protein bound iron is low
2. TIBC tends to rise
3. UIBC is higher than normal

Chills is قشعريرة برد

Pica is a tendency or craving to eat substances other than normal food (such as clay, plaster, or ashes), occurring during childhood or pregnancy, or as a symptom of disease.

Iron (Fe)

- **Hemochromatosis (Hemosiderosis)**

- **Hemosiderin** is formed in states of iron overload when the synthesis of apoferritin and its uptake of iron is maximal.

- **Hemosiderin** contains a larger content of iron than ferritin and exists as microscopically visible iron staining particles.

- **Hemosiderosis** is accumulation of Hemosiderin in tissues.

Iron (Fe)

- **Hemochromatosis (Hemosiderosis)**

Causes of Hemosiderosis:

1. Excessive administration of injectable iron
2. Repeated blood transfusion over a period of years e.g. hemolytic anemias patients
3. Increased absorption of iron from intestine e.g. inherited anomaly of iron absorption (about 20-45% of dietary iron absorbed), acquired siderosis of dietary origin (combination of low phosphate diet and high intake of iron this enhances absorption of iron)

Symptoms:

– Deposition of iron **in liver** causing liver cirrhosis, **in pancreas** causing bronzed diabetes, and **in skin** causing bronzed pigmentation (hemochromatosis)

Copper (Cu)

- Adult male body weighing 60-70 kg contain about **0.09-0.15** g of copper

- **Distribution of copper in the body:**
  - **About 64 mg** is present in the muscles
  - **About 23 mg** is present in the bones
  - **About 18 mg** is present in the liver (**hepatocuprein**)

- **Copper in blood:**
  - **In RBCs:** 80% of RBCs copper is present as superoxide dismutase [(SOD (erthrocuprein)]
  - **In serum:** about 90 microgram/dl present in two forms:
  - **80-95%** is firmly bound, consists of **ceruloplasmin**.
  - **5-20%** is loosely bound to plasma albumin and it is known as **direct reacting copper**

Copper (Cu)

- **Food sources:** dried legumes, fish, kidney, liver, and nuts

- **RDA**
  - adults: 2.5 mg/day

- **Absorption:**
- Mainly in the upper intestinal tract where acidity facilitate its availability as soluble element

- **Excretion:**
- Excess copper is excreted mainly via bile and to small extent in feces.

Copper (Cu)

- **Functions:**
  - Essential for: hemoglobin synthesis, normal bone formation, and maintenance of myelin within the nervous system
  - It is a constituent of **hemocyanin** present in the blood of crtain invertebrates and act like hemoglobin as an oxygen carrier
  - Component of certain enzymes:
    - Cytochrome oxidase
    - SOD
    - L-ascorbic acid oxidase
    - Tyrosinase
    - Prolyl and lysyl hydroxylases
    - Dopamine hydroxylase
    - copper/zinc superoxide dismutase

Copper (Cu)

- **Deficiency:**
  - A variety of human copper deficiency conditions are recognized. It is observed in:
    - Premature newborne infants
    - Malnutrition, malabsorption syndrome and chronic diarrhea
    - Prolonged intravenous fluid therapy

- **Symptoms:**
  - Hypochromic anemia and neutropenia (decrease in neutrophiles number)
  - Osteoporosis and bone and joint abnormalities
  - Abnormal hair and decreased pigmentation of the skin due to impaired tyrosine metabolism and melanin synthesis
  - Vascular abnormalities
  - Neurologic disorders in advanced deficiency