

## View Abstract

---

**CONTROL ID:** 3165191

**TITLE:** ALLOIMMUNIZATION FOLLOWING RED BLOOD CELL EXCHANGE IN A PATIENT WITH RH(D) VARIANT

**AUTHORS (FIRST NAME INITIAL LAST NAME):** D. Allison<sup>1</sup>, D. Loeffler<sup>1</sup>, S. Campbell-Lee<sup>1</sup>, L. Sereseanu<sup>3</sup>, S. Saraf<sup>2</sup>, J. Crane<sup>3</sup>, V. Vidanovic<sup>1</sup>, C. Sheppard<sup>4</sup>

**INSTITUTIONS (ALL):** 1. Pathology, University of Illinois at Chicago, Chicago, IL, United States.

2. Hematology/Oncology, University of Illinois at Chicago , Chicago, IL, United States.

3. Vitalant-Illinois, Chicago, IL, United States.

4. Virginia Blood Services, Richmond, VA, United States.

**PRESENTATION TYPE:** Oral

**CURRENT CATEGORY:** Red Blood Cell Exchange

**ABSTRACT BODY:**

**Purpose:** The Rh system is the most important blood group after ABO. More than 60 Rh antigens are known, and Rh(D) is the most clinically significant. Alloantibodies against Rh antigens are the most commonly encountered clinically significant red blood cell alloantibodies. Rh variants may be mistyped by hemagglutination methods even with the more selective antisera in use today. Partial D antigens can be indistinguishable from the unaltered antigen by hemagglutination, however, exposure can still cause Rh alloimmunization. We present a D variant stem cell transplant recipient who developed anti-D following red blood cell exchange (RBCX) days before receiving hematopoietic stem cell transplantation (HSCT).

**Methods :** A 27-year-old Nigerian male with sickle cell disease (Hb SS) complicated by acute chest syndrome and frequent vaso-occlusive crises was referred for allogeneic matched sibling HSCT. As part of his pre-transplant evaluation, he was typed as B Rh(D) positive, with a detectable cold autoantibody, and negative direct antiglobulin test (DAT) in July 2017. He was serologically negative for C, E, K1, Jk<sup>b</sup>, S, M, Fy<sup>a</sup>, and Fy<sup>b</sup>

(GATA mutation). His brother, HLA-identical, blood type A Rh(D) positive, was the potential stem cell donor.

The recipient underwent RBCX ten days before HSCT (SCT D-10). The nine donor RBCs were Rh(D) positive, leukocyte-reduced, irradiated, negative for Hb S, antigenically matched for C, E, K1, and crossmatch compatible at the anti-human globulin phase. He experienced citrate toxicity during his exchange and was given additional calcium and a 250 mL NS bolus. He improved and completed the procedure. His pre-exchange Hb was 10.3 g/dL and Hb S fraction was 85%. His post-exchange Hb was 10.4 g/dL and Hb S fraction was 22%.

**Results :** He received pre-transplant conditioning with alemtuzumab on SCT D-7, D-6, D-5, and D-4 and total body irradiation 300 cGy on SCT D-2. Hb peaked at 10.4 g/dL after RBCX, then dropped to 8.9 g/dL on SCT D-3, and continued to decline to 5.4 g/dL on SCT day plus 3 (SCT D+3). Type and screen on SCT D-3 showed a newly positive DAT, with 2+ strength against polyspecific and IgG reagents, and microscopic strength against complement reagent. Antibody identification revealed anti-D in the eluate. Since the recipient's blood type was Rh(D) positive, an Rh(D) variant was suspected, and genotyping revealed *RHD\*DIIIa-CE(9)* variant in the recipient and wild-type *RHD* in the donor. The partial DIIIa phenotype frequently goes unrecognized until alloimmunization following exposure.

In light of his new anti-D prior to receiving HSCT from an Rh(D) positive donor, he was further immunosuppressed with hydrocortisone 100 mg on SCT D-1, IVIgG on SCT D 0, dexamethasone 40 mg on SCT D 0 and SCT D+1, and rituximab 700 mg on SCT D+1. During hospitalization, he was transfused four units of Rh(D) negative RBCs. There were no signs of hemolysis.

He was discharged on SCT D+15. Serial anti-D titers on 2/15, 2/20, and 2/26/18 were 1:8. On 3/20/18, the anti-D was undetectable. He had successful bone marrow engraftment and has not had further hemolysis.

**Conclusion :** Rh(D) variants can threaten HSCT when a patient who is serologically Rh(D) positive develops anti-D antibodies following transfusion of Rh(D) positive RBCs. Rh genotyping should be used to identify the specific Rh(D) variant. In our case, the recipient had the clinically significant *RHD\*DIIIa-CE(9)* variant, and became alloimmunized against Rh(D) following RBCX. Fortunately, he had a successful HSCT.

(No Table Selected)

**DEGREE:**

David Allison : Other

David Loeffler : Other

Sally Campbell-Lee : MD

Lavinia Sereseanu : Other

Santosh Saraf : MD

Jason Crane : Other

Vladimir Vidanovic : MD

Chelsea Sheppard : MD

**DEGREE IF OTHER:**

David Allison : D.O.

David Loeffler : D.O.

Lavinia Sereseanu : M.L.S.

Jason Crane : D.O.

**PRIMARY AUTHOR?:**

David Allison : Selected

**ASFA Membership (Abstract Submission):** Yes, I am an ASFA member

(No Image Selected)

---

© Clarivate Analytics | © ScholarOne, Inc., 2019. All Rights Reserved.

ScholarOne Abstracts and ScholarOne are registered trademarks of ScholarOne, Inc.

ScholarOne Abstracts Patents #7,257,767 and #7,263,655.

 @ScholarOneNews |  System Requirements |  Privacy Statement |  Terms of Use

Product version number 4.16.0 (Build 80). Build date Tue Mar 12 14:56:36 EDT 2019. Server ip-10-236-28-233