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ALLOIMMUNE THROMBOCYTOPENIA AFTER BLOOD TRANSFUSION: PATHOGENESIS, DIAGNOSIS AND CLINICAL FEATURES

Ergasheva Hilola Erkinovna

Student of the 3rd year of 344 group of medical faculty

Parahatova Aidana Ruslanovna

Student of the 3rd year of 344 group of medical faculty

Uzokova Oyzhamol Narzullaevna

Scientific supervisor: Assistant of Hematology Department

Abstract: Alloimmune thrombocytopenia, which occurs after blood transfusion, is one of the few diseases encountered in modern medicine and remains an important problem requiring attention to patients in need of blood transfusions.

Keywords:platelets, alloimmune thrombocytopenia, thrombocytopenia after blood transfusion, antigens.

Introduction. In the modern twenty-first century, people are not often transfused blood without proper examination and tests. But still there are cases when it is necessary to perform emergency blood transfusion without tests for individual compatibility of blood of donor and recipient. In case of huge blood loss, blood disease, labor complications, severe anemia for any reason, blood transfusions can be performed. It is the hemolytic reaction that poses a threat, when incompatible blood components are transfused, in the presence of sufficient existing alloantibodies in the recipient's blood. Some studies have shown that transfusion of incompatible blood is not dangerous. According to world statistics, this pathology is diagnosed in 1-2% of patients. Overall, the incidence of ATTP after blood transfusion is about 1 case per 20,000 transfusions (0.005% of cases) in countries with developed medical systems. In the United States, there are between 300 and 500 new cases of transfusion-related alloimmune thrombocytopenia each year (according to a study published in the journal o "Transtfusion" in 2018). In Europe, the rate ranges from 0.003% to 0.007% for every 1000 transfusions performed.

The aim of this review is to study and familiarize ourselves with the pathogenesis, diagnosis and clinical features of alloimmune thrombocytopenia in patients after blood transfusion.

Basic information. Alloimmune thrombocytopenia, which occurs after transfusion of platelet mass, is a rare but dangerous complication in which the patient's immune system produces antibodies against antigens present on donor platelets. The cause of the disease is often thought to be antigens formed by the same species in the body. Which then prompts an immune response not produced by it. These alloantigens can be platelet, erythrocyte and leukocyte

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antigens. In contrast to autoimmune thrombocytopenia, this form of the disease develops due to the formation of antibodies directed against specific antigens on the platelet surface (e.g., HPA-1a), which leads to their destruction and a decrease in the level of platelets in the blood. As we know the number of platelets in the norm is 150-300x109/l, depending on sex can vary. If this number is reduced to 50x109/l, hemorrhagic syndrome may occur. With life-threatening bleeding, the platelet count falls below 20x109/L, which requires immediate hospitalization of the patient. Alloimmune thrombocytopenia is most often caused by transfusion of incompatible platelet mass.

The pathogenesis of the disease involves the recipient's immune response to unfamiliar donor platelet antigens. This process leads to a decrease in the number of platelets in the patient's body, which significantly slows down the restoration of normal levels of cells in the blood. Human platelet antigens (HPA) such as HPA-1a, HPA-5b, and others are the most common consequence of the disease.

Diagnosis of alloimmune thrombocytopenia involves several aspects:

- medical history is necessary, especially if the patient has had previous negative reactions to donated blood;
- clinical symptoms: spot hemorrhages and bleeding;
- laboratory tests: general blood count, serologic tests, HPA (determination of antibodies to platelet antigens) and others.

Clinical manifestations of the disease may vary from mild thrombocytopenia, subcutaneous hemorrhages, nosebleeds, menorrhagia to severe bleeding, threatening the life of the patient. Also, the patient easily bruises even with minor herbs, and of course fatigue and weakness due to anemia associated with bleeding. Rarely, enlargement of the liver and spleen may be observed.

Timely diagnosis and correct treatment (e.g., selection of donor platelets with minimal antigenic mismatch) play a key role in successful treatment. In some cases, the use of immunosuppressors or antibodies aimed at suppressing anti-platelet antibody production may be necessary.

Conclusion: Alloimmune thrombocytopenia after transfusions is an important clinical condition that requires attention from both clinicians and researchers. In countries with developed medical systems, efforts are being made to reduce the incidence of these complications, including through the use of donor and recipient platelet typing techniques.

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