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CLINICAL AND LABORATORY DIAGNOSTICS OF HEMOLYTIC ANEMIA

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Abstract
The relevance of laboratory diagnosis of hemolytic anemia lies in the high level of their occurrence, as well as the complexity of the clinical picture, which requires accurate and timely detection. Hemolytic anemia is a group of diseases characterized by accelerated destruction of red blood cells, which leads to a lack of oxygen in organs and tissues, and also causes a variety of complications.

Keywords: Hemolytic anemia, hereditary and acquired, laboratory research methods, treatment.

Introduction

Hemolytic anemias are a group of diseases characterized by the destruction of erythrocytes (red blood cells) before their normal life span has expired, which leads to a decrease in the level of hemoglobin in the blood and, as a consequence, to a lack of oxygen supplied to tissues and organs. The etiology of hemolytic anemia can be diverse and includes both hereditary and acquired factors. Hereditary hemolytic anemias are often caused by genetic mutations that affect the structure and function of red blood cells. Examples of such diseases include sickle cell anemia and thalassemia, where an abnormal form of hemoglobin or a lack of it leads to premature destruction of red blood cells [1, 13, 18].

Acquired hemolytic anemias can occur as a result of autoimmune processes, where the body's immune system begins to attack its own red blood cells, or as a result of infections such as malaria. In addition, toxic exposures such as drugs or chemicals may also contribute to hemolysis.

Understanding the etiology of hemolytic anemia is of great importance for diagnosis and selection of adequate treatment, which, in turn, improves the quality of life of patients.

The classification of hemolytic anemia is based on several key criteria: the mechanism of destruction of red blood cells, the etiological factor and the localization of the pathology. According to the mechanism of destruction, intravascular and extravascular hemolysis are distinguished [1, 15, 16]. Intravascular hemolysis occurs as a result of various factors, such as

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mechanical trauma, infection or autoimmune reactions, while extravascular hemolysis occurs predominantly in the spleen and liver.
From the point of view of etiology, hemolytic anemias are divided into hereditary and acquired. Inherited forms include diseases such as spherocytic anemia and thalassemia, while acquired forms may be associated with infections, drugs, or autoimmune processes [1, 12, 19].
Another important aspect is the clinical picture, which can vary from mild anemia to severe forms requiring immediate treatment.
The pathogenesis of these diseases is multifaceted and can be caused by both hereditary and acquired factors.
Hereditary hemolytic anemias, such as spherocytic anemia or thalassemia, are caused by genetic defects that lead to abnormalities in the structure of the red blood cell membrane or hemoglobin synthesis. These abnormalities make cells more susceptible to mechanical injury and ultimately to phagocytosis by macrophages in the spleen and liver.
Acquired forms, such as autoimmune hemolytic anemia, develop as a result of the immune system reacting against its own red blood cells. Autoantibodies can lead to opsonization and subsequent destruction of red blood cells.
It is also important to consider mechanisms related to infections, toxins, and drugs that can initiate hemolysis. All this emphasizes the complexity and diversity of the pathogenetic mechanisms of hemolytic anemias [1, 8, 10].
The clinical symptoms of these conditions are varied and depend on the degree of anemia, the rate of cell destruction and concomitant diseases. The main manifestations include general weakness, fatigue, pallor of the skin and mucous membranes, as well as shortness of breath during exercise.
In later stages of the disease, specific symptoms may develop, such as jaundice, caused by increased levels of bilirubin in the blood, as well as an enlarged spleen and liver, which is associated with the active destruction of red blood cells. These changes may manifest as abdominal pain and discomfort. It is important to note that clinical manifestations may vary depending on the etiology of hemolytic anemia - both autoimmune and hereditary forms have their own unique features [3, 9, 11].
Laboratory tests play a key role in diagnosis: determination of hemoglobin, reticulocyte and bilirubin levels, as well as shunt tests and the presence of anti-erythrocyte antibodies help the doctor assess the extent and causes of hemolysis.
The key laboratory tests for diagnosing hemolytic anemia are hemoglobin and hematocrit tests, as well as reticulocyte and bilirubin levels. Elevated levels of indirect bilirubin and reticulocytes may indicate active hemolysis. Coagulation status should also be assessed and tests for the presence of antibodies to red blood cells, such as direct and indirect Coombs tests, should be activated [3, 14, 17].
Additional tests, such as hemoglobin electrophoresis and testing for red blood cell membrane defects, can help confirm the diagnosis and identify specific types of hemolytic anemias.
In addition to the tests described above, it is important to consider immunoagglutination and ferritin levels, since changes in these indicators may indicate various forms of hemolytic anemia. For example, a decrease in ferritin levels may indicate concomitant iron deficiency anemia, which is often observed in patients with chronic hemolysis.

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Molecular genetic studies are becoming increasingly relevant in the diagnosis of hereditary forms of hemolytic anemia, such as sickle cell anemia and thalassemia. Identification of mutations in the genes responsible for the synthesis of globins or erythrocyte membrane proteins makes it possible to establish the exact cause of the disease and, therefore, clarify the treatment strategy [2, 5, 7].

Treatment for hemolytic anemias depends on the cause and may include corticosteroids, immunosuppressants, blood transfusions, or even a splenectomy - the removal of the spleen, which helps reduce the destruction of red blood cells. In the case of hereditary forms, such as spherocytic anemia, the main method is splenectomy, which can significantly reduce hemolysis by removing the spleen, where red blood cells are destroyed. Therapy for thalassemia often includes regular blood transfusions and the use of drugs that capture excess iron to prevent iron overload [1, 4, 6].

Acquired forms of hemolytic anemia require a special approach. For example, in autoimmune hemolytic anemia, the use of corticosteroids to suppress the immune response may be indicated. In some cases, immunosuppressants or new therapies such as CD20 blockers are used to help reduce the amount of autoantibodies.

Conclusions. Modern advances in laboratory medicine, such as automated analyzers and molecular genetic methods, have significantly increased the accuracy and speed of diagnosis. This, in turn, allows doctors to make treatment decisions more quickly, improving the prognosis for patients and reducing the risk of serious complications. Prevention of these factors, as well as timely response to symptoms, can significantly improve the quality of life of patients. Finally, scientific research continues to expand our knowledge of the pathogenesis of hemolytic anemias, which opens new horizons for the development of more effective treatments.

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