

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



Pathology

MID | Lecture 2

﴿وَقُلْ رَبِّ ادْخُلْنِيْ مُدْخَلَ صِدْقٍ وَّاَخْرِجْنِيْ مُخْرَجَ صِدْقٍ وَّاجْعَلْ لِّيْ مِنْ لَّدُنْكَ سُلْطٰنًا نَّصِيْرًا﴾
ربنا آتنا من لدنك رحمة وهى لنا من أمرنا رشداً

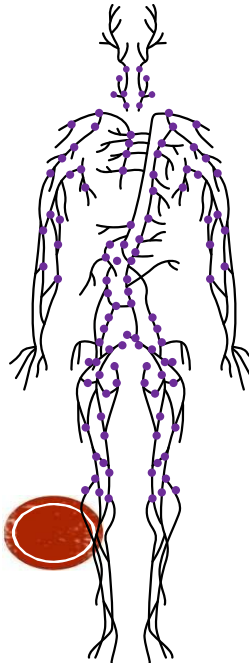
Anemia of Blood Loss

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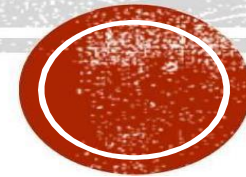


PATHOLOGY OF BLOOD AND LYMPHATIC SYSTEM

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ANEMIA



DEFINITION

.Definition of anemia is the opposite polycythemia

Anemia decrease in red blood cell mass below the normal level, which may lead to:

- ☐ Reduction of oxygen carrying capacity of blood to the systems
- ☐ secondary to decrease in red cell mass

Leads to tissue hypoxia; د. طارق العديلي

-Diagnosis by tests...again numeric diagnosis.
(symptoms alone are insufficient).

- ☐ Practically, measure by Hemoglobin concentration (g/dL), and Hematocrit (Percentage of red cell to the whole blood)

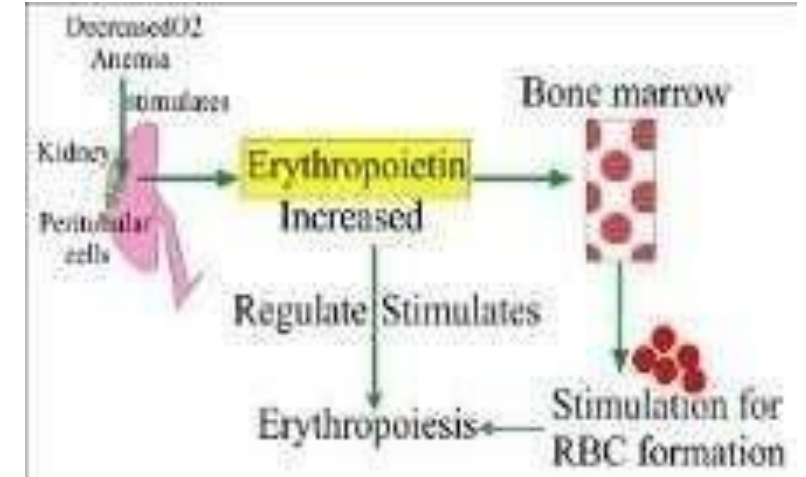
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ANEMIA AND ERYTHROPOIETIN

Anemia → hypoxia → sensed by kidneys → erythropoietin → erythropoiesis

- 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. Healthy BM can produce x5 times the amount of RBC in normal conditions, and can effectively correct anemia.
- In severe cases (or congenital anemia); erythropoietin is high but cannot reverse the anemia, erythropoiesis takes place outside the BM causes extramedullary hematopoiesis in secondary hematopoietic organs (spleen, liver and lymph nodes) so the patient will develop large spleen and liver.
- Exceptions (erythropoietin is low, مايزيد): (anemia of renal failure (kidneys don't function ...they don't secrete erythropoietin) , anemia of chronic inflammation in the next lecture.



CLASSIFICATION ACCORDING TO CAUSE

1) Blood loss

2) Diminished RBC production

(most common) ,decreased production from BM..BM is inactive and doesn't secrete enough amount from RBC.

- 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)

BM produces RBCs but they are abnormal so they die early/immaturely and thus there will be shortage of RBC in circulation

That's mean RBC undergo premature destruction instead of complete their normal lifespan.



We examine anemia according to the morphology in the blood film
(remember! Diagnosed through numerical tests)

CLASSIFICATION ACCORDING TO MORPHOLOGY BLOOD FILM

RBC is a bit smaller
than lymphocyte
nucleus

- Size of RBC: normo, micro, macrocytic (MCV)
- Color: normo, hypochromic (MCH) or hyperchromic by examining the central(thin)one third of RBC which allows light to pass through it, it will appear white.

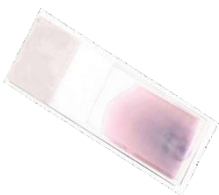
And I can know without checking them by complete blood count that they give MCV...RBC HAVE NORMAL RANGE FROM 80 TO 100 fL.... if it's less than 80 → micro ...if more than 100 →macro and if it's between 80-100 → normo

- Shape: anisopoikilocytosis different shape and size (spherocytes, sickle, schistocytes)
(RBC distribution width)
- Hypochromic microcytic anemia usually reflects impaired Hg synthesis Hg consist of heme group(iron) and globin(protein from DNA)
- Macrocytic anemia reflects stem cell disease (in bone marrow) and maturation

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It's very early
disease...they
can't mature
enough like RBC

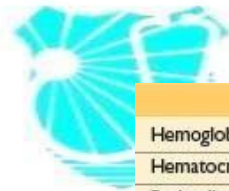
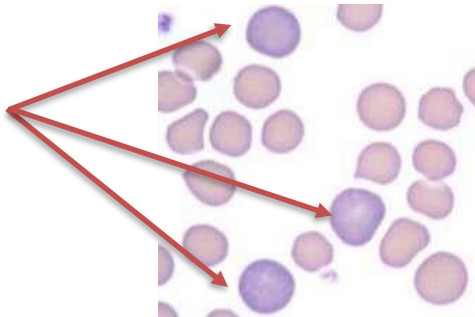


RBC INDICES

- Can be directly measured, or automated
- Slight variation is present between labs, geographic areas
- Sex, age, race, mobility status have effect
- Reticulocyte count: helps differentiate hemolytic anemia (high) from aregenerative anemia (low)

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These are Reticulocyte they are large and blue in color because of streaks of DNA so you can count them from blood film



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See the next slide

	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.



Reticulocyte count: helps differentiate hemolytic anemia (high) from aregenerative anemia (low)

Reticulocyte is an immature RBCs without nucleus. The differences between it and the mature RBCs; its size (is larger) and has streaks of residual DNA.

It is found in bone marrow and in peripheral blood, it's life as Reticulocyte is one day and then it will become a mature RBC.

The normal percentage of Reticulocytes is 1% if it's more this indicates that bone marrow is active and produce extra RBCs to compensate anemia (usually hemolytic anemia).

Anemia + reticulocytes → healthy (functional) BM. {RBCs are the problem/cause}

Anemia + x reticulocytes → nonfunctional BM. {BM is the problem/cause}

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CLINICAL FEATURES OF ANEMIA

General symptoms for anemia

- ☐ Dizziness (hypotension) In polycythemia hypertension
- ☐ Fatigue Loss of energy and tiredness as a result of hypoxia of skeletal system
- ☐ Pallor In polycythemia plethora which is the skin is red in color
We can examine the pall skin in highly vascular areas like finger nails , oral , lips and tongue to make sure the patient is pallor
- ☐ Headache

Adaptive changes: the body tries to compensate the hypoxia

- ☐ Tachycardia High heart rate to make oxygen reach the body faster
- ☐ Tachypnea Rapidly breathing
- ☐ Increased red cell 2,3-diphosphoglycerate

Remember the biochemistry lecture

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

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CLINICAL SYMPTOMS IN SPECIAL TYPES OF ANEMIA

□ Chronic hemolytic anemia:

The RBCs are degraded, the concentration of hemoglobin will increase and will be metabolized into bilirubin (it's an insoluble compound eliminated through the biliary system), if the concentration of bilirubin is increased the liver can't handle it and will be deposited in the tissues.

jaundice, pigmented gall bladder stones, red urine



The sclera of the eye will be yellow



Because of the insolubility of bilirubin it will accumulate in the biliary system and cause black bile stones



The hemoglobin will reach the kidney and will go with so its color will be red like blood



CLINICAL SYMPTOMS IN SPECIAL TYPES OF ANEMIA

- Extramedullary hematopoiesis: splenomegaly, hepatomegaly
- Thalassemia major and sickle cell anemia : (they are Congenital - life long disease) the concentration of erythropoietin is chronically high which leads to extramedullary hematopoiesis, the patients are children because it's Congenital.

Persistent hypoxia even high erythropoietin can't reverse it, it's genetic, the body can't make Hg, it's persistent anemia.

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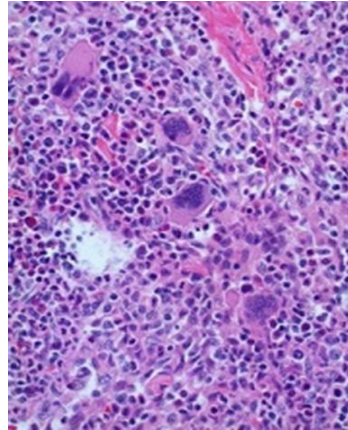
growth retardation, bone deformity especially face bones they are protruded and large regarding to increase erythropoietin so the bone marrow will increase.

secondary hemochromatosis the cure is **blood transfusion** to increase blood concentration and decrease the hypoxia. as we know the blood contains iron and it's difficult to rid from, so the only method is to shed epithelial cells, because if the iron accumulates in the tissues, it becomes toxic and causes damage to heart (cardiomegaly as a result of hemochromatosis), endocrine glands (any gland can be destroyed by iron)

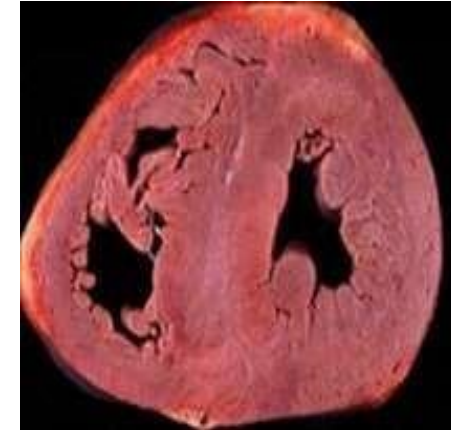




Large abdomen due to
hepato/splenomegaly.
Growth retardation;
they're short.
Skeletal deformities.



Bone biopsy;
erythropoietic cells.



Very thick wall

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^{Sudden} ANEMIA OF ACUTE BLOOD LOSS

Symptoms are related to decreased intravascular volume,

If loss is >20% of blood volume, patient might have hypovolemic shock especially vital organs like brain, kidney they suffer from ischemia and death

Body responds by shifting fluid from interstitial to intravascular space, causing dilutional anemia which cause decreasing in the concentration of hemoglobin and worse hypoxia (stays 2-3 days)

Erythropoietin secretion is stimulated, activating BM erythropoiesis (needs 5-7 days)

Iron is difficult to gain (the absorption is limited)and difficult to get rid of so if hemorrhage happened:

In internal hemorrhage, iron is restored from extravasated RBCs and used again in erythropoiesis

In external and GIT hemorrhage, iron is lost, and the patient will develop iron deficiency which complicates anemia and it takes a lot of time to appear

The anemia is normochromic normocytic, with reticulocytosis



ANEMIA OF CHRONIC BLOOD LOSS

Loss of small amount of blood but it is repeated

- ❑ Occurs when the rate of RBC loss exceeds regeneration
- ❑ Mostly occurs in gastrointestinal diseases, also in excessive menstruation
- ❑ Results in iron deficiency, anemia appears hypochromic and microcytic (depletion in iron stores → anemia starts to fully develop), low reticulocytes also due to ↓ iron stores, the BM can't produce RBCs.



Causes of hemorrhage in gastrointestinal diseases:

Peptic Ulcers, stomach and esophagus cancers, hemorrhoids,



Pathology Quiz



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For any feedback, scan the code or click on it.



Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	16	No quiz  	Added quiz
V1 → V2		 Faculty of Medicine	