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Hemolytic disease of newborn with pure red cell aplasia due to anti-M

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Introduction: Foetal erythroblastosis as reported in the literature is caused by Rhesus alloimmunization. However many other blood group incompatibilities may cause hemolysis in newborn but are without any treatment consensus. Antibodies with anti-M specificity, usually IgM, have been reported to be detected in 10% of pregnant women with a positive antibody screen. However, 0.01% to 0.7% of pregnant women would trigger anti-M IgG that can cross the placenta, resulting in variable degrees of hemolysis in fetuses.

Case: Blood bank received a requisition for reconstituted whole blood for an exchange transfusion for newborn twins having billirubin in the exchange zone, with falling hematocrit and reticulocyte count of 3%. Mother was gravid 2 with one living daughter 3 years and no history of transfusion. The twins were normal vaginal deliveries at 38 weeks of gestation. Blood group of mother as well as twins was found to be O Rh(D) positive. Direct Antiglobulin test (DAT) of the twins was negative, Indirect Antiglobulin test (IAT) of mother was positive at room temperature (RT), 37°C and AHG phase, indicating presence of an alloantibody of both IgM and IgG type (by DTT treatment). Further identification panel showed antibody against M antigen (IgM and IgG type). Similar antibody was identified in the serum of both twins but reacting at AHG phase only (IgG type). The Antibody Titre was 16 for IgG and 8 for IgM type in mother at the time of birth whereas IgG titre of 16 in twin 1and 8 in twin 2. Mother and father were homozygous for N & M antigen respectively and phenotype of twins as well as elder daughter was M+N+. The twins initially presented with the feature of hemolysis till 3 weeks of life and subsequently features of hemolysis subsided but continuous fall of HCT without reticulocytosis was observed till 45 days of life. Twin 1 required transfusion twice whereas twin 2 required it only once, with M-N+ packed red cells.

Discussion: We present a case of anti-M immunization due to previous pregnancy and severe neonatal anemia. Only a few cases have been reported in the literature where anti-M has caused fetal and/or perinatal anemia. The other diagnoses such as G6PD deficiency, Diamond Blackfan anemia and Parvovirus infection were also ruled out. Such prolonged anemia with normal reticulocyte count and normal platelet and leukocyte count suggests toward destruction of erythroid precursor cells by the anti M, which being previously also reported. Detection of such cases is crucial for the point of view of management of these neonates. There are known reported cases with high titre of anti M who have been successfully treated with intrauterine transfusions and also by therapeutic plasmapheresis.

Conclusion: This case was a rare presentation of maternal alloimmunisation by M antigen causing haemolytic disease. The biologic mechanisms explaining why a normally clinically insignificant antibody, in a small proportion of cases, would be aggressive and cause severe fetal anemia still remains to be elucidated. Thus, attention to and further studies of anti-M immunization is desired.

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