

PATHOLOGY OF BLOOD AND LYMPHATIC SYSTEM

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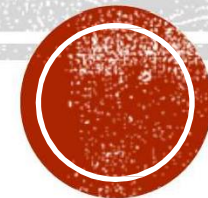
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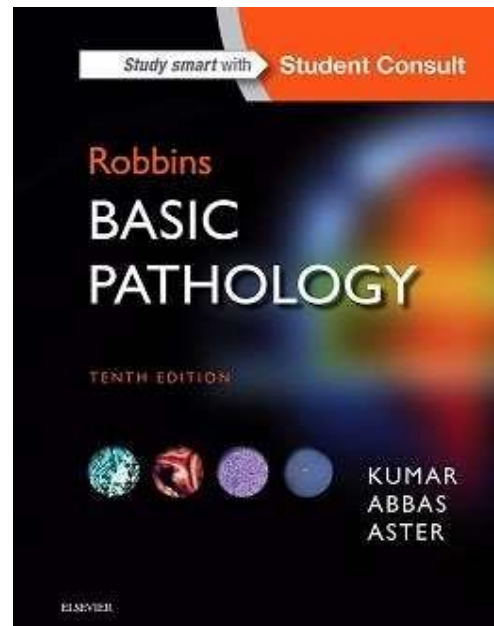
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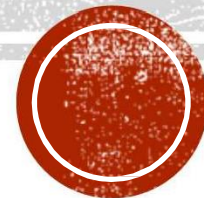


- First Semester 2021 / 2022
- Reference: Robbins Basic Pathology 10th ed



ANEMIA

By Lujain Ahmad



DEFINITION

- ❑ Reduction of oxygen carrying capacity of blood secondary to decrease in red cell mass
- ❑ Leads to tissue hypoxia triggers production of erythropoietin
- ❑ Practically, measure by Hemoglobin concentration, and Hematocrit

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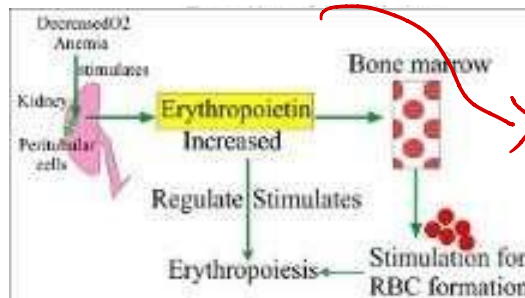


ANEMIA AND ERYTHROPOIETIN

- Anemia triggers production of erythropoietin
- Causes compensatory erythroid hyperplasia in bone marrow (BM)
- In acute anemia, production can increase by 5x or more in healthy people. If the anemia persisting for a long time it will activate other organs to produce RBCs, which are spleen, liver and lymph nodes
- In severe cases, causes extramedullary hematopoiesis in secondary hematopoietic organs (spleen, liver and lymph nodes)
- Exceptions: anemia of renal failure, anemia of chronic inflammation



The patient here has anemia but the erythropoietin is not high



kidney is the organ responsible for erythropoietin production and the kidney here is not functioning

We have two classifications system, the 1st one is related to the cause

CLASSIFICATION ACCORDING TO CAUSE

1) Blood loss

2) Diminished RBC production

The problem in BM

- Iron deficiency anemia
- Anemia of chronic inflammation
- Megaloblastic anemia
- Aplastic anemia
- Pure red cell aplasia
- Myelophthisic anemia
- Myelodysplastic syndrome
- Anemia of renal failure
- Anemia of hypothyroidism

3) Increased destruction

(hemolytic anemia)

- Extrinsic factors (infection, antibody, mechanical)
- Intrinsic RBC abnormalities:
 - 1) Hereditary (membrane, enzyme, Hg abnormalities)
 - 2) Acquired (Paroxysmal nocturnal hematuria)

Here the BM is normal but Destruction either happens outside of the RBCs (induced by other factors eg : malaria induced hemolysis or (mechanical damage), Or it happens inside the RBC due to a preexisting defect

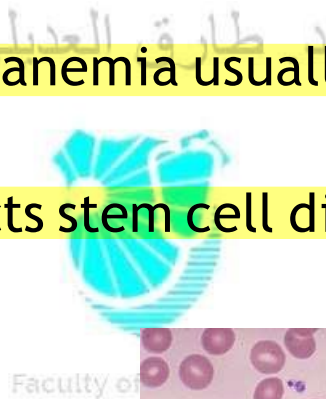
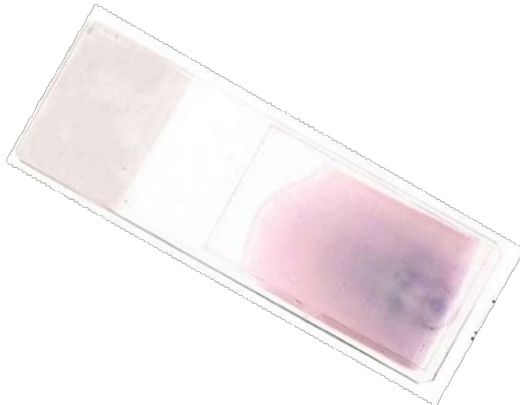
Has a lot of types and it's the most common anemia

CLASSIFICATION ACCORDING TO MORPHOLOGY

BLOOD FILM

Lymphocyte's nucleus is almost the same size of RBCs

- Size: normo, micro, macrocytic (MCV) هدول التصنيفات أخذناهم بالهستو
- Color: normo, hypochromic (MCH)
- Shape: anisopoikilocytosis (spherocytes, sickle, schistocytes) (RBC distribution width)
- Hypochromic microcytic anemia usually reflects impaired Hg synthesis
- Macrocytic anemia reflects stem cell disease and maturation



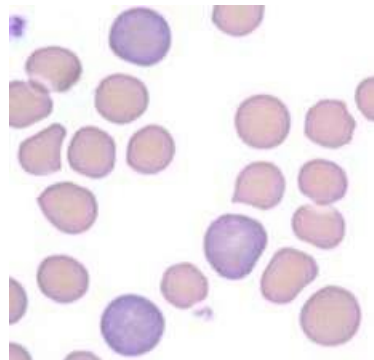
RBC INDICES

- Can be directly measured, or automated
- Slight variation is present between labs, geographic areas *If the patient elder and doesn't move the HB is less*
- Sex, age, race, mobility status have effect

In male more than female

- Reticulocyte count: helps differentiate hemolytic anemia (high) from aregenerative anemia (low) *High bcz the BM try to compensate*

Immature RBCs without nucleus



عمرهم يوم واحد

They are normally present in the blood and reflect the function of the BM

	Units	Men	Women
Hemoglobin (Hb)	g/dL	13.2–16.7	11.9–15.0
Hematocrit (Hct)	%	38–48	35–44
Red cell count	$\times 10^6/\mu\text{L}$	4.2–5.6	3.8–5.0
Reticulocyte count	%	0.5–1.5	0.5–1.5
Mean cell volume (MCV)	fL	81–97	81–97
Mean cell Hb (MCH)	pg	28–34	28–34
Mean cell Hb concentration (MCHC)	g/dL	33–35	33–35
Red cell distribution width (RDW)		11.5–14.8	

*Reference ranges vary among laboratories. The reference ranges for the laboratory providing the result should always be used in interpreting a laboratory test.



CLINICAL FEATURES OF ANEMIA

- Dizziness **With headache as a result of hypotension**
- Fatigue **Bcz there is no oxygen for the skeletal muscle**
- Pallor
- Headache

Adaptive changes:

- Tachycardia
- Tachypnea **Increase the respiration rate**
- Increased redcell 2,3-diphosphoglycerate**

If the patient has heart or lung diseases, symptoms will be worse

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<https://youtu.be/4JYqTSjMpJA?si=abhFwuBqTcueuySB>



CLINICAL SYMPTOMS IN SPECIAL TYPES OF ANEMIA

- Chronic hemolytic anemia: jaundice, pigmented gall bladder stones, red urine



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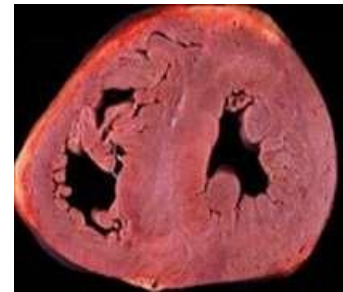
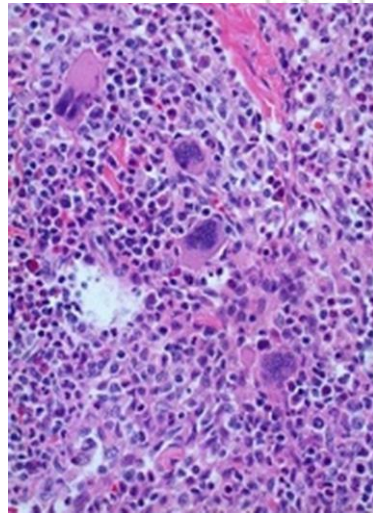


Hemolysis→ degradation of hemoglobin→ turns to bilirubin which is not soluble and gets deposited in tissue and causes the yellowish discoloration (jaundice) It also forms pigmented (black) gallbladder stones different from the cholesterol white stones, Hemoglobin also after RBCs hemolysis could pass through the kidney and cause red coloration of the urine (not bleeding)



CLINICAL SYMPTOMS IN SPECIAL TYPES OF ANEMIA

- Extramedullary hematopoiesis: splenomegaly, hepatomegaly
- **Thalassemia major** and **sickle cell anemia**: growth retardation, bone deformity, secondary hemochromatosis (damage to heart, endocrine glands)



Prolonged exposure to erythropoietin can also suppress the hormone hepcidin, which plays a crucial role in blocking iron absorption. As a result, patients may absorb excessive amounts of iron from their diet, and since the body has limited mechanisms to excrete iron (primarily through the normal shedding of epithelial cells), it can accumulate in tissues, including the heart (leading to cardiomegaly and potentially heart failure) and endocrine glands, causing physical damage and in this phase it's usually fatal (secondary hemochromatosis) And this will occur as a result of regular blood transfusion

These patients do not grow normally due to hypoxia, have enlarged abdomen (enlarged spleen and liver)

ANEMIA OF ACUTE BLOOD LOSS

- Symptoms are related to decreased intravascular volume,
- If loss is > 20% of blood volume, patient might have hypovolemic shock and death Bcz of the ischemia threat occur to
- Body responds by shifting fluid from interstitial to intravascular space, causing dilutional anemia and worse hypoxia (stays 2-3 days) As a result for dilution the HB will drop down
- Erythropoietin secretion is stimulated, activating BM erythropoiesis (needs 5-7 days)
- In internal hemorrhage, iron is restored from extravasated RBCs and used again in erythropoiesis
- In external and GIT hemorrhage, iron is lost, which complicates anemia The patient will develop iron deficiency anemia
- The anemia is normochromic normocytic, with reticulocytosis

If the patient survived, those changes will occur:

↳ the vital organs

This to keep the BP stable



ANEMIA OF CHRONIC BLOOD LOSS

Minor blood loss but at repeated times

- Occurs when the rate of RBC loss exceeds regeneration
- Mostly occurs in gastrointestinal diseases, also in excessive menstruation
Like ulcers
- Results in iron deficiency, anemia appears hypochromic and microcytic, low reticulocytes
د. طارق العيسى → Bcz the loss is outside the body



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