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Opinion Article

Differential Diagnosis of Hemolytic Anemia and its Clinical Signs on Peripheral Blood Smear

Rein Vollix*

Department of Hematology, University of California, California, United States of America

DESCRIPTION

Hemolytic anemia is a form of anemia that occurs when Red Blood Cells (RBCs) are destroyed faster than they can be replaced by the bone marrow RBCs are essential for carrying oxygen from the lungs to the rest of the body. When they are destroyed, the oxygen delivery is impaired and the body becomes starved of oxygen.

Classification

Hemolytic anemia can be classified into two main types:

Intrinsic hemolytic anemia: Intrinsic hemolytic anemia is caused by defects in the RBCs themselves, such as abnormal shape, structure, or function. These defects can be inherited or acquired. Some examples of inherited intrinsic hemolytic anemia are sickle cell anemia, thalassemia, and deficiency. These conditions affect the hemoglobin, the protein that carries oxygen in the RBCs, or the enzymes that protect the RBCs from oxidative damage. Some examples of acquired intrinsic hemolytic anemia are Paroxysmal Nocturnal Hemoglobinuria (PNH) and pyruvate kinase deficiency. These conditions affect the membrane or metabolism of the RBCs, making them more prone to destruction.

Extrinsic hemolytic anemia: Extrinsic hemolytic anemia is caused by factors outside the RBCs that damage or destroy them. These factors can be immune or non-immune. Immune-mediated hemolytic anemia occurs when antibodies or complement proteins attach to the surface of the RBCs and mark them for destruction by the spleen, liver, or other organs.

This can happen in autoimmune disorders, such as Systemic Lupus Erythematosus (SLE), where the body produces antibodies against its own RBCs in transfusion reactions, where the recipient's antibodies attack the donor's RBCs, or in drug-induced hemolytic anemia, where certain medications trigger antibody production against the RBCs. Non-immune-mediated hemolytic anemia occurs when physical or mechanical forces damage or rupture the RBCs.

Infections such as

- Malaria or sepsis; in tumors or enlarged spleen that compress or trap the RBCs;
- In prosthetic heart valves that shear or fragment the RBCs;
- In Microangiopathic Hemolytic Anemia (MAHA), where small blood clots obstruct and cut through the RBCs.

The diagnosis of hemolytic anemia is based on clinical features, laboratory tests, and peripheral blood smear findings. Clinical features include signs and symptoms of anemia and hemolysis, such as jaundice, dark urine, splenomegaly, and gallstones.

Laboratory tests include Complete Blood Count (CBC), reticulocyte count, Lactate dehydrogenase (LDH), bilirubin, haptoglobin, and Direct Antiglobulin Test (DAT). CBC shows low hemoglobin and hematocrit levels, indicating anemia; low Mean Corpuscular Volume (MCV) and Mean Corpuscular Hemoglobin Concentration (MCHC), indicating microcytic or hypochromic anemia; and high Red Cell Distribution Width (RDW), indicating variation in RBC size. Reticulocyte count shows high levels of immature RBCs, indicating increased bone marrow production to compensate for hemolysis and it shows high levels of enzyme released from damaged RBCs. Bilirubin shows high levels of pigment derived from hemoglobin breakdown.

Haptoglobin shows low levels of protein that binds to free hemoglobin in plasma. Direct Antiglobulin Test (DAT) shows positive result if antibodies or complement are attached to RBCs. Peripheral blood smear shows abnormal morphology of RBCs, such as spherocytes (small round cells with no central pallor), schistocytes (fragmented cells with irregular shapes), sickle cells (crescent-shaped cells), target cells (cells with a central dot), bite cells (cells with a semicircular defect), or blister cells (cells with a vacuole). The peripheral blood smear can also show parasites, such as malaria or babesia, inside the RBCs.

Correspondence to: Rein Vollix, Department of Hematology, University of California, California, United States of America, E-mail: rein00@ac.edu

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