A Case of RhD Antigen Blocking in a Newborn with Severe HDFN

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Background

Antigen blocking is a rare phenomenon caused by maternal IgG antibodies (ab) saturating neonatal red blood cell (RBC) antigens. This may lead to false negative (-) typing results using monoclonal IgM reagents. We report a case of Rhesus (Rh) D antigen blocking in a neonate with severe HDFN caused by high titre maternal (G III, inadequate RhD prophylaxis at G II) anti-D (4096), beside an anti-Jk^a (64) and anti-C (2).

After early delivery (week 36 + 0) the newborn needed top-up transfusions (ccddee, Jk^a-). Initially, the neonate was typed Ccddee, Jk^a+ and the direct antiglobulin test (DAT) was strongly positive (IgG 4+, C3d 3+). The eluate was specific for anti-D, -Jk^a and -C.

Methods

To elucidate the discrepancy between the declared Rh phenotype (Ccddee) and eluate specificity (anti-D), a sample (neonate EDTA heel blood) was sent to our reference laboratory. Pheno- and genotype analysis was performed using standard techniques including two different saline reactive anti-D antisera (Grifols, CH) and commercially available PCR-SSP kits (inno-train, DE). The neonatal Rh phenotype was reevaluated serologically after dissociating the maternal IgG ab from the RBC by EGA treatment (EDTA Glycine-Acid Kit, Immucor, DE). DAT was performed using a polyspecific anti-human globulin card (BioRad, CH) before and after EGA treatment.

Results

Untreated RBC were typed as RhD-, an observed mix field reaction with anti-C was consistent to the top-up transfusion directly after birth. Three consecutive EGA treatments revealed a second mixed field reaction with anti-D, predicting CcD.ee beside a ccddee RBC population. The initially strongly positive DAT (3+) decreased markedly after EGA treatment (1+), as expected. A subsequent genotyping confirmed the serological typing as CcDdee, Jk^a+.

Due to prolonged anemia the newborn received a total of three top-up transfusions, directly after birth (Hb 64 g/L), on day 9 (65 g/L) and 28 (84 g/L). Additionally, intensive photo- and O_2-therapy were given to treat hyperbilirubinemia and low saturation levels.

Conclusions

Here, we present a case of RhD antigen blocking by maternal Anti-D in a neonate initially mistyped as RhD-. After dissociation of maternal high titre IgG by EGA the RhD+ phenotype of neonatal RBC became detectable and was confirmed by genotyping. Antigen blocking should be considered in cases with severe fetal anemia in the presence of high titre maternal ab and strongly positive DAT of neonate RBC not reacting with the corresponding IgM antiserum.