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Hemolytic Disease of the Newborn

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Abstract: This article discusses the relevance, etiology, pathogenesis, clinic, differential diagnosis and treatment of hemolytic disease of the newborn.

Keywords: hemolytic disease of the newborn, hemolysis, neonatal care, treatment.

Relevance

Currently, among the problems of state medical and social importance, the problem of protecting motherhood and childhood, in particular, improving medical care at the stage of a specialized maternity hospital, occupies a particularly important place. The versatility of this problem lies in the fact that it includes a set of tasks that determine the quality of public health. These include indicators of perinatal and early neonatal mortality, the health of children at different stages of their lives, disability since childhood, congenital and hereditary diseases, congenital infections, etc. In this regard, the focus in both prevention and diagnosis is shifting from early childhood in the antenatal and perinatal periods [1]. Based on the foregoing, the Ministry of Health and Social Protection of the Population of the Republic of Uzbekistan has taken a number of measures aimed at reducing the rates of perinatal morbidity, disability and mortality.

The significance of hemolytic disease of the newborn (THN) is determined by a complication of increased hemolysis - hyperbilirubinemia, a certain effect of unconjugated bilirubin (NB) on the brain and the possibility of damage (staining) of its nuclei, which was first identified by J. Orth in 1887, on the basis of which in 1904 G Schmorl proposed the term "nuclear jaundice". At the same time, it must be emphasized that if, with timely qualified assistance, the initial stages of bilirubin brain damage are reversible, then severe manifestations of HDN (edematous form, “nuclear jaundice”), despite the therapy provided, usually end in death or profound disability. This is especially true for premature infants prone to bilirubin intoxication, due to greater sensitivity to the toxic effects of NP, due to the lack of ligandin (a specific protein that binds NP in the cytoplasm) and low activity of the bilirubin oxygenase system.

Hemolytic disease of the newborn is a pathological hyperbilirubinemia caused by increased hemolysis of erythrocytes, the cause of which is the incompatibility of the blood of the mother and child for erythrocyte antigens.

According to the international classification of diseases of the X revision, there are: hemolytic disease of the fetus and newborn (P55); Rh isoimmunization of the fetus and newborn (P55.0); ABO isoimmunization of the fetus and newborn (P55.1); other hemolytic disease of the fetus and newborn (P55.8); haemolytic disease of fetus and newborn, unspecified (P55.9).

The classification of hemolytic disease of the newborn provides for the establishment of: a) the type of conflict (Rh-factor, ABO-system, other factors); b) clinical form (edematous, icteric, anemic, antenatal death with signs of maceration); c) severity (mild, moderate, severe); d) complications (bilirubin intoxication, anemia).

When formulating the diagnosis, special attention is paid to the risk factors for the development of hyperbilirubinemia, which are divided into: a) maternal; b) perinatal; c) neonatal.

Etiology. There are 14 main erythrocyte group systems that combine more than 100 antigens, as well as numerous private and common erythrocyte antigens with other tissues. As a rule, the child's red blood cells have some paternal antigens that are absent from the mother.

Pathogenesis. Fetal erythrocytes are regularly found in the mother's bloodstream from 16 to 18 weeks of gestation. Immediately before childbirth, fetal erythrocytes can be found in the blood of 75% of pregnant women, as a rule, their number is small - 0.1-0.2 ml. The most pronounced transplacental transfusion occurs during childbirth, but again it is usually small - 3-4 ml of fetal blood. The erythrocytes of the fetus (even in the amount of 0.1 ml) that have entered the mother's bloodstream, having a D-antigen that is absent in the mother, lead during the first pregnancy to the synthesis of Rh-antibodies at the beginning, related to class M immunoglobulins, which do not penetrate the placenta, but then IgG class antibodies, which can already cross the placenta. During pregnancy and due to the low number of erythrocytes in the fetus, as well as due to active immunosuppressive mechanisms, the mother's primary immune response is reduced, but after the birth of the child and due to the large number of erythrocytes of the child in the mother's bloodstream that penetrated there during childbirth, and due to the removal of immunosuppression, an active synthesis of Rh antibodies occurs. That is why the introduction of exogenous Rh antibodies (anti-O-immunoglobulin) within 24-72 hours after childbirth or abortion (D-antigens appear in the embryo at the beginning of the second month of gestational age) is an effective method to reduce Rh sensitization, and RH-THN frequencies.

In the first week of life (beginning of day 3), approximately 60% of full-term and 80% of premature infants develop physiological jaundice, with special attention to be paid to jaundice that appears on the first day of life, which may indicate its pathological nature. This will help the history data, which are one of the diagnostic criteria:

- ✓ positive indirect Coombs test in a pregnant woman;
- ✓ jaundice in a previous child due to group (ABO) or Rh incompatibility;
- ✓ early jaundice in previous children, anemia;
- ✓ increase in antibody titer in Rh (-) women during pregnancy;
- ✓ Ultrasound signs of fetal dropsy.

Among the clinical signs, the leading one is the appearance in the first 24 hours of a child's life of jaundice (from the proximal part of the body), which gradually increases and descends distally. This is due to the specifics of the deposition of bilirubin in the tissues. In this case, it is necessary to assess the rate and intensity of the increase in jaundice. Based on the external examination of skin color, the localization of jaundice can be clinically determined using the Cramer Scale. This is necessary to define the criterion for "dangerous jaundice". To do this, in a well-lit room in a completely undressed child, lightly press a finger to the level of subcutaneous fat until the blanching of the skin indicates its main color. It should be noted that in premature and hypotrophic children, the degree of visualization of hyperbilirubinemia is less pronounced. Among other clinical signs, pallor of the skin, hepatosplenomegaly, and edematous syndrome are distinguished.

Mild HDN is diagnosed in the presence of moderate clinical and laboratory or only laboratory data. In this case, in the absence of any complications, severe background conditions and concomitant diseases, only phototherapy is required. The level of hemoglobin in cord blood in the first hours of life is more than 140 g/l, NB in cord blood is less than 68 µmol/l.

Moderate-severe HDN is observed with hyperbilirubinemia, requiring exchange transfusion. At the same time, there is no clinic of bilirubin intoxication of the brain or other complications. These children usually require early exchange transfusion combined with intensive phototherapy.

The severe course of HDN, as a rule, involves the presence of an edematous form of the disease, severe anemia (hemoglobin less than 100 g / l), jaundice (bilirubin in umbilical blood more than 85

μmol / l), the presence of symptoms of bilirubin brain damage of any severity and at all stages of the disease, respiratory and cardiac disorders in the absence of data indicating concomitant pneumo- or cardiopathy.

Diagnosis of HDN. When conducting laboratory studies in favor of HDN, they are evidenced by: Rh (-) of the mother and Rh (+) of her child, or O (I) mother's blood type and A (II) or B (III) groups in the child, or Rh (-) O (I) mother's blood group Rh (+) A (II) or B (III) groups in the child; the presence of antibodies in the blood of the mother; decrease in Hb in cord blood less than 160 g/l; increase in cord blood bilirubin above 51 μmol/l; reticulocytosis (6-40%); positive direct Coombs test from umbilical cord blood; polychromia, anisocytosis with Rh incompatibility, microspherocytes with ABO incompatibility.

Differential diagnosis is carried out with physiological jaundice, which, unlike hemolytic jaundice, appears on the 2nd-3rd day of a child's life, gradually increases and tends to fade from the end of the 1st week of life, the content of bilirubin in the cord blood is less than 51 μmol / l, and its increase in 1 day does not exceed 5.1 μmol/l. Hemoglobin (Hb) blood within normal limits.

Jaundice in premature newborns is characterized by an earlier onset (1-2 days of life), which makes it difficult to differentiate it from hemolytic disease of the newborn. However, history data (blood type of mother and child, absence of sensitization) and laboratory tests (normal hemoglobin, erythrocytes, absence of reticulocytosis) help to make a correct diagnosis.

Phototherapy. The technique of traditional phototherapy (exposure to light with a wavelength of 425-475 nm) was developed in 1958 and is based on the effect of 3 chemical reactions occurring in the body (photooxidation, configurational isomerization, structural isomerization), in which bilirubin passes into the skin and, being converted into isomer - lumirubine, excreted in bile and urine.

Traditionally, an intermittent technique is used, in which breaks are made for feeding, procedures, etc. They also use continuous (with severe hyperbilirubinemia) and the most effective - "double" phototherapy (increased intensity, in which the child is placed in a special chamber illuminated from all sides or from both sides, using a conventional lamp and a luminous mattress).

The procedure is terminated if: if the concentration of bilirubin in the blood is below the level from which phototherapy was started or the concentration of total serum bilirubin has decreased by at least 50 μmol / l below the threshold values of serum bilirubin at which phototherapy is prescribed; if the newborn has light stools or dark urine; after 3 days (if it is not possible to check the level of serum bilirubin).

At the same time, 12-18 hours after the cessation of phototherapy, it is advisable to repeat the measurement of serum bilirubin to check the possibility of its re-growth.

Technique of the ZPK operation. To perform an exchange transfusion, sterile gloves, a scalpel or scissors, umbilical ligature, 3.5F, 5F umbilical catheters, a three-way stopcock, 2, 5, 10 ml syringes, masks, an infusion system, resuscitation equipment (if necessary).

Preparing a child for an IPC. Fixing the child with a special swaddling, put the child under an open source of heat or in a pitcher. Insert an 8F gastric tube and leave its end open.

After processing the surgical field with alcohol, we limit it with sterile diapers, fixing it with clamps. Then we cut off the rest of the umbilical cord and with the help of a probe we find the umbilical vein for its catheterization.

Immediately before the operation, the transfusion medium should be warmed up to 36.7-37°C. volume of circulating blood (in newborns weighing less than 1000 to 1500 g - 5 ml, from 1500 to 2500 g - 10 ml, more than 2500 g - 20 ml). The ratio of injected erythrocyte blood to fresh frozen plasma should be 2:1. Every 100 ml of blood, 0.5-1.0 ml of a 10% solution of calcium gluconate in 3-5 ml of 10% glucose should be injected. At the final stage of the replacement transfusion, an erythrocyte mass is injected at the rate of 10 ml / kg, blood is taken to determine bilirubin, after which the catheter is removed or left in place (if necessary).

During the PKK operation, constant monitoring of vital functions (heart rate, respiratory rate, body temperature, diuresis, blood glucose, if possible SaO2, CBS) is necessary. The duration of the operation depends on the body weight of the child and is 2.5-3.0 hours. The protocol of the replacement blood transfusion operation is filled out in accordance with the established rules. N.B. The criterion for the effectiveness of treatment is a decrease in bilirubin by more than 2 times.

An indication for repeated exchange transfusion is an increase in total bilirubin of more than 8.6 µmol/l per hour.

Care and monitoring of the newborn after PKK surgery includes: continuation of phototherapy; monitoring the location of the catheter for possible bleeding and inflammation; thermometry (every hour three times for 2 hours); control of blood pressure, heart rate, respiratory rate (every 15 minutes for 2 hours); control of diuresis (time of the first urination, color, volume) and stool (for the presence of blood); control of the level of bilirubin (after 6 hours, then according to indications); when feeding is resumed, watch for signs of food intolerance (gastric aspirate, vomiting, bloating).

Infusion therapy is carried out only when the child is in a serious condition and it is impossible to satisfy his physiological need for fluid (ineffective feeding, regurgitation, pathological maximum weight loss). N.B. It is necessary to inform the parents about the progress of the FPC operation and about the condition of the child after the operation.

Conclusion. The decision to discharge a child is made individually in each case, according to the results of the examination and the presence or absence of risk factors. In this case, the following conditions must be met: a satisfactory clinical condition of the child; established breastfeeding; localization of jaundice within 1-3 zones on the Cramer scale; the mother/family is informed and trained on how to look after the baby with jaundice, feed on demand (day and night), keep the baby warm, when to call for help immediately (if the baby is having trouble feeding or the baby seems ill).

Literature

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