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IMMUNE CONFLICT DEPENDING ON THE ANTIGENIC STRUCTURE OF ERYTHROCYTES

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Some of the more than 300 antigens of human erythrocyte membrane antigens are combined into 23 genetically controlled blood group systems (ABO, Rh-Hr, Dafie, M, N, S, Levy, Diego) [1,2,3].

Keywords: *antigen system, erythrocytes, blood group, antigen, antibodies, genes;*

The AVO system of red blood cell antigens contains natural anti-A and anti-B antibodies in serum. The genetic locus controlling the formation of antigens of this system is located in the long arm of the 9th chromosome and is represented by the H, A, B and 0 genes. The A, B, and H genes control the synthesis of enzymes that form the special monosaccharides or antigens of the erythrocyte membrane - A, B, and H. Antigen formation begins with the H gene, which, through the enzyme glycolysyltransferase controlled by it, forms the erythrocyte antigen H from a special precursor substance - ceramipentasaccharide. Then genes A and B through the activity of enzymes controlled by them form antigens A or B from H-antigen, which is the starting material for them. Gene “0” does not control the transferase, and H-antigen remains unchanged, forming blood group 0(I). Thus, A, B and H antigens are present on the membrane of human erythrocytes [4,5,6].

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In 20% of people, antigen A has antigenic differences (A1 and A2). Antibodies against antigens A, A1, A2 and B begin to be formed after birth by the immune system in response to stimulation by antigens from food and bacteria, for example, inhaled air. The maximum production of anti-A and anti-B antibodies falls on 8-10 years of age. At the same time, more anti-A than anti-B is accumulated in the blood plasma. The anti-A and anti-B antibodies are called isoantibodies or agglutinins, and the corresponding membrane antigens are called agglutinogens.

Antigens are high-molecular-weight polymers of natural or artificial origin that carry features of genetically foreign information [7,8,9].

Antibodies are immunoglobulins formed when an antigen is introduced into the body.

Isoantigens (intraspecies antigens) are antigens originating from the same species of organisms but genetically foreign to each individual. The most important are erythrocyte antigens, especially antigens of the AB0 and Rh-hr systems [10,11].

Immunologic conflict in the AB0 system occurs when antigens and antibodies of the same name meet, causing agglutination of red blood cells and their hemolysis. Immunologic conflict is observed:

1) when transfusion of blood group incompatible in group relation;

2) when transfusion in large quantities of blood group to people with other blood groups.

When transfusing blood, the forward and reverse Ottenberg's rule are taken into account. Ottenberg's direct rule: when transfusing small volumes of blood (1/10 of the circulating blood volume) pay attention to the donor's red blood cells and the recipient's plasma - a person with type I blood is a universal donor [12,13,14].

Inverse Ottenberg's rule: when transfusing large volumes of blood (more than 1/10 of the circulating blood volume) pay attention to the donor's plasma and the recipient's erythrocytes. A person with type IV blood is a universal recipient.

Currently, it is recommended to transfuse only single-group blood and only in small quantities.

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The Rh antigenic system was discovered in 1940 by K. Landsteiner and A. Wiener.They found in the blood serum of macaque monkeys, rhesus antibodies - anti-rhesusagglutinin [15,16,17].

Antigens of the Rh system are lipoproteins. Erythrocytes of 85% of people contain Rh agglutinogen, their blood is Rh-positive, 15% of people have no Rh antigen, their blood is Rh-negative. Six varieties of Rh antigens have been described. The most important are Rh0 (D), rh`(C), rh"(E). The presence of at least one of the three antigens indicates that the blood is Rh-positive.

The peculiarity of the Rh system is that it has no natural antibodies, they are immune and are formed after sensitization - contact of Rh blood with Rh+. In the primary transfusion of Rh- to Rh+ blood to a Rh- person, a Rh-conflict does not develop because there are no natural anti-rhesus agglutinins in the recipient's blood.

Immunologic conflict on the Rh antigenic system occurs with repeated transfusion of Rh(-) blood to an Rh+ person, in cases of pregnancy, when the woman is Rh(-) and the fetus is Rh+ [18,19,20].

In the first Rh(-) pregnancy of an Rh(-) mother with an Rh+ fetus, Rh-conflict does not develop because the antibody titer is low. Immune anti-rhesus agglutinins do not penetrate the placental barrier. They have a large protein molecule size (class M immunoglobulin). In repeat pregnancies, the antibody titer increases. Anti-rhesus agglutinins (class G immunoglobulins) have a small molecular weight and easily penetrate the placental barrier into the fetus, where they cause agglutination and hemolysis of red blood cells [21,22,23,24,25].

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