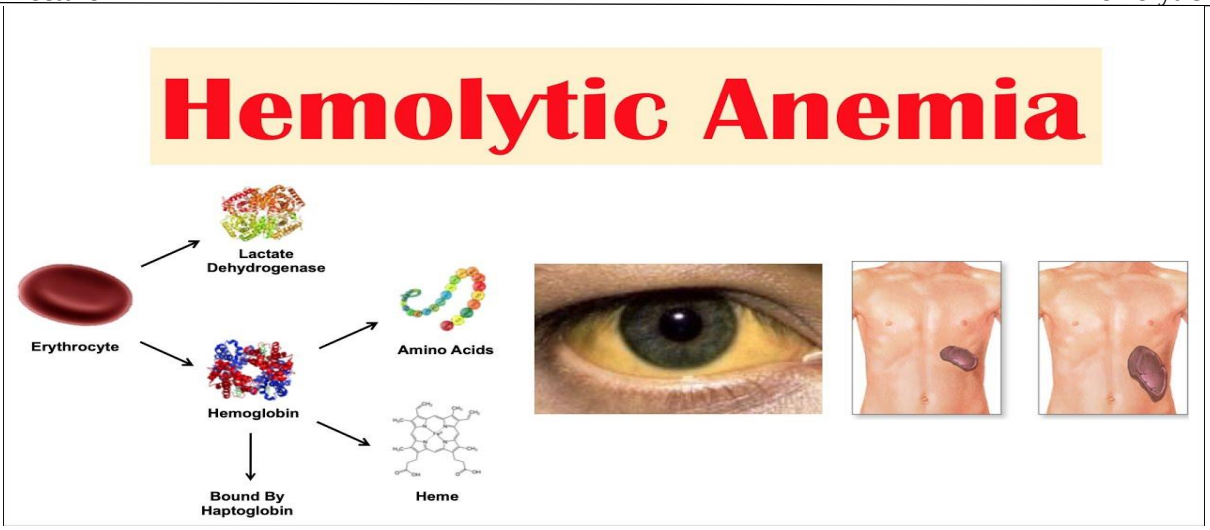


It is a form of anemia, occur after abnormal breakdown of red blood cells, either in the blood vessels (intravascular hemolysis) or in the human body (extravascular), represented 5% of all existing anemias. This most commonly occurs within the spleen, but also can occur in the reticuloendothelial system or mechanically, and **bilirubin released after hemoglobin destroyed which gives the skin and eyes a yellowish color.**

There are two main mechanisms whereby red cells are destroyed in hemolytic anemia:

1-Intravascular hemolysis

Red blood cell was broken down directly in the circulation in a process known intravascular hemolysis, free hemoglobin is released which rapidly attached and saturates plasma protein called **haptoglobins** and the excess free hemoglobin is filtered by the glomerulus. If the rate of hemolysis saturates the renal tubular re absorptive capacity, free hemoglobin enters urine.

2-Extravascular hemolysis

Red cell destruction usually occurs after a mean lifespan of 120 days, when the cells are removed extravascularly by the macrophages of the **reticuloendothelial (RE) system**, especially in the marrow but also in the **liver** and **spleen.**

The breakdown of **heme** from red cells liberates iron for recirculation via plasma transferrin to marrow erythroblasts, **protoporphyrin** which is broken down to bilirubin. It circulates to the liver after conjugated to glucuronides which are excreted into the gut via bile and converted to stercobilinogen and stercobilin (excreted in faeces)

Globin chains are broken down to amino acids which are reutilized for general protein synthesis in the body.

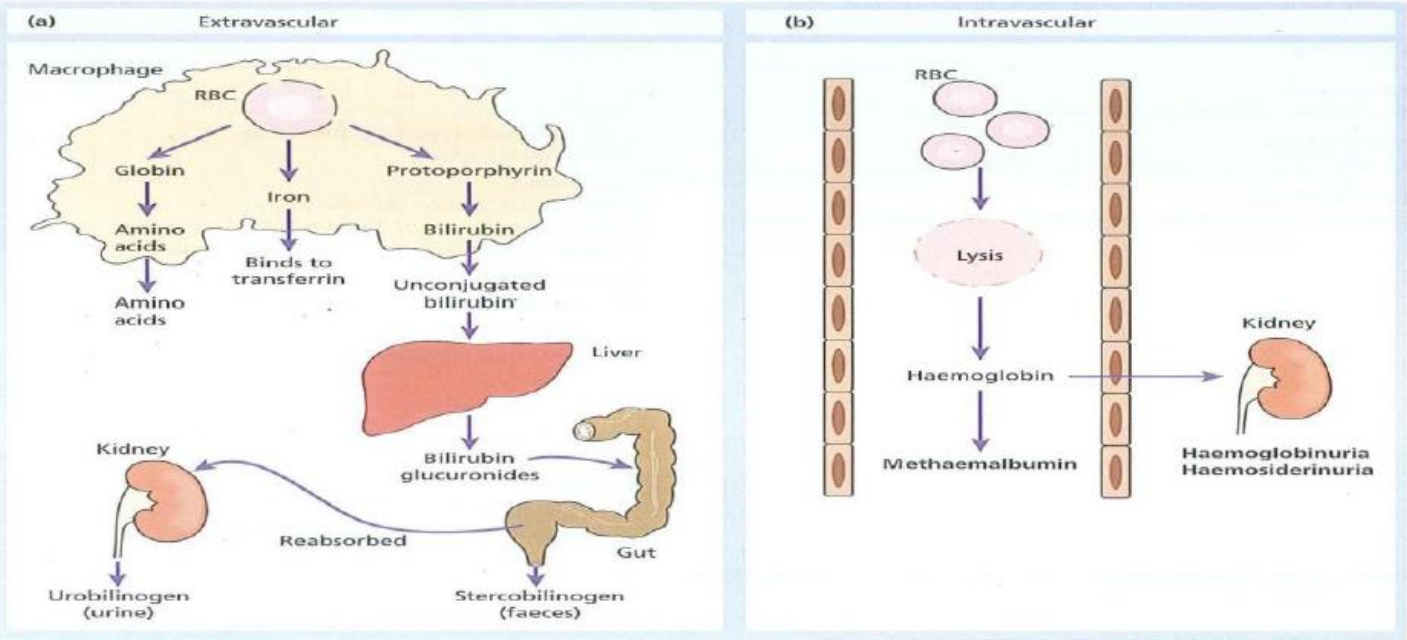


Figure 1 extravascular and intravascular of RBC.

Hemolytic anemia classification:

A- Inherited hemolytic anemia

It is genetic, without therapy and permanent, that are start early in the life, but some start after 7 month of the birth and some needs other factors to starting.

Types of Inherited hemolytic anemia called Hemoglobinopathies:

1-Quantitative defects: absence of one or two or three or four chain of hemoglobin (ex: **thalassemia, sickle-cell disease)**

2-Structure defects: the abnormality in the sequence of amino acids on the beta chains of hemoglobin only

3-Enzymopathies: absence or decreased function of a metabolic enzymes (ex: **G6PD** or **PK**)

4-Membrane defects: abnormalities in the proteins that make up the cytoskeleton of the cell membrane (ex: **hereditary spherocytosis**)

B-Acquired hemolytic anemia

Destruction of red blood cells (RBCs) not due to genetic or congenital disorder that has therapy, acute and not permanent, that starting late in the life. It caused by factors during the life such as: **Malaria, antibodies against RBC (auto-immunity), toxins, chemicals** and **some drugs.**

Three proteins are removed toxic iron from the plasma, that release after RBC hemolysis:

1-Haptoglopin (HP)

The first protein which remove the toxic iron immediately after hemolysis, it is Alpha protein produced from the liver.

2-Hemopexin:

The **second** protein which remove the toxic iron, its work start after the depleted of haptoglobin It is beta protein also produced from liver.

3-Meth-hem-albumin.

A. if the haptoglobin and hempexin depleted and the hemolysis is severing the free iron compound with **albumin** and called **methhemalbumin**

B. when the liver produces new **haptoglobin and hemopexin** they take the toxic iron from albumin to carry it into the liver.

(Symptoms of hemolytic anemia are similar to the general signs of anemia.)

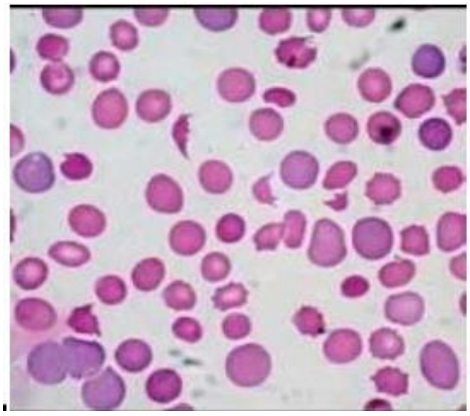
Diagnosis: Two main principles

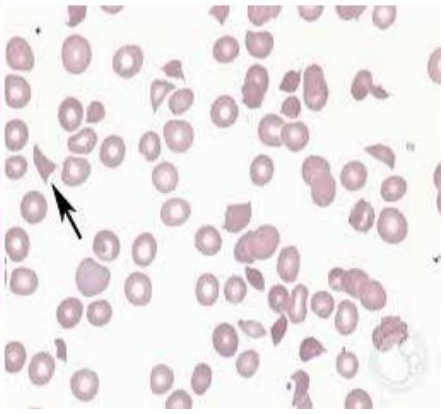
a. One is to confirm that it is hemolysis

b. Two is to determine the cause

Laboratory findings of hemolytic anemia:

1. CBC (anemia).
2. Reticulocyte count.
3. Plasma hemoglobin (Hb) **(increased).**
4. Serum Haptoglobin (HP) **(absent).**
5. Serum Hemopexin **(absent).**
6. Meth-hem-albumin **(positive).**
7. Serum bilirubin.
8. Blood film.





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