IRON DEFICIENCY ANEMIA

IDA

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OBJECTIVES

- Prevalance of IDA
- Metabolism & absorption of Iron
- Iron stores
- Pathogenesis of IDA
- Causes of IDA
- Features of IDA
- Investigations of IDA
- Treatments of IDA

Around 30% of the total world population is anaemic and half of these, some 600 million people, have iron deficiency. The classification of anaemia by the size of the red cells (MCV) indicates the likely cause.

Red cells in the bone marrow must acquire a minimum level of haemoglobin before being released into the blood stream. While in the marrow compartment, red cell precursors undergo cell division, driven by erythropoietin. If red cells cannot acquire haemoglobin at a normal rate, they will undergo more divisions than normal and will have a low MCV when finally released into the blood.

- The MCV is low because component parts of the haemoglobin molecule are not fully available: that is, iron in iron deficiency, globin chains in thalassaemia, haem ring in congenital sideroblastic anaemia and, occasionally, poor iron utilisation in the anaemia of chronic disease/anaemia of inflammation.

Iron deficiency is the leading cause of anemia worldwide. Although the presentation of classic iron deficiency anemia is linked with a microcytic anemia, early iron deficiency is associated with a normocytic anemia. Consequently, iron deficiency should be considered in all patients with anemia, and iron indices should be a part of the evaluation of any patient with hypopro-ductive anemia, regardless of the MCV.

Iron is acquired in the diet from heme sources (i.e., meat) and from nonheme sources (e.g., vegetables such as spinach).

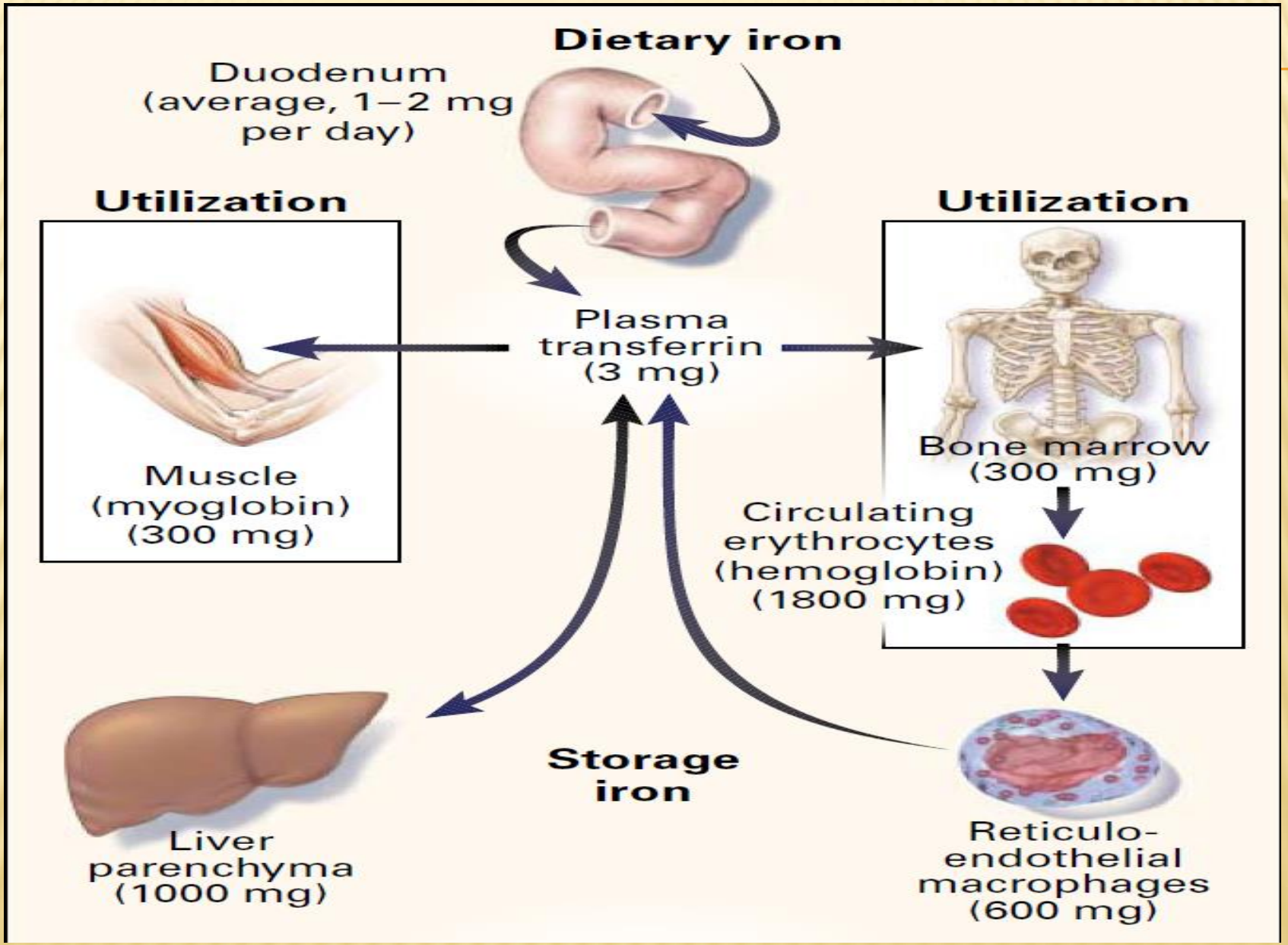
Iron from heme is better absorbed than nonheme iron.

Iron absorption is increased in iron deficiency, hypoxia, ineffective erythro-poiesis, and hereditary hemochromatosis.

Iron is absorbed from the proximal small intestine; it is transported in the cell bound to ferroportin and through the plasma bound to transferrin.

Its uptake into the RBC precursors is mediated through the transferrin receptor. Iron absorption from the intestine is further regulated by hepcidin. Iron outside hemoglobin-producing cells is stored in ferritin. Men and women have total-body iron concentrations of 50 mg/kg and 40 mg/kg, respectively. Between 60% and 75% of the iron is found in hemoglobin. A small amount (2 mg/kg) is found in heme and nonheme enzymes, and 5 mg/kg is found in myoglo-bin. The remainder is stored in ferritin, which resides primarily in liver, bone marrow, spleen, and muscle.

- The capacity for excreting iron is limited, and iron overload occurs in patients with excessive absorption from the gastrointestinal tract (as a result of ineffective erythropoiesis or congenital hemochromatosis) and in those with chronic transfusions. Iron overload leads to increased iron deposition in these tissues and secondary deposition in endocrine organs, resulting in liver dysfunction, diabetes, and other endocrine abnormalities.


Dietary iron
Duodenum
(average, 1–2 mg
per day)
Utilization
Utilization
Plasma
transferrin
(3 mg)
Muscle
(myoglobin)
(300 mg)
Bone marrow
(300 mg)
Circulating
erythrocytes
(hemoglobin)
(1800 mg)
Storage
iron
Liver
parenchyma
(1000 mg)
Reticulo-
endothelial
macrophages
(600 mg)

PATHOGENESIS OF IRON DEFICIENCY

1- Blood loss

Occult or overt GI losses, traumatic or surgical losses

2- Failure to meet increased requirements

Rapid growth in infancy and adolescence

Menstruation, pregnancy

3- Inadequate iron absorption

Diet low in heme iron

Gastrointestinal disease or surgery

Excessive cow’s milk intake in infants

TABLE 1. CAUSES OF IRON DEFICIENCY.

Inadequate absorption

Poor bioavailability
Antacid therapy or high gastric pH
Excess dietary bran, tannin, phytates, or starch
Competition from other metals (e.g., copper or lead)
Loss or dysfunction of absorptive enterocytes
Bowel resection
Celiac disease
Inflammatory bowel disease
Intrinsic enterocyte defects

Increased loss

Gastrointestinal blood loss
- Epistaxis
- Varices
- Gastritis
- Ulcer
- Tumor
- Meckel's diverticulum
- Parasitosis
- Milk-induced enteropathy of early childhood
- Vascular malformations
- Inflammatory bowel disease
- Diverticulosis
- Hemorrhoids

Genitourinary blood loss
- Menorrhagia
- Cancer
- Chronic infection

Pulmonary blood loss
- Pulmonary hemosiderosis
- Infection

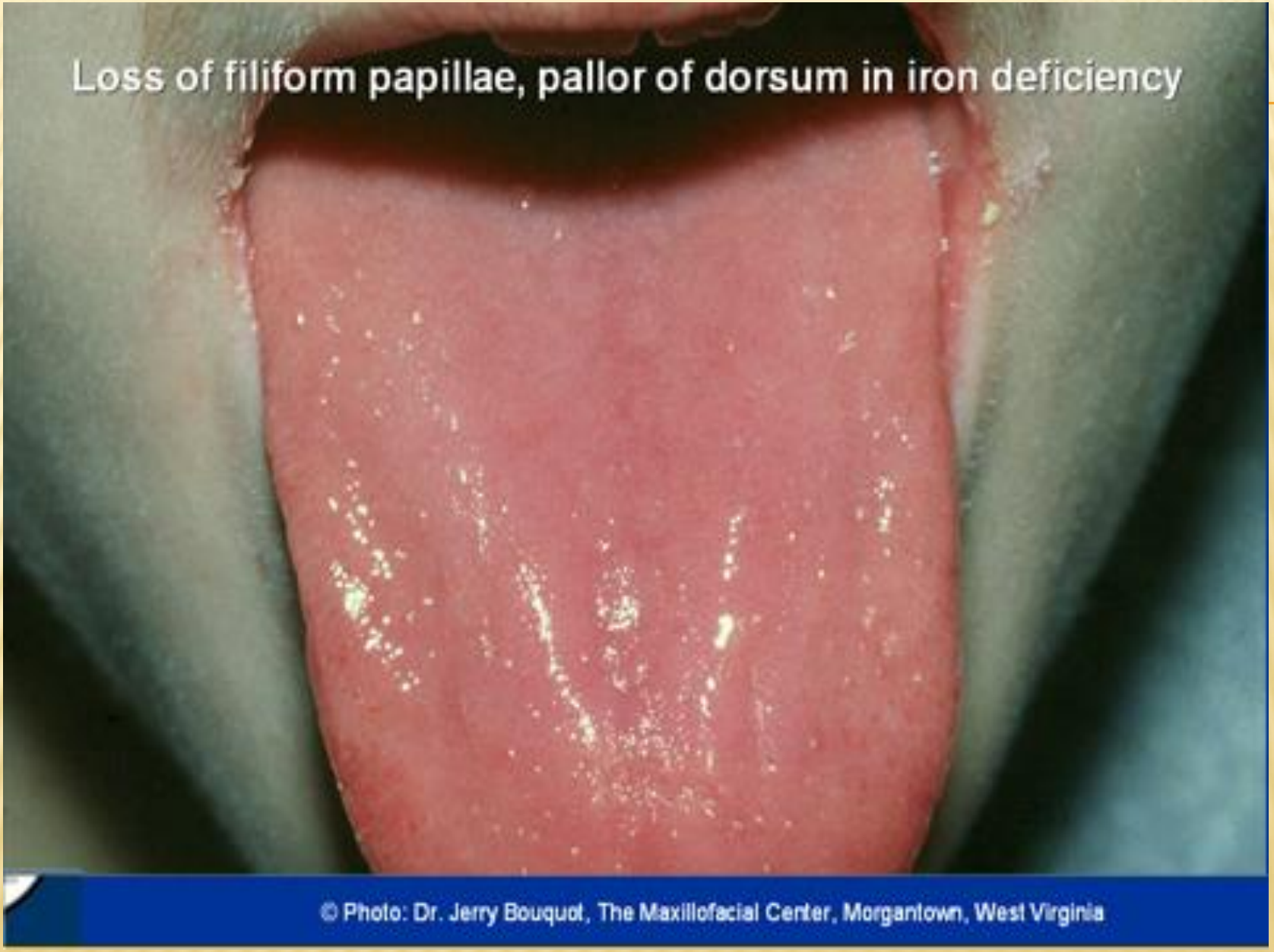
Other blood loss
- Trauma
- Excessive phlebotomy
- Large vascular malformations

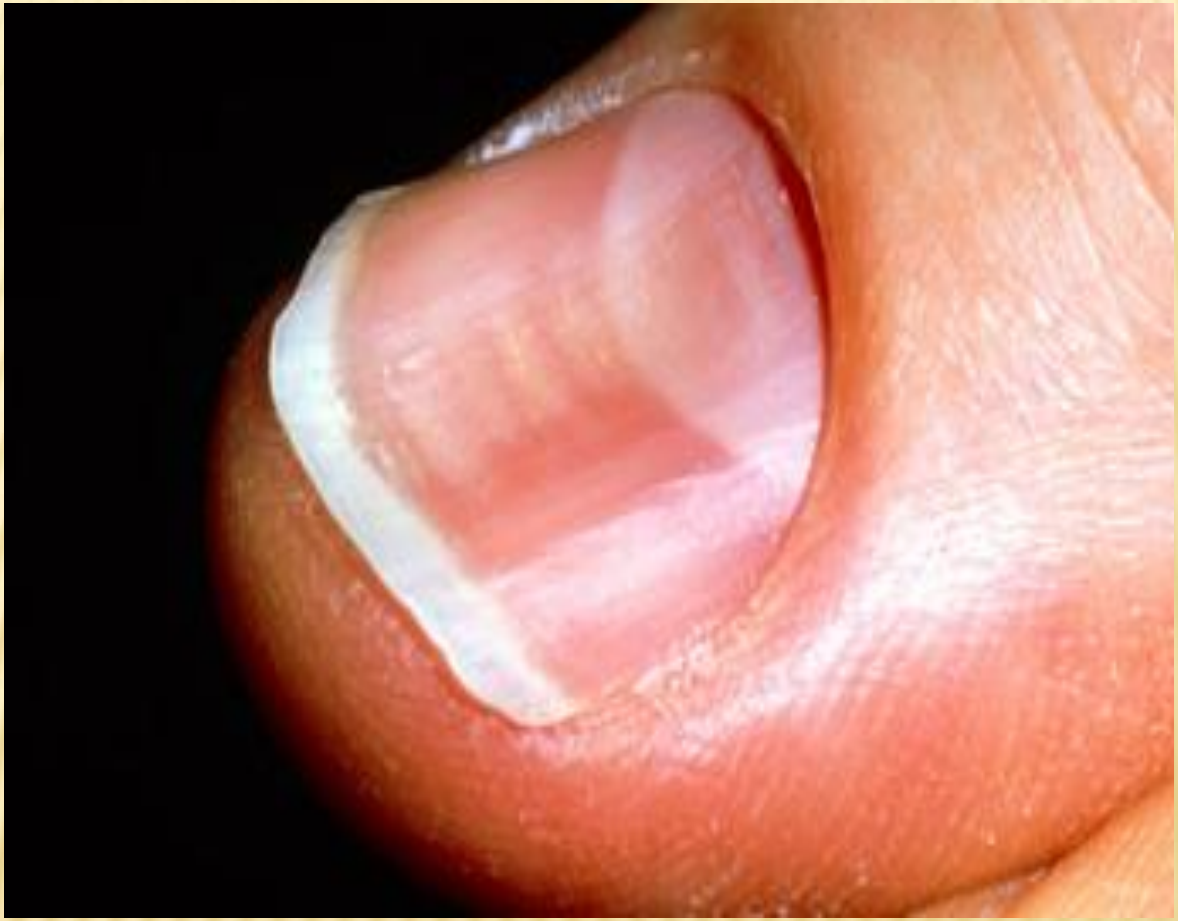
FEATURES OF IRON DEFICIENCY ANEMIA

- Depends on the degree and the rate of development of anemia
- Symptoms common to all anemias:
  - pallor, fatigability, weakness, dizziness, irritability

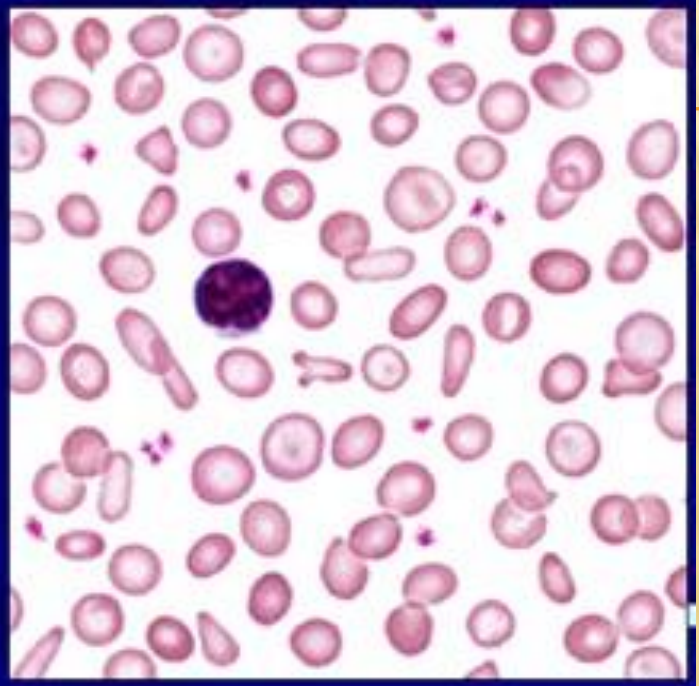
OTHER FEATURES OF IDA

- Pagophagia - craving ice
- Pica - craving of nonfood substances
  - e.g., dirt, clay, laundry starch
- Glossitis - smooth tongue
- Restless Legs
- Angular stomatitis - cracking of corners of mouth
- Koilonychia - thin, brittle, spoon-shaped fingernails


Loss of filiform papillae, pallor of dorsum in iron deficiency
© Photo: Dr. Jerry Bouquot, The Maxillofacial Center, Morgantown, West Virginia

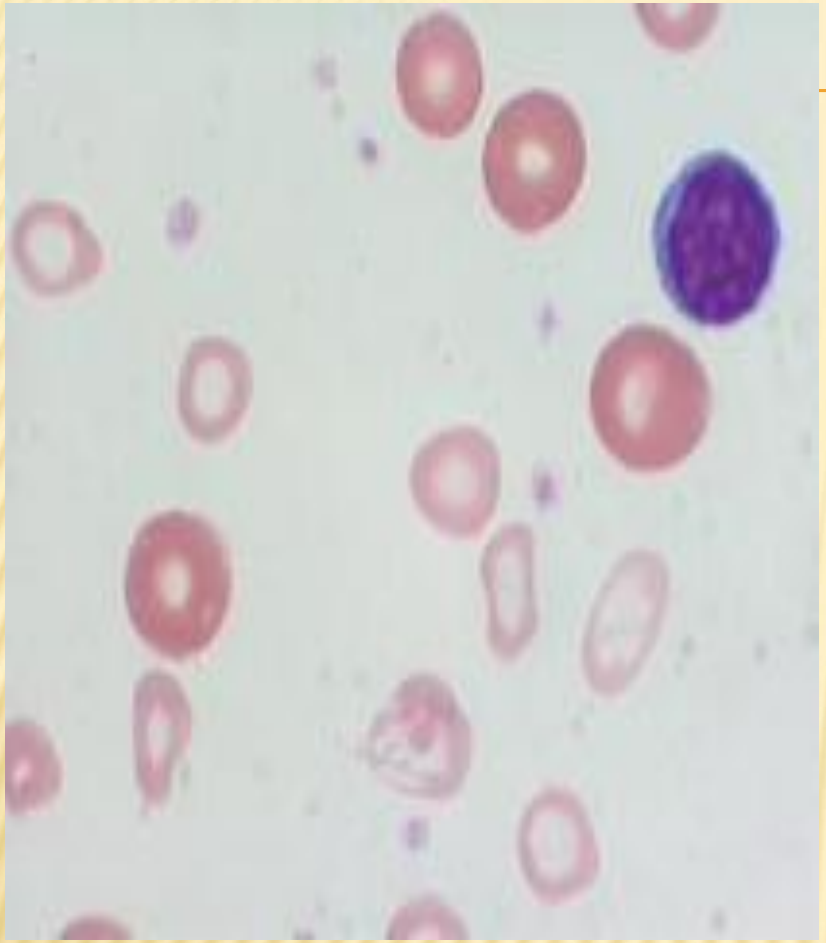


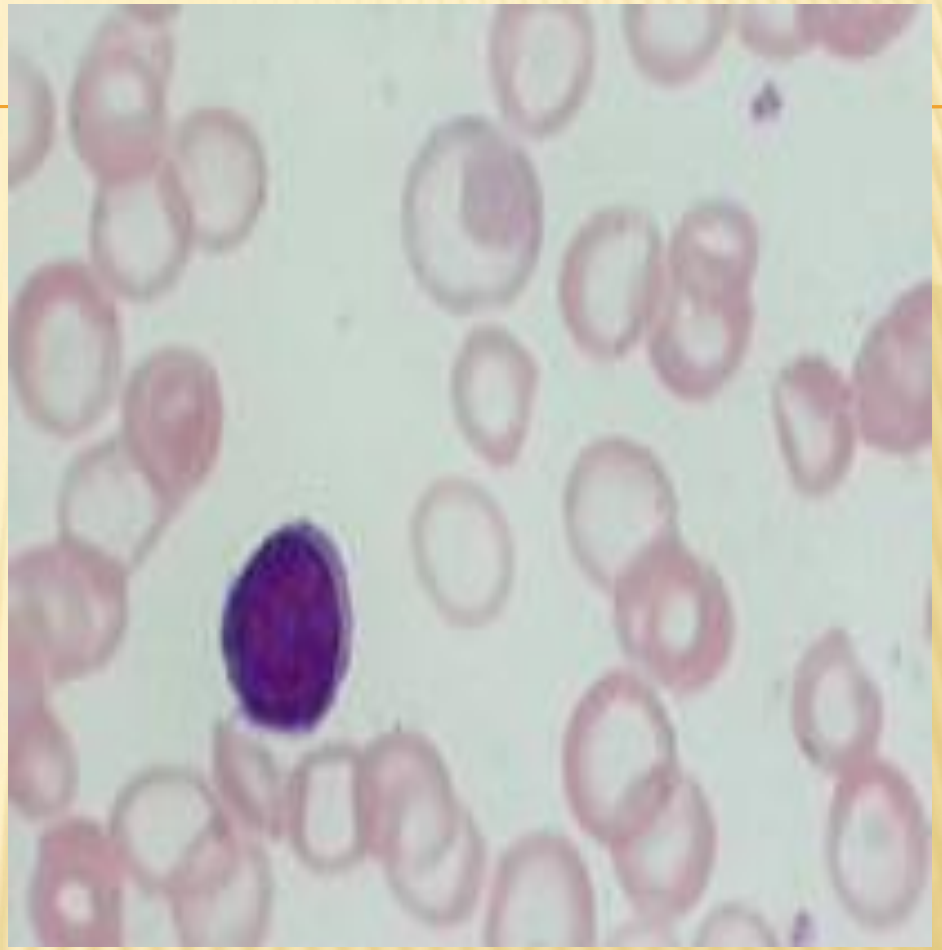
Laboratory findings:



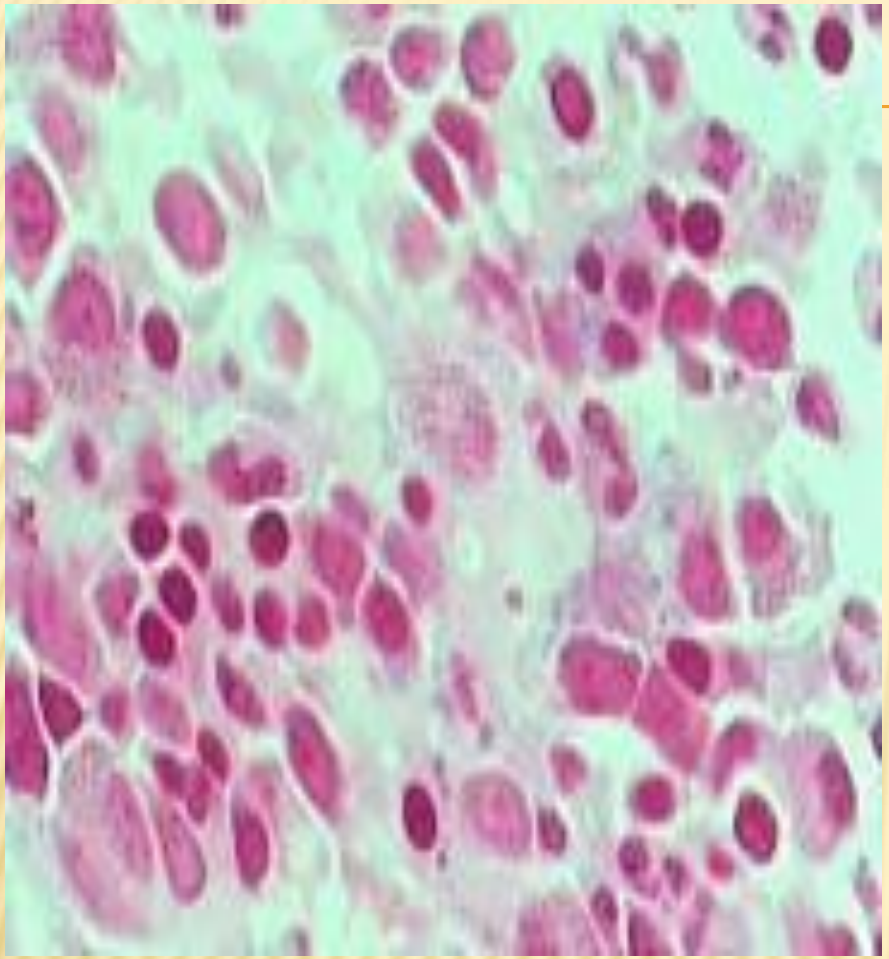
- Red cell indices:
  - Low Hb conc.
  - MCV, MCH, MCHC* ↓
- Blood film:
  - Hypochromic microcytic Picture.
  - Occasional Target cells.
  - Pencil shaped poikilocytes.
  - Normal reticulocyte count.
- Bone marrow iron:
  - Normal to hypercellular.
  - RBC precursors are increased in number.
  - Iron stain negative.
- Chemical testing on serum:

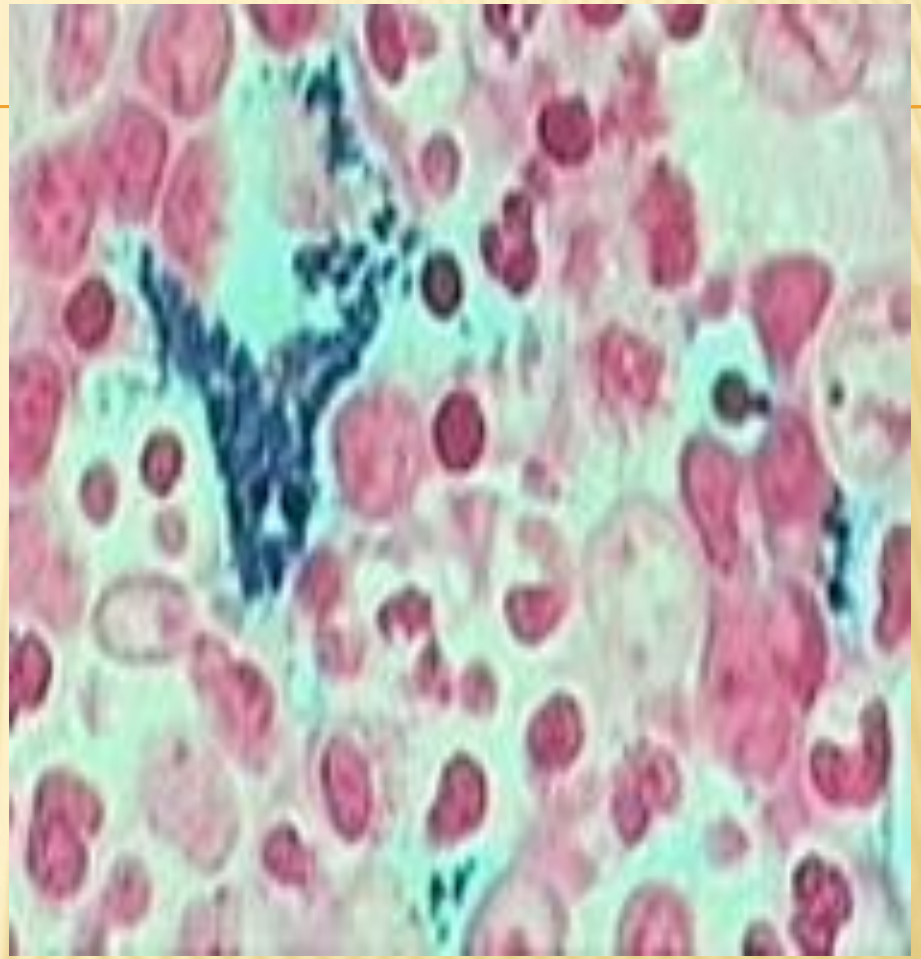
Serum iron	Decreased
Transferrin/TIBC	Normal to High
Serum ferritin	Decreased (Very low)





HYPOCHROMIC MICROCYTIC PICTURE (IDA)





-VE BM IRON STAIN +VE

TREATMENT

Oral iron supplementation, with administration of ferrous sulfate or ferrous gluconate two or three times daily, is the treatment for iron deficiency. Patients should be educated about the potential gastrointestinal side effects, including diarrhea or constipation, and some may benefit from a gradual increase in the dose based on tolerance. Iron should be administered for several months after resolution of anemia to allow for the reconstitution of iron stores.

In patients with malabsorption, a complete inability to tolerate oral iron, or iron demands that outstrip replacement with oral supplements, parenteral iron may be administered. The paren-teral administration of iron, especially iron dextran, has been associated with anaphylaxis. However, newer preparations such as sodium ferric gluconate, iron sucrose, ferumoxytol, and ferric carboxymaltose are significantly safer.

All male patients and postmenopausal women with iron deficiency require evaluation for a source of gastrointestinal bleeding.

The haemoglobin should rise by around 10 g/L every 7–10 days and a reticulocyte response will be evident within a week. A failure to respond adequately may be due to non-adherence, continued blood loss, malabsorption or an incorrect diagnosis. Patients with malabsorption, chronic gut disease or inability to tolerate any oral preparation may need parenteral iron therapy. Previously, iron dextran or iron sucrose was used, but new preparations of iron isomaltose and iron carboxymaltose have fewer allergic effects and are preferred. Doses required can be calculated based on the patient's starting haemoglobin and body weight. Observation for anaphylaxis following an initial test dose is recommended.

THANKS