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THE ROLE OF THE ERYTHROCYTE ANTIGEN SYSTEM

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The role of erythrocyte antigens in the occurrence of hemolytic disease of the fetus and newborn is different and is determined by the ability of alloantibodies formed to a certain class of antigens to destroy erythrocytes or disrupt the processes of their formation in the fetal body [5,6,7].

Keywords: *immune antibodies, antigen, alloantibodies, blood group, erythrocytes;*

The process of formation of immune antibodies to fetal erythrocyte antigens depends on the presence of an antigen absent in the mother, its immunogenicity, and the number of fetal erythrocytes entering the bloodstream of the pregnant woman [1,2,3].

Antibodies to erythrocyte antigens (alloantibodies) come in natural (regular) and immune (irregular) forms. Thus, in the blood serum of people (except for individuals with blood group AB) there are constantly present congenital natural antibodies to the A and/or B antigens of the ABO system, which consists of four antigens [4].

The disease in the fetus and, subsequently, in the newborn is caused by incompatibility of maternal and fetal red blood cells according to the AVO antigen system in 10-20% of all cases. In this case, 40 times more often hemolytic disease of

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the fetus / newborn develops in women with blood group 0 (I) and a spouse with a different blood group. However, when this type of isoimmunization occurs, severe forms of the disease in the fetus and newborn are observed only in isolated cases - 1:3000 births [8,9,10].

The greatest clinical significance in the pathogenesis of severe forms of hemolytic disease of the fetus and newborn have antigens of the Rhesus system (Rhesus), which has 48 antigens. The development of Rhesus isoimmunization is possible in pregnancy of a Rhesus-negative (D-negative) mother with a Rhesus-positive fetus, i.e., the formation of antibodies in the mother's blood to the antigens of fetal erythrocytes of the Rhesus system. A similar obstetric situation occurs when a Rh-negative woman has a Rh-positive spouse [11,12,13].

The occurrence of Rh-negative blood in a population depends on its ethnicity. It is most often detected in Spanish Basques - in 30-32%, almost absent in Africans, and in European populations it is observed in 15-17% of the population [14,15,16].

According to various authors, D-negative blood is most common (12-18% of cases) in Caucasian women living in Europe and North America. In general, D antigen incompatibility with the fetus is observed in about 10-13% of all pregnant women, with isoimmunization developing in 5-8% of women. Severe course of the disease in the fetus and newborn is most often - in 95% of cases - due to the formation of antibodies in the mother's blood to the D antigen of fetal erythrocytes. This antigen has the greatest immunogenic properties among the antigens of the Rhesus system [17,18,19].

The risk of fetal and neonatal disease in a Rh-conflicted couple in which the mother is Rh-negative is different and depends primarily on the Rh0 (D) zygosity of the father. If the father is a homozygous Rh0 (D) carrier (genotype DD), then all children of a Rh-negative mother (genotype dd) will have Rh-positive blood (genotype Dd). If the father in the couple is heterozygous (genotype Dd), the risk of the offspring having Rh-positive blood is about 50%. About 45% of people with Rh0 (D) are homozygous carriers [20,21,23].

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Although about 60% of D-negative women carry a D-positive fetus, isoimmunization as a result of termination of pregnancy by a D-positive fetus is not as frequent as one might expect, predicting its frequency solely on the basis of D incompatibility between maternal and paternal blood. This is explained by several reasons. First of all, the volume of fetal cells entering the maternal bloodstream through the placenta may be insufficient to initiate an immune response. In addition, maternal and fetal ABO blood incompatibility may play a protective role. Another factor influencing the immune response is the Rh phenotype of the fetus. In addition, individuals can exhibit different sensitivities to small antigenic stimuli [22].

In some cases, more often with ABO incompatibility, sensitization occurs already in the first pregnancy. According to different authors, for the development of immunization in the first pregnancy is sufficient to penetrate into the maternal bloodstream from 80 to 150 ml of fetal blood. It is known that women with blood type 0 (I) and a partner with blood type A, B, or AB have a 50-75% lower risk of Rh immunization than pregnant women with a different blood type [24,25].

The severity of isoimmunization depends on the immunogenicity of the antigen causing it. In descending order of immunologic activity, the first two places among all erythrocyte antigens are occupied by A and B antigens (ABO system), which are present in fetal tissues from the 5th-6th week of pregnancy. Next is the D-antigen (Rhesus system), which is detected in fetal tissues from the 30-45th day of pregnancy. In descending order of immunogenic activity among the antigens of the Rhesus system, D-antigen is followed by c, E, C, and e.

Antigens from other erythrocyte systems such as Duffy, Kidd, Kell, etc. can also cause isoimmunization in pregnancy. However, these antigen systems cause clinically significant forms of disease in the fetus and newborn rarely. Table 2 shows the distribution of erythrocyte antigens into groups, depending on their immunologic ability to cause hemolytic disease in the fetus and newborn.

Severe forms of the disease in the fetus and newborn are caused by antibodies formed to those systems of erythrocyte antigens that are highly immunogenic. As

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shown in Table 2, antibodies of high immunogenicity include such Rhesus antigens as D, C, and E [1,2,3].

It was previously thought that anti-E antibodies rarely caused severe fetal hemolytic disease because they rarely caused severe anemia in the fetus. However, it turned out that they can cause severe hyperbilirubinemia in newborns, requiring invasive postnatal treatment. In recent years, including at the D. O. Ott Research Institute of Anemia and Gastrointestinal Surgery of the Russian Academy of Sciences. In recent years, including at the D.O. Ott Research Institute of Ophthalmology of the Northwestern Department of the Russian Academy of Medical Sciences, a number of studies have been carried out that proved the high immunogenicity of antigen E and its ability to cause severe anemia in fetuses.

The remaining antigens of the Rhesus system, the K-antigen of the Kell system and several antigens of other systems are considered by some authors to be so-called “minor” erythrocyte antigens. They are relatively rare, however, sometimes cause even more severe course of hemolytic disease of the fetus and newborn than that caused by D-antigen. Thus, it is known that anti-c antibodies cause hemolytic disease of the same severity as anti-D [4,5,6].

Antibodies to non-aggressive “small” erythrocyte antigens, Cweek antigen, rarely cause hemolytic disease requiring antenatal or postnatal treatment. There is an opinion that sensitization to “small” erythrocyte antigens is observed in pregnancy when women have a history of previous hemotransfusions. It is explained by incomplete immunologic compatibility of donor and recipient blood [7,8,9].

Rare antigens causing hemolytic disease of the fetus and newborn with a cumulative incidence of 0.24% include antigens of the Kell, Duffy, Kidd, P, MNS, Lutheran, and Xg systems. Thus, antibodies of the Lutheran system, which most often cause the development of the disease, are represented by aHTH-Lua and aHTH-Lub antibodies. However, these antibodies are mainly IgG4, which explains the mild form of the disease in newborns [10,11,12].

Antibodies to MNS antigens are mainly represented by anti-N, anti-U, anti-S, anti-s antibodies, which are also unable to cause disease in the fetus and newborn or

cause it in a mild form. At the same time, there are anti-M antibodies of this system, which are able to cause hemolytic disease with a very severe course in newborns. However, such pathogenesis of the disease is extremely rare and only a few such cases have been described in the literature.

Mild forms of the disease cause antibodies to antigens of the Lewis, Diego, Daffy and Kidd systems. This is due to their low concentration or complete absence of erythrocytes on the membrane in fetuses and newborns.

A special place among the erythrocyte antigens is occupied by the Kell antigens, which are found in 7–9% of immunized women. They can cause a severe course of hemolytic disease of the fetus, accompanied by the development of not hemolytic, but aplastic alloimmune anemia. Antibodies to all antigens of erythrocytes of the Kell system are clinically significant and cause severe forms of hemolytic disease of the fetus and newborn due to their high immunogenicity [14,15,18].

The K-antigen is of the greatest importance in clinical practice, since antibodies to this antigen in 95–98% belong to aggressive subclasses of immunoglobulins. The incidence of the disease due to the K-antigen of the Kell system is 1:10000-1:20000 births. Entering the fetal bloodstream, anti-K antibodies lead to the suppression of erythro- and thrombopoiesis in the fetus.

Thus, alloimmune anemia, including hemolytic disease of the fetus, can be caused by incompatibility of the blood of the mother and the fetus not only for one, but also for several antigens of both one and several systems of erythrocyte antigens at the same time [20,21,23].

REFERENCES

1. Abduhakimov B. A. et al. Bolalar va o'smirlarda birlamchi tuberkulyozning o’ziga xos kechish xususiyatlari va klinik-laboratoriya usullari //Ta'lim innovatsiyasi va integratsiyasi. – 2024. – Т. 32. – №. 3. – С. 139-143.

2. Бердиярова Ш. Ш. и др. Клинико-лабораторная диагностика фолиевой кислотодефицитной анемии //TADQIQOTLAR. UZ. – 2024. – Т. 49. – №. 3. – С. 46-53.

3. Umarova T. A., Kudratova Z. E., Axmadova P. Role of conditionally pathogenic microflora in human life activities //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – T. 2. – №. 11. – C. 29-32.

4. Muhamadiyeva L. A., Kudratova Z. E., Sirojeddinova S. Pastki nafas yo'llari patologiyasining rivojlanishida atipik mikrofloraning roli va zamonaviy diagnostikasi //Tadqiqotlar. Uz. – 2024. – T. 37. – №. 3. – C. 135-139.

5. Umarova T. A., Kudratova Z. E., Norboyeva F. Modern aspects of etiology and epidemiology of giardias //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – T. 2. – №. 11. – C. 25-28.

6. Isomadinova L. K., Daminov F. A. Glomerulonefrit kasalligida sitokinlar ahamiyati //Journal of new century innovations. – 2024. – T. 49. – №. 2. – C. 117-120.

7. Umarova T. A., Kudratova Z. E., Maxmudova H. Mechanisms of infection by echinococcosis //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – T. 2. – №. 11. – C. 18-21.

8. Даминов Ф. А., Исомадинова Л. К., Рашидов А. Этиопатогенгетические и клинико-лабораторные особенности сальмонелиоза //TADQIQOTLAR. UZ. – 2024. – T. 49. – №. 3. – C. 61-67.

9. Umarova T. A., Kudratova Z. E., Baxromova M. Autoimmune diseases: new solutions in modern laboratory diagnostics //International Conference on Modern Science and Scientific Studies. – 2024. – C. 78-81.

10. Бердиярова Ш. Ш. и др. Узловой зоб и его клинико-лабораторная диагностика //TADQIQOTLAR. UZ. – 2024. – T. 49. – №. 3. – C. 38-45.

11. Umarova T. A., Kudratova Z. E., Muhsinovna R. M. The main purpose of laboratory diagnosis in rheumatic diseases //International Conference on Modern Science and Scientific Studies. – 2024. – C. 82-85.

12. Umarova T. A., Kudratova Z. E., Ruxshona X. Contemporary concepts of chronic pancryatitis //International Conference on Modern Science and Scientific Studies. – 2024. – C. 11-15.

13. Хамидов З. З., Амонова Г. У., Исаев Х. Ж. Некоторые аспекты патоморфологии неспецифических язвенных колитов //Молодежь и медицинская наука в XXI веке. – 2019. – С. 76-76.

14. Umarova T. A., Kudratova Z. E., Muminova G. Instrumental diagnostic studies in chronic pancreatitis //International Conference on Modern Science and Scientific Studies. – 2024. – С. 16-20.

15. Umarova T. A., Kudratova Z. E., Norxujayeva A. Etiopathogenesis and modern laboratory diagnosis of prostatitis //International Conference on Modern Science and Scientific Studies. – 2024. – С. 6-10.

16. Амонова Г. У., Сулаймонова М., Кизи Ж. Пневмопатиянинг ателектатик шаклида чақалоқлар мия структураларидаги ўзгаришларнинг патоморфологияси //Новости образования: исследование в XXI веке. – 2024. – Т. 2. – №. 22. – С. 163-166.

17. Sabirovna I. N., Raykhona K. Clinical and laboratory changes in post-term infants //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2024. – Т. 2. – №. 5. – С. 96-99.

18. Ибрагимова Н. С., Юлаева И. А. Сложности диагностики и лечения внебольничной пневмонии у детей раннего возраста //TADQIQOTLAR. UZ. – 2024. – Т. 39. – №. 1. – С. 58-62.

19. Laboratory diagnosis of torch infection bs Shukurullaevna, TF Uktamovich TADQIQOTLAR. UZ 48 (1), 200-206

20. Амонова Г. У., Исмоилов Ж. М. Реорганизация цитоархитектоники эпителиального пласта бронхов у кроликов с хроническим экспериментальным ларингитом //Молодежь и медицинская наука в XXI веке. – 2017. – С. 51-51.

21. Clinical and laboratory characteristics of renal pathology of pregnancy in the first trimester bs Shukurullayevna, MN Komilzhonovna TADQIQOTLAR. UZ 39 (1), 74-79

22. Umarova T. A., Kudratova Z. E., Maxmudova D. Pathogenesis of bronchial asthma development at the present stage //International Conference on Modern Science and Scientific Studies. – 2024. – С. 21-24.

ISSN 3060-4567

23. Differential diagnosis of jaundice literature review BS Shukurullaevna Web of Medicine: Journal of Medicine, Practice and Nursing 2 (1), 41-49

24. Эшкабилов Тура Жураевич, Хамидова Фарида Муиновна, Абдуллаев Бахтиёр Саидович, Амонова Гулафзал Узбекбаевна, Исмоилов Жасур Мардонович Патоморфологические изменения легких при идиопатических фиброзирующих альвеолитах // Вопросы науки и образования. 2019. №28 (77).

25. Хамидов З. З., Амонова Г. У., Исаев Х. Ж. Некоторые аспекты патоморфологии неспецифических язвенных колитов //Молодежь и медицинская наука в XXI веке. – 2019. – С. 76-76.