Introduction

In Switzerland extended antigen-matching for Duffy, Kidd and MNS - besides Rhesus and Kell - is recommended for sickle cell disease (SCD) patients. The ethnic diversity of red blood cell (RBC) antigen polymorphism engenders that these patients are often transfused with RBCs from donors of African origin. This strategy, however, increases the likelihood of being exposed to certain low-prevalence antigens, such as Dm (Rh23), as these are almost exclusive to African populations [1].

Dm is encoded by several types of RHD*DV alleles as well as by DAU-5 (RHD*10.05) [2]. Anti-Dm is associated with delayed hemolytic transfusion reactions (HTR) and may cause moderate hemolytic disease of the fetus and newborn (HDFN).

Previously, we reported a case of a pregnant woman with SCD and rare anti-Fy5 among other more common alloantibodies, the latter presumably a consequence of earlier pregnancies (DGTI, 2017). Only four registered Swiss blood donors were compatible to supply her with a total of eight RBC products during pregnancy.

Methods

Standard serological methods were used for antibody specification (BioRad, Cressier, CH and in-house). Crossmatches were carried out by indirect antiglobulin test (IAT) at 37°C. Molecular typing of donors’ and parental blood group antigens was performed by PCR-SSP (inno-train GmbH, Kronberg i. T. D and in-house).

Results

The patient’s predicted phenotype was O RhD, K-k+, Fy(a-b+), Jk(a-b+) and Dm+. Pretransfusion testing showed strong positive crossmatches with two of the compatible donors. Further serological analysis (INTS, Paris) revealed an anti-Dm in addition to anti-Fy5, anti-E and anti-Jkα.

Genotyping of the two donors causing positive crossmatches presented heterozygosity for RHD*10.05 which encodes Dm+.

The newborn’s phenotype was A RhD, K-K+, Fy(a-b+) and most likely Dm+ and Jk(a-b+), considering both maternal and paternal (A RhD, K-k+, Fy(a-b+), Jk(a-b+), Dm+) predicted phenotypes. The neonatal serum contained maternal anti-A1, anti-Da and anti-E.

The direct antiglobulin test was positive but elution only showed nonspecific reactions with papain-treated cells. Latter might have been caused by anti-Fy5 possibly in combination with anti-Jkα.

The newborn showed no clinical signs of HDFN.

Conclusion

Recently, we reported a pregnant SCD patient with a specific anti-public-antibody (anti-Fy5) amongst other alloantibodies. During her present pregnancy we were able to demonstrate that two positive crossmatches of two former compatible donors were caused by a new alloantibody against a low-prevalence antigen, namely anti-Dm, derived from several Df+ RBC transfusions during the previous pregnancy.

Despite this challenging blood supply we were able to support the patient with a total of ten antigen compatible and crossmatch negative RBC units from French and Swiss donors until delivery. This case illustrates the growing importance of national and international collaboration for provision of rare blood products.

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References


Young-Lan Song1, Stefan Meyer2, Gabriella Rizzi3, Inga Hegemann2, Christoph Gassner2, Beat M. Frey1, Charlotte Engström1

1Immunochemistry, Blood Transfusion Service Zurich, Swiss Red Cross, Switzerland
2Molecular Diagnostics and Flow Cytometry, Blood Transfusion Service Zurich, Swiss Red Cross, Switzerland
3Department of Medical Oncology and Hematology, Zurich University Hospital, Switzerland