



Genetic and Acquired Factors in Hemolytic Anemia Pathology

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About the study

Hemolytic anemia is a diverse group of disorders characterized by the premature destruction of Red Blood Cells (RBCs) and the bone marrow's incapacity to replace them. This condition results in a reduced lifespan of RBCs, leading to anemia, which manifests with symptoms such as fatigue, pallor, and shortness of breath. The pathology of hemolytic anemia involves intricate processes within and outside the red blood cells, encompassing various genetic and acquired factors. An RBC has a lifespan of about 120 days on average. These cells develop and acquire their distinctive biconcave form in the bone marrow, where they first appear. Once released into the bloodstream, RBCs circulate, delivering oxygen to tissues and organs. Aging RBCs undergo senescence, becoming more rigid and less deformable. Eventually, macrophages in the spleen and liver phagocytose these aged cells, recycling their components.

Hemolytic anemia can be classified into intrinsic and extrinsic types, depending on whether the defect lies within the RBC itself or is caused by external factors. Inherited disorders such as sickle cell anemia, thalassemia, and hereditary spherocytosis result in intrinsic defects within the RBC membrane, hemoglobin structure, or enzyme deficiencies. These abnormalities compromise the structural integrity and function of RBCs, leading to premature destruction. Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency is a common enzymatic disorder causing intrinsic hemolysis. G6PD is crucial for protecting RBCs from oxidative stress. Deficient individuals are more susceptible to hemolysis triggered by certain medications, infections, or oxidative agents.

Conditions like hereditary spherocytosis and elliptocytosis involve defects in the RBC membrane, rendering cells vulnerable to destruction. The altered membrane makes RBCs more susceptible to

shear stress, increasing their fragility. Autoimmune Hemolytic Anemia (AIHA) and drug-induced immune hemolytic anemia are examples of extrinsic hemolysis. Drugs can induce hemolysis by forming immune complexes or directly affecting RBC membranes. Conditions such as Microangiopathic Hemolytic Anemia (MAHA) involve physical damage to RBCs as they pass through narrowed blood vessels. This can occur in diseases like Thrombotic Thrombocytopenic Purpura (TTP) and Hemolytic Uremic Syndrome (HUS), where microthrombi shear RBCs.

Some infections, such as malaria, trigger hemolysis by invading RBCs. Throughout its life cycle, the parasite grows inside red blood cells, breaking them to release merozoites that continue the infection and hemolysis cycle. The consequences of hemolysis extend beyond the mere reduction in RBC count. As RBCs break down, they release hemoglobin into the bloodstream, which can surpass the scavenging capacity of haptoglobin and lead to the formation of free hemoglobin. Free hemoglobin, in turn, can cause oxidative damage to tissues and organs, particularly the kidneys. The release of heme during hemolysis contributes to the formation of bilirubin, which, when accumulated, can lead to jaundice.

The clinical presentation of hemolytic anemia is varied and depends on the underlying cause, severity of hemolysis, and the compensatory response of the bone marrow. Common symptoms include fatigue, pallor, jaundice, and shortness of breath. Splenomegaly may be present in conditions where the spleen is actively involved in the destruction of RBCs. Laboratory findings in hemolytic anemia typically reveal anemia, reticulocytosis, and elevated levels of Lactate Dehydrogenase (LDH) and indirect bilirubin. The peripheral blood smear often shows features indicative of hemolysis, such as schistocytes in conditions like MAHA.

Accurate diagnosis of hemolytic anemia involves a thorough clinical evaluation, laboratory tests, and sometimes specialized studies. Treatment strategies aim to address the underlying cause, alleviate symptoms, and prevent complications. In autoimmune hemolytic anemia, immunosuppressive therapies may be employed. Patients with enzyme deficiencies, such as G6PD deficiency, are advised to avoid triggers like certain medications. Folate supplementation is often recommended to support increased erythropoiesis in hemolytic anemia. The diverse mechanisms underlying hemolysis necessitate a meticulous diagnostic approach and tailored treatment strategies.