

# hr Problems

**American Red Cross** 

Is this a discussion of your Human Resource ghtmares?

# We are actually talking about the Rh Blood Group System!



hr Problems

## Hr and hr Antigens

## Hr<sub>o</sub> (Rh17) Hr or Hr<sup>s</sup> (Rh18) Hr<sup>B</sup> (Rh34)

## hr<sup>B</sup> (Rh31) hr<sup>S</sup> (Rh19)

These antigens are extremely high prevalence with an incidence of >99.9%. The antigens are lacking on *Rh-deletion* haplotypes. These antigens have an incidence of about 98%, just like the e antigen (Rh5). These antigens are considered e variants. Since the antigens are not as prevalent, we encounter antibodies to these antigens on a more frequent basis.



## The History of hr<sup>S</sup> and hr<sup>B</sup>

The antigen hr<sup>s</sup> was first identified in 1960. The serum of a Bantu woman, Mrs. Shabalala contained an antibody which reacted with all cells that possessed E or e antigen. The antibody which remained after adsorption with R2R2 cells was named anti-hr<sup>s</sup>.

The antigen hr<sup>B</sup> was not identified until 1972 and was found in a South African woman named Mrs. Baastian. Reactivity was very similar to anti-hr<sup>S</sup>.



## Partial Expression of the e Antigen

"In the majority of, but not quite all cases, e and anti-e in Whites, are straightforward. The antibody can be formed by E+e- (usually R2R2) people and when made, reacts with all e+ samples. In Blacks, the story is rather different for some of them, who have red cells that type as e+, make antibodies that closely resemble anti-e (or anti-f or anti- $rh_i$ ), yet which do not react with the antibody makers' own red cells. In this respect, it seems that the e antigen must involve many epitopes and that the state of lacking some of them, that is having red cells that carry partial e, is more common in Blacks than in Whites."

Applied Blood Group Serology, 4th edition; Peter D Issitt, David J. Anstee; Chapter 12, pg 368



Can we think of partial e antigen the same way as partial D antigen?

- No we 't!
- Partial D has very specific definitions and the epitopes are well defined.
- Partial e seems to be made in a more individualistic manner. The antigens and antibodies are much more difficult to define.



## Partial Expression of the e Antigen

The e antigen must involve many epitopes and the state of having red cells that carry partial e antigen is more common in African-Americans than in Whites.

Cells that lack one epitope of e are more than likely to lack *more* than one epitope. This brings in a lot of variation when looking at a partial e antigen. An added complication is that not all red cell samples called hr<sup>S</sup>- or hr<sup>B</sup>-, or all antibodies called anti-hr<sup>S</sup> or anti-hr<sup>B</sup>, are as alike as the names imply. Examples frozen in Reference and research laboratories over the years have revealed that many examples of "anti-hr<sup>B</sup>" are incompatible with cells identified as "hr<sup>B</sup>-".

The safest way to report these antibodies without extensive serological and molecular testing is "anti-e-like".





## Laboratory Management of e-like antibodies

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## Could my patient have an anti-e-like antibody?

- The patient would type as e+.
- Anti-e (or anti-f or anti-rh<sub>i</sub>) is identified in the serum.
- The DAT is negative.
  - CAUTION: the patient's DAT may be positive due to an autoantibody, a transfusion reaction (is anti-e identified in the eluate too?), or just because nothing is easy in the Blood Bank!
- The patient's race would most likely be African-American.



## What about transfusion recommendations?

- Is your patient E+? Your answer is easy: Transfuse e- units (R2R2)
- If your patient has anti-E already, the answer is more difficult. Precise information about the clinical significance of anti-hr<sup>B</sup> and anti-hr<sup>S</sup> is limited. Even more limited is the availability of compatible units for these patients. Throw in the additional complication that many of these patient's red cells also possess a partial D antigen (and so they can make allo-anti-D as well), and transfusion becomes very difficult. In an emergency, transfuse a patient with anti-D, -E, and -e-like antibodies with D-, E- e+ units.



## But we need compatible units!!!

- Molecular testing can identify patients and blood donors with altered Rhce/RhD.
- The American Rare Donor Program keeps a file of all the donors for whom they have molecular testing results which indicate partial e phenotype. If a request comes in to the ARDP, they can match donor to patient based on the exact molecular types of each. The ARDP notifies each member Blood Center who has compatible donors in order for them to ship products where they are needed or recruit their donors.
- Unfortunately this is not a quick process, and patients may go untransfused or need incompatible blood.



#### IMMUNOHEMATOLOGY STUDIES

#### **Case study 1**

This sample was from an African American sickle cell patient with a previously identified anti-E, -K, and -Js<sup>a</sup> underlying a warm autoantibody. We had not tested a sample from this patient up since 2009.

The patient had been transfused on 01/17/2014, and the specimen was dated 02/19/2014.

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#### **Case study – could this be an anti-e-like antibody?**

A selected cell panel of E-, K-, Js(a-) cells was tested. Looking at the strength of the serum reactivity at PEG/IAT, we noted stronger reactions with the test cells compared to the reactions with the autocontrol. The eluate was reactive with all cells tested.





#### **Adsorption studies**

Adsorptions were performed using PEG and untreated allogeneic cells.

Following adsorptions, an apparent anti-e was detected in the R2R2 adsorbed serum.

What do we need to do to determine if the patient has developed an allo-anti-e-like antibody or whether the specificity is simply a component of the autoantibody?

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#### **Testing with DAT Negative Autologous Red Cells**

A cell separation was performed using hypotonic 0.3% saline. The separated autologous red cells were treated with EGA (EDTA Glycine-Acid) to remove bound IgG. The DAT negative autologous red cells were tested against the patient's eluate and the R2R2 adsorbed serum. The eluate and serum were reactive, confirming the presence of an autoantibody, rather than an alloantibody.

If the DAT negative autologous cells were non-reactive, it would have indicated that the reactivity in the patient's serum was due to an alloantibody.

Another sample from this patient was submitted on 03/19/2014. The warm autoantibody was still reactive, with no auto-anti-e specificity detected.

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#### Case study 2

This sample was from an African American sickle cell patient collected 12/28/2013. He had been transfused in 2011, but had no recent transfusions.

The patient was B Positive, D+,C-,E+,c+,e+; M+,N+,S-,s+; K-; Fy(a-b-); Jk(a+b-); Le(a-b+), P1+





#### **Initial panel**

All cells but one were reactive with the initial panel at PEG-IgG. The results showed some variability in the strength of reaction.

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#### Phenotypically similar selected cells

Often when we are puzzled, we will test cells that are phenotypically similar to the patient. These can show us whether we are looking at multiple antibodies or a high prevalence antibody.

In this case, two cells were positive and one was negative. We've now noticed that both our negative cells are R2R2.

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#### Additional e- selected cells

We test additional e- cells which are positive for antigens which our patient's cells lack. What we find is a pattern of anti-Fy<sup>a</sup> and anti-Jk<sup>b</sup>.

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#### **Confirmation of alloantibodies**

Additional cells confirm the presence of anti-Fy<sup>a</sup>, and anti-Jk<sup>b</sup> in the patient's serum along with the anti-e-like antibody.

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#### Is this anti-hr<sup>B</sup> or anti-hr<sup>S</sup>?

We know the patient is e+ with an anti-e-like antibody which does not react with his own red cells.

We have several examples of anti-hr<sup>B</sup> frozen, so we use them to type the patient's red cells. Unfortunately we do not have any ABO compatible anti-hr<sup>S</sup>.

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#### **Selected rare cells**

We have some cells frozen which have been characterized as hr<sup>s</sup> negative, so we tested them against the patient's serum. These e+, hr<sup>s</sup>- cells are nonreactive. Can we go ahead and call this anti-hr<sup>s</sup>?





## Molecular Confirmation and Transfusion Recommendations

- The patient's cells were submitted for molecular characterization of the e antigen. The cells were confirmed as hr<sup>s</sup>- and the exact molecular variation of the patient's antigen was determined.
- If you recall our patient's phenotype, they were E+. We were unable to rule out anti-K, so we recommended the transfusion of e-, K-, Fy(a-), Jk(b-) units. Of course these are still quite rare, but not as hard to find as E-, hr<sup>s</sup>- units.



## Conclusions

Generally when a patient has an autoantibody, we don't recommend antigen negative units for transfusion, as this may immunize them to the alternate antigen. In the case of an apparent auto-anti-e, patient history including race needs to be strongly considered. Additional testing must be performed to show whether the specificity is autoantibody or alloantibody.



## Conclusions

Patients with e-like alloantibodies who type e+ should be tested molecularly to characterize their e antigen. This is particularly important for E- patients who may become immunized to both E and e.



## Conclusions

It is always best to avoid transfusion if you have a patient with anti-e-like antibody. In an emergency, transfusion of e+ red blood cells may be necessary.



### Questions



