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ПРОБЛЕМЫ С ПЕРИНАТАЛЬНЫМ СТАТУСОМ У БЕРЕМЕННЫХ ЖЕНЩИН И ПУТИ ИХ УСТРАНЕНИЯ

Резюме: Резус-изоиммунизация - наличие в крови матери IgG-антител (анти-Rh (D) антител) как проявление вторичного иммунного ответа у сенсибилизированных пациенток вследствие несовместимости крови матери и плода по антигенам системы Резус. Синонимы - резусконфликт, резус-сенсибилизация, резус-аллоиммунизация.

В данной статье представлены вопросы оценки перинатального состояния у беременных, получавших антирезусную профилактику, и лечения патологий их видов.

Ключевые слова: перинатальный статус, антирезусная профилактика, беремкенная женщина.

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PROBLEMS WITH PERINATAL STATUS IN PREGNANT WOMEN AND WAYS TO ELIMINATE THEM

Resume: Rh-isoimmunization - the presence of IgG antibodies (anti-Rh (D) antibodies) in the mother's blood as a manifestation of a secondary immune response in sensitized patients due to incompatibility of maternal and fetal blood by Rh system antigens. Synonyms - Rh conflict, Rh sensitization, Rh alloimmunization.

This article presents the issues of assessing the perinatal condition in pregnant women who received antiresus prophylaxis, and the treatment of pathologies of their types.

Keywords: perinatal status, antiresult prevention, pregnant woman.

Introduction. Hemolytic disease of the newborn (GBN) (a consequence of GBP) in the world is diagnosed in approximately 1.6%-2.5% of newborns, while the frequency of Rh-isoimmunization in recent years has not yet had a significant downward trend [2]. Perinatal mortality in GBN is 0.037%.

The incidence of bilirubin encephalopathy in various countries of the world ranges from 0.4 to 2.7 per 100,000 newborns [4].

Rh-isoimmunization mainly develops when the blood of the mother and fetus is incompatible with the RhD antigen. GBP can also develop when the blood of the mother and fetus is incompatible with antigens C, c, E, e. A woman with Rh-negative blood affiliation is sensitized either during pregnancy when Rh(D) antigen of the fetus inherited from the biological father enters the bloodstream, or outside pregnancy with transfusion of Rh(D) components-positive donor blood.

During pregnancy, fetal erythrocytes penetrate through the placental barrier into the mother's bloodstream during the 1st trimester in 5-7%, in the 2nd trimester in 15-16% and in the 3rd trimester in 29-30% of women [1]. The first stage of the mother's immune response is the production of IgM antibodies that have a high molecular weight and do not pass through the placental barrier into the fetal bloodstream.

The next stages in the development of isoimmunization are the formation of IgG antibodies with low molecular weight and freely penetrating into the fetal bloodstream from the mother through the placental barrier, including the G1 and G3 immunoglobulin subclasses, which actively interact with Fc receptors (FcR) of lymphocytes and macrophages, which play an important role in the hemolysis of fetal erythrocytes. In the 1st pregnancy, GBP is rare, since the ingestion of fetal erythrocytes into the mother's bloodstream occurs mainly in late pregnancy or during childbirth, and the primary immune response does not have time to

form. GBP in the 1st pregnancy may be a consequence of isoimmunization that has already taken place, for example, when a Rh-negative woman is injected with components of Rh-positive blood in the anamnesis[6].

In subsequent pregnancies, the ingestion of fetal erythrocytes into the mother's bloodstream causes a rapid immune response, IgG antibodies penetrate to the fetus, hemolysis, anemia, activation of foci of extramedullary hematopoiesis and hepatosplenomegaly develop. Due to the "overload" of liver cells with iron and globin breakdown products, its protein synthetic function is disrupted, which leads to hypoproteinemia, hypoalbuminemia, and subsequently to increased permeability of vascular walls[3].

Against the background of progressive anemia, hypoxemia develops, causing a hyperdynamic type of blood circulation in the fetus, with the gradual formation of heart failure and portal hypertension, contributing to a further increase in the size of the liver and the appearance of anasarca[5].

This is how severe anemia develops with fetal dropsy. In the absence of intrauterine treatment, antenatal fetal death may occur. Mild anemia is caused by the later onset of hemolysis of fetal erythrocytes shortly before delivery or immediately after the birth of a child [6].

The purpose of the study. Optimization of the observation of pregnant women with Rh-negative blood, based on the determination of the Rh factor of the fetus from the early stages of pregnancy using domestic test systems "DNA-rhesus of the child".

Materials and methods of research. A comprehensive medical and social study of reproductive health, medical activity of young first-borns (13-17 years, 11 months, 29 days) and first-borns of late reproductive age in Andijan and Andijan region, the health status of their first-borns, assessment of the organization of medical care for these categories of women was conducted. Regarding the late reproductive age, from which a pregnant woman should belong to a high-risk group, there is no unambiguous opinion: 30, 35, 40 years,

because a group of first-time mothers 30 years and older was formed for the study.

The results of the study. The causes of the development of hemolytic disease of the fetus and newborn are rhesus sensitization in women, developing in 2.9% after a history of transfusion of Rh-incompatible blood and in 97.1% as a result of the lack of prevention with antiresus immunoglobulin. Risk factors predisposing to the activation of antibody production and aggravating the course of Rh sensitization in 45.9% of pregnant women are exacerbation of herpes and cytomegalovirus infection, in 28.7% the development of placental insufficiency.

The determination of subclasses and ^OZ, in order to assess the risk of erythrocyte hemolysis, allows predicting the probability of development and severity of fetal hemolytic disease. A high degree of risk of erythrocyte hemolysis was determined in 62% of pregnant women, an average degree in 19, g5% of women and a low degree in 1'8.4%.

The increase in the maximum blood flow rate of the medial cerebral artery during dopplerometry is a predictor of the severity of fetal anemia and a prognostic factor regarding the severity of fetal hemolytic disease, allowing- to determine the further tactics of pregnancy and the timing of delivery.

In women with high Rh-antibody titers and a burdened obstetric history, therapeutic plasmapheresis and immunoglobulin therapy as a pre-gravidar preparation can prevent the development of severe fetal hemolytic disease and improve perinatal outcomes in 80% of patients.

Rh sensitization in 18.4% of pregnant women is accompanied by changes in the hemostasis system, expressed in the development of hypercoagulation occurring against the background of activation of intravascular coagulation. Therapeutic plasmapheresis followed by immunoglobulin therapy helps to reduce the processes of intravascular coagulation, stabilization of coagulation and 124 fibrinolytic potential of the blood by reducing the level of fibrinogen by

23.5%, the content of high-molecular soluble fibrin-monomer complexes (PCMF) and ETC by 30%.

In pregnant women with Rh sensitization during therapeutic plasmapheresis, there are no significant changes in the main parameters of the biochemical status (total protein, glucose, direct and total bilirubin, creatinine, urea) and hemogram parameters, which indicates the safety of this treatment method.

Therapeutic plasmapheresis is a pathogenetically justified method of treating Rh sensitization, leading to a significant decrease in the titer of Rh antibodies ($p< 0.01$), and subsequent immunoglobulin therapy reduces the likelihood of rebound effect, thereby preventing the development of fetal hemolytic disease and allows prolonging pregnancy by an average of 4.5 ± 0.1 weeks, reduces the frequency of premature births from 69 to 44%.

The developed algorithm for the management of women with Rh-sensitization with the inclusion of therapeutic plasmapheresis in complex therapy followed by immunoglobulin therapy allows prolonging pregnancy to the optimal term of delivery in 59% of pregnant women, reducing the frequency of birth of children with severe hemolytic disease by 2.4 times, reducing the frequency of replacement transfusion of washed erythrocytes by 2 times and the number of phototherapy sessions in newborns by 1.5 times.

Conclusion. Optimization of monitoring of pregnant women with Rh-negative blood will allow to clearly define the medical tactics of pregnancy management in each case, which solves the psychological and economic problems of managing this category of women.

New domestic diagnostic kits for the identification of the fetal Rh factor gene in the mother's blood can be used in obstetric practice.

The implementation of the developed algorithm for the management of Rh-negative pregnant women in the activities of obstetricians and gynecologists

will optimize obstetric and gynecological care for these women and improve pregnancy outcomes for mother and fetus.

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