RH Sensitization

Abstract

The Perinatal Hemolytic Disease is a disease caused by the immune passage through the placenta and maternal antibody specific classIg G for the antigen (the paternal) present in fetal red blood cells, shortening its lifetime. Haemolytic disease by the system Rh (Cc, Ee,Dd) is the prototype of maternal all immunization and fetal hemolytic disease. In around 98% of cases of maternal all immunization to erythrocyte antigens not (ABO hemolytic disease _ DHPN) are due to Rh (D), the remaining 2% atypical antigens as Kell factors, E or C (Bowman,1997).The D antigen is expressed only in red blood cells and is part of RH system with other antigens such as Cc, Ee, these alsohemolytic properties, but D is 50 times more immunogenic than the other antigens of the Rh system. The process of raising the maternal system Rh (D) is given by presence of Rh positive red blood cells in your

bloodstream, either during pregnancy via maternal-fetal transfusion (which can reach 29% inlast quarter), transfusion of incompatible blood, abortion, and today, deserves the use of injected illicit drugs.

Introduction

The process of hemolysis can start as early as 16 weeks gestation (Fairs, Agbetile, Hargadon, Bourne, Monteiro, Brightling, & Pashley,2010). The D maternal antibodies react with the antigens of fetal red blood cells, which are specific, covering them, but why not turn complement hemolysis not occurs in the intravascular system and yes, through the recognition of sensitized erythrocytes by the reticulo endothelial system of the fetus, mainly spleen, and then destroyed. Installed the destruction of RBCs of the fetus it produces anemia if untreated intensifies and forces the body to try to compensate for it, releasing into circulation young RBCs that are erythroblasts and also depending on the seriousness. It is important to frame the formation of foci of extramedullary hematopoiesis (Mollisonet al, 1997).

Anemia can be intensified in such a way, leading to a fetus extremely anemic conditions that justify this hepatos plenomegaly, generalized edema, effusion of serous cavities, congestive heart failure characterizing fetal hydrops. The untreated jaundice develops for the deposition of bilirubin in the system. Central Nervous leading to acute bilirubin encephalopathy or Kernicterus late.

After discharge, these infants have a picture of anemiahiporregenerativa (low reticulocyte count by low production blood by the bone) due to own illness and the pathophysiological also for these patients receive transfusions in the neonatal period. Maternal antibodies have a half-life of 28 days, and can remain the circulation of the newborn

for a long period, an average of 2-3 months life. During this period the rigorous monitoring is to assess the need for blood transfusion before clinical decompensation this patient.

Discussion

Haemolytic disease of the newborn secondary to Rh alloimmunization has a major cause of perinatal morbidity and mortality. The use suitable anti-human immunoglobulin A process prevents Specific awareness, and after their introduction in the 60s and 70s came to change this panorama. When administered properly, can make the risk of awareness, almost nil.

This milestone decreased in developed countries the number of deaths from haemolytic disease Rh from 18.4 to 1.3 per 100,000 live births. Unfortunately, the introduction of prophylaxis in our country was not universal despite. Since 1972, already proving its effectiveness60 women in the Santa Casa de Misericordia do Rio de Janeiro. In a review article that cites work done in some of the countries indicated that the main failures in the prophylaxis. The use of Human Immunoglobulin was introduced in our department from and 2000, as evidenced by some authors (OVALI, 1996;Gottstein, 2003), we found that there was an increase in the number of blood transfusions in the period after discharge for newborns who received nonspecific human immunoglobulin instead of performing exchange transfusion.

This discussion is important, since there is an major difference between our newborn population and where thiswas added guidelines. Unlike the United States our index all immunized newborns must be greater than 1% in the prophylaxis a sour country is not universal. In addition to the greater part of hemolytic disease in U.S. population in perinatal is a framework whose ABO Incompatibility clinician is much milder anemia and the risk of late

is very small and different from our population of newborns with hemolytic diseaseperinatal Rh factor. A meta-analysis of the Cochrane Library describes the delayed incidence of anemia is a major outcome and can not be forgotten, especially in countries where there is a difficulty safe blood supply and can not recommend it for this reason as routine use (Wilson, Whitehead, Nakano, Free, Kolls, & Cook, 2009).

The connection between fetal erythroblastosis and Rh incompatibility begins withpublished in 1939 that described LEVINE & STETSON reaction after blood transfusion in a woman after giving birth to a newborn dropsy. She had hemorrhage, and three times was transfused with blood from his spouse. Levine showed that the patient had an antibody that agglutinated cells from her husband and raised the possibility that she had been touchedagainst a fetal antigen inherited from the father.

Conclusion

In 1940, Landsteiner and WIENER determined antigen responsible and performed experiments, which showed that the serumcoming from rabbits immunized with pre monkey cells rhesus contained an antibody that agglutinated 85% of erythrocytes beings human Caucasians. These guys were called rhesus (Rhpositive). The remaining 15% of cells had not coalesced withthis serum and were called Rhesus negative (Rh). Levine et alin 1941, using serum anti-Rh Landsteiner and WIENER, determined the patient was reported in 1939 that they had Rh negative and antibody anti-Rh which agglutinated the erythrocytes of her husband and son, demonstrating the etiology of the disease.

References

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