

ETIO-PATHOGENETIC ASPECTS OF THROMBOCYTOPENIA

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Abstract

Thrombocytopenia, characterized by a decrease in the level of platelets in the blood, is an urgent and multifaceted problem in modern medicine. It can result from a variety of causes, including decreased platelet production in the bone marrow, increased platelet destruction, or sequestration in the spleen. It is important to note that thrombocytopenia can serve as a marker for various diseases, such as infections, autoimmune disorders, and even malignancies. This problem requires an interdisciplinary approach involving both hematologists and other specialists, which emphasizes the importance of a comprehensive understanding of thrombocytopenia to improve the quality of life of patients.

Keywords: Thrombocytopenia, pathogenesis, autoimmune thrombocytopenia, antiplatelet antibodies, immunological diagnostics.

Introduction

Thrombocytopenia is a condition characterized by a decrease in the number of platelets in the blood, which can lead to a variety of complications. Platelets, or thrombocytes, play a key role in the blood clotting process, and their deficiency can cause an increased tendency to bleed and bruise. There are many causes of thrombocytopenia, one of the main ones is a disruption of their production in the bone marrow. This can occur in diseases such as aplastic anemia or myelodysplastic syndromes, where normal hematopoiesis is disrupted.

The second group of causes is increased platelet loss, which can occur with thrombocytopenic purpura or as a result of massive blood loss. Also, in the case of infectious diseases, such as viral hepatitis or HIV, platelets can be destroyed faster than usual.

An equally important cause is autoimmune disorders, where the immune system mistakenly attacks platelets. Certain medications, including antibiotics and anti-inflammatory drugs, can also cause thrombocytopenia [1, 5, 6].

The pathogenesis of thrombocytopenia is a complex process based on the interaction of various factors leading to a decrease in the level of platelets in the blood. One key cause is increased





platelet destruction, which can occur in autoimmune diseases such as idiopathic thrombocytopenic purpura, where the immune system mistakenly attacks its own cells.

Another mechanism involves insufficient platelet production in the bone marrow, which can be caused by various diseases, including hypoplastic anemia, infectious processes, or exposure to toxic substances.

In addition, thrombocytopenia may develop as a result of platelet sequestration in the spleen, which is often encountered in diseases accompanied by an enlargement of the organ. The interaction of these factors leads to clinical manifestations of thrombocytopenia, such as bleeding, bruising and other hemorrhagic disorders, which requires careful diagnosis and timely treatment.

The classification of thrombocytopenias is based on the etiology, mechanism of development and platelet levels.

Autoimmune thrombocytopenias: occur as a result of the body's immune response, such as in idiopathic thrombocytopenic purpura (ITP).

Microangiopathic thrombocytopenia: Typically seen in diseases such as thrombotic thrombocytopenic purpura or hemolytic uremic syndrome, where platelets are destroyed in small vessels.

Hypoproliferative thrombocytopenia: develops due to insufficient production of platelets in the bone marrow, which can occur with aplastic anemia or as a result of exposure to toxic substances.

The clinical symptoms of thrombocytopenia are varied and depend on the degree of reduction in the number of platelets, as well as on the causes of this condition. The most characteristic manifestations include an increased tendency to bleeding, which can manifest as petechiae, ecchymosis and nosebleeds. Patients often experience minor bruising on the skin, which can occur even without significant trauma [3, 10, 16].

In severe cases, thrombocytopenia can lead to serious complications such as hemorrhagic syndrome, which is accompanied by profuse internal and external bleeding. It is important to note that thrombocytopenia can be asymptomatic, especially in mild forms. Therefore, testing for platelet disorders becomes mandatory in the presence of other syndromes, such as anemia or inflammatory processes.

It is important to note that thrombocytopenia can be asymptomatic, especially in mild forms. Therefore, testing for platelet disorders becomes mandatory in the presence of other syndromes, such as anemia or inflammatory processes. The first step is to determine the number of platelets, their morphology, and their relationship to other blood cells. Platelets, or thrombocytes, play a key role in hemostasis, and their deficiency can lead to serious complications, including platelet-related bleeding [2, 11, 15]. Platelet count is usually assessed as part of a complete blood count using automated analyzers. When thrombocytopenia is detected, it is necessary to determine not only the quantitative indicators, but also the morphology of platelets, which allows identifying possible causes of their decrease. Changes in the shape, size and structure of platelets may indicate various pathologies, such as aplastic anemia, myelodysplastic syndromes or infectious diseases [1, 7, 19]. For more in-depth analysis, microscopic examination of stained blood smears may be required to provide information on platelet quality and functionality and to detect the presence of abnormal cell forms.





To better understand the mechanisms of thrombocytopenia, additional tests may be performed: immunological studies to detect autoimmune diseases, tests for infections such as viruses (eg, HIV, hepatitis), and molecular genetic tests.

Immunological studies of thrombocytopenia are an important aspect in the diagnosis and treatment of diseases associated with disorders of the hematopoietic system. Thrombocytopenia, characterized by a decrease in the level of platelets in the blood, can be caused by various reasons, including idiopathic, autoimmune and infectious factors [3, 9, 17].

The most important direction in this context is the determination of the presence of anti-platelet antibodies, which helps to identify the autoimmune nature of the disease. To conduct such studies, various methods are used, such as enzyme immunoassay and cytometry, which allow for the reliable determination of the presence of antibodies and assessment of their impact on platelet function [1, 13].

In addition, it is important to conduct a differential diagnosis by excluding other causes of thrombocytopenia, such as hyposplenism or drug reactions.

Molecular genetic testing of thrombocytopenias is an important tool for diagnosing and understanding the various pathogenetic mechanisms that lead to decreased platelet counts. Thrombocytopenia can have many causes, including autoimmune disorders, inherited diseases, infections, and drug side effects. Molecular genetic testing of thrombocytopenias is an important tool for diagnosing and understanding the various pathogenetic mechanisms that lead to decreased platelet counts. Thrombocytopenia can have many causes, including autoimmune disorders, inherited diseases, infections, and drug side effects.

It has been noted that a number of patients with thrombocytopenia have mutations in genes such as MPL, CSFR and THPO, which are responsible for the regulation of thrombocyto-genesis.

The prognosis of thrombocytopenia depends on many factors, including the etiology of the disease, the severity of thrombocytopenia, and the patient's response to therapy. The main types of thrombocytopenia, such as primary and secondary, require different approaches to treatment.

In the case of primary thrombocytopenia associated with autoimmune processes, the prognosis may be favorable with timely diagnosis and the use of immunosuppressive therapy. As for secondary forms caused by infections, drugs or other diseases, the prognostic factor may vary depending on the possibility of eliminating the underlying cause [3, 12, 14].

In addition, platelet count, underlying medical conditions, and overall health of the patient also play a key role in determining outcome. Regular monitoring and individualized treatment can significantly improve the quality of life of patients suffering from thrombocytopenia. It is important to remember that each case is unique and requires a comprehensive analysis by a qualified specialist.

Conclusions

Thus, the complexity of diagnosis and treatment of this condition is explained by its diverse etiological factors. In recent years, scientists have continued to study the molecular mechanisms underlying the pathogenesis of thrombocytopenia, which, in turn, opens new horizons for the development of effective therapeutic approaches.





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