

D.SAHAR, <u>LECTURE 2</u>, ANEMIAS 2019, physiology, medical collage

Summary – iron absorption and transport





Molecules involved in iron metabolism

Iron transport in circulation and storage Transferrin Ferritin Hemosiderin

Protein controls iron homeostasis by regulating all above proteins

Hepcidin

Functional compound Hemoglobin, Myoglobin Cytochrome Catalase, Peroxidase.



*Normal blood film



Condition occur due to lack enough healthy Red <u>blood cells</u> or hemoglobin

1- loss of large volume of blood

usually when the blood loss occur like hemorrhoid, gastritis. here is blood loss, plasma replaced quickly while RBC takes few weeks to replaced .this caused by some chronic condition.



2-Decrease production of Red blood cell

A-Iron deficiency anemia

Lack of iron cause decrease Hb production

B-Bone marrow aplasia

-Loss of function of bone marrow due to

- -Drug poisoning
- -Gamma ray irradiation
- -Viral infection.

3- failure of maturation of RBC

-B12 and folic acid

-B12 important for synthesis of DNA ,and caused failure of nuclear maturation and division so inhibit RBC production. that cause (megaloblastic anemia),pernicious anemia



Megaloblastic Anemia



<u>3-Hemolysis of RBCS</u>

- a. Drug poisoning
- b. Hemolytic disease (sickle cell anemia, spherocytosis)
- C. Erythroblastosis fetalis , disease of newborn antibodies from mother during pregnancy destroy fetal RBC
- d. Thalassemia(cooly's anemia),also called Mediterranean anemia ,due to deficiency of globulin caused low concentration of Hb

EFFECT OF ANEMIA ON CIRCULATING SYSTEM

Viscosity of blood decreased.

Decreased resistance of blood flow in peripheral blood vessels.

Cardiac out put increased 2 time, Hypoxia caused increased return of blood to the heart and increased cardiac output more.

symptoms of anemia

fatigue.

weakness.

pale skin.

Shortness of breath.

Dizziness

Soreness of the mouth with cracks at the corners

strange cravings to eat items that aren't food, such as dirt, ice, or clay. (pica) (IRON DEFICIENCY)

A tingling, "pins and needles" sensation in the hands or feet ,Lost sense of touch ,tongue swelling (B12 DEFICIENCY)

mean cell volume(mcv)

□ is the average volume of individual RBCS.

□ MCV=PCV/RBC×10

□ the normal value 80–100 fl. femtoliters (fL, or 10⁻¹⁵L)

RBC is the quantity expressed in millions per microliter (10⁶ / μl).

MCV decreased in (e.g., microcytic anemia).<80fL

- **iron deficiency anemia**
- thalassemia
- and other chronic diseases

MCV increased in (e.g., macrocytic anemia).>100fL

- in folic acid deficiency
- **vitamin** B₁₂deficiency

mean corpuscular hemoglobin (MCH)

- or "mean cell hemoglobin" (MCH) :
- is the average mass of hemoglobin per red blood cell in a sample of blood.

It is calculated by dividing the total mass of hemoglobin by the number of red blood cells in a volume of blood.

- * MCH=(Hgb)/RBC ×10
- * PICOgram=10⁻¹²
- A normal value in 27 to 31 picograms/cell.
 It depends on hemoglobin synthesis.
 It decreases when Hb synthesis reduces

Mean corpuscular hemoglobin concentration:(MCHC)

a measure of the concentration of hemoglobin in a given volume of packed red blood cells.

- It is calculated by dividing the hemoglobin by the hematocrit.
- HB/PCV×100(gm/dl) *dl=10⁻¹
- normal ranges <u>32 to 36 g/dL</u>



Clinical sign of anaemia



PALLOR



KOilonychia



ANGULAR STOMATITIS

ATROPHIC GLOSSITIS



CHEALITIS

*Iron deficiency anemia

*Diagnosis of iron deficiency anemia

- *Low Hb
- *Low pcv
- *Low MCV
- *Low MCH, MCHC, LOW IRON, LOW S.FERRITIN
- *Blood film ,hypochromic ,microcytic ,pencil cell ,normal wbc, normal platelets



Without a mutation enough Hemoglobin



No thalassemia carrier

With one mutation less Hemoglobin



β-thalassemia carrier without illness, but less hemoglobin (slight aneamia) With two mutations no β-globin



β-thalassemia major patient with severe aneamia

thalassemia

CLINICAL PRESENTATION

THALASSEMIA MAJOR

GENERAL FEATURES

- Pallor
- Fatigue
- Dyspnea on exertion
- Poor apetite
- Palpitations
- Poor growth

FEATURES OF HEMOLYSIS

- Jaundice
- Hyperuricaemia (Gout)
- Gallstones

EXCESSIVE ERYTHROPOEISIS

- Maxillary overgrowth (chipmunk)
- Increased spaces, overbite and malocclusion of teeth
- Frontal bossing
- Chronic sinusitis
- Impaired hearing



Lab. Investigations of Thalassemia:

- Laboratory findings:
- ightarrow Hb \downarrow , MCV \downarrow , MCH \downarrow , MCHC \downarrow
- ≻RBCs :
 - Hypochromic, Microcytic RBCs. Target cells, nucleated red cells
 - Anisocytosis
 - Poikilocytosis
 Basophilic stippling
 - RBC inclusions
- Platelets: Normal
 WBCs: Normal
 Plasma: ↑ iron
 - Normal or \uparrow ferritin.



RH INCOMPATIBILITY

MOTHER RH negative ,FETUS RH positive

- RBCs from the fetus can go into the mother's bloodstream through the placenta.
- Rh-Negative mother's immune system treats the Rh-Positive fetal cells as a foreign substance and makes *antibodies* against them.
- These anti-Rh antibodies may cross the placenta into the fetus, where they destroy the fetus's circulation red blood cells.

Signs & Symptoms of Erythroblastosis Fetalis



Kernicterus www.medindia.net



Hemolytic Anemia



Jaundice





Symptoms

Hemolytic Anemia

- Jaundice due to hyperbilirubinemia
- Kernicterus
- Treatment phototherapy
- **Exchange transfusion**

- Anemia
- Hyperbilirubinemia
- Reticulocytosis (6 to 40%)
- ↑ nucleated RBC count (>10/100 WBCs)
- Thrombocytopenia
 - Leukopenia
 - **Positive Direct Antiglobulin Test**
- Hypoalbuminemia

DIRECT COOMB'S TEST



Patient Sample A anti- Hu IgG Coomb's Reagent



Example: The baby's sample is positive for the presence of the mother's Ab on the surface of RBCs in erythroblastosis fetalis

Agglutination





Megaloblastic Anemia ↓ RBCs (**↓** Hematocrit) 个MCV (size RBCs) **Hypersegmented Neutrophils ↑** Homocysteine

