Case Study: Identification of a high prevalence antibody; multiple, common alloantibodies; and a warm autoantibody after transfusion of 1 unit of packed cells in a patient with a previously negative antibody screen

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**Background/Case Studies:** The Js^b^ antigen is a high prevalence antigen, added to the Kell blood group system in 1965. It is antithetical to Js^a^ and is expressed in >99% of all ethnicities. The Js(b-) phenotype is found in 1% of persons of African ancestry. Like other antigens in the Kell blood group system, Js^b^ is sensitive to thiol reagents such as 0.2M dithiothreitol (DTT) as well as EDTA Glycine Acid (EGA).

The f antigen is part of the Rh blood group system and is expressed when the c and e antigens occur on the same haplotype such as with the R_0P_0 (Dce/Dce) genotype. When the c and e antigens are inherited on separate haplotypes, as is the case with the R_1R_2 (Dce/Dce) genotype, the f antigen is not expressed. The f antigen is present in 65% of Caucasians and 92% of Blacks. Here we present a case of an African American female presenting for total hip arthroplasty revision. She had history of a previously negative antibody screen done 4 months prior, at which time she received 1 unit of packed red blood cells (PRBCs). She presented a week before her scheduled surgery with an Hgb/Hct of 11.5/34.6. A type and screen was ordered and the hospital blood bank submitted the sample to the Immunohematology Reference Laboratory (IRL) reporting that their testing showed panagglutination.

**Study Design/Methods:** The patient typed as group A, D+ with a positive direct antiglobulin test (DAT) using anti-IgG. The patient’s plasma reacted 2+ with all reagent red blood cells (RBCs) by LISS-IAT and 3+ by PEG-IAT. The autocontrol was significantly weaker than the reagent RBCs, reacting weakly positive macroscopically by LISS-IAT and 1+ by PEG-IAT. The eluate made from the patient’s red cells also showed panagglutination at IAT. A serologic phenotype was performed. Autologous cells treated with EDTA/glycine-acid (EGA) and chloroquine diphosphate (CDP), to obtain a negative DAT, were used for typing with reagents using the indirect antiglobulin procedure. The results were C+, E+, c+, e+, K-, k+, Fy(a+b+), Jk(a+b-), S-, s+, M+, N+.

Adsorption studies were performed at 37°C with ZZAP-treated autologous cells. The autoadsorbed plasma reacted at the same strength as the neat plasma with all reagent RBCs tested at LISS-IAT. When the autoadsorbed plasma was tested against phenotypically similar, ficin-treated RBCs the reactivity was unchanged; however, no reactivity was seen with phenotypically similar, 0.2M DTT-treated RBCs. Further serologic phenotype testing was performed, and results were Kp(a-b+), Js(a+b-). Based on the additional phenotype testing and the antigen’s sensitivity to DTT, anti-Js^b^ was suspected. The autoabsorbed plasma was then tested against 0.2M DTT-treated screening cells, and reactivity of 1-2+ was observed at LISS-IAT.

Alloadsorptions at 37°C were performed. Testing with the alloadsorbed plasma confirmed the presence of anti-f, -K, -Fy^a^, -Jk^b^, -S, and -C^w^.

A panel of phenotypically similar, Js(b+) reagent RBCs was tested before and after 0.2M DTT treatment. The patient’s neat plasma tested positive at LISS-IAT with all cells before DTT treatment, but tested negative with all DTT-treated cells, confirming the anti-Js^b^.

To prove the warm autoantibody, EGA-treated, DAT negative autologous cells were tested against the neat plasma and eluate, producing positive reactions.
Results/Findings:

<table>
<thead>
<tr>
<th>Anti-A</th>
<th>Anti-B</th>
<th>Anti-A,B</th>
<th>Anti-D</th>
<th>Anti-D control</th>
<th>A1 cells</th>
<th>A2 cells</th>
<th>B cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
</tbody>
</table>

Poly AHG  IgG AHG  C3b,C3d AHG  Saline Control

|          | 1+     | 0      | 0      | 0           |

C          E          c          e          K          Fy<sup>a</sup>          Fy<sup>b</sup>          Jk<sup>a</sup>          Jk<sup>b</sup>

|          | +      | +      | +      | +          | 0       | 0<sup>*</sup> | +      | +      | 0     |

M          N          S          s          Kp<sup>a</sup>          Kp<sup>b</sup>          Js<sup>a</sup>          Js<sup>b</sup>          C<sup>w</sup>

|          | +      | 0<sup>*</sup> | +<sup>*</sup> | 0<sup>**</sup> | +<sup>**</sup> | +<sup>**</sup> | 0<sup>**</sup> | 0      |

*Antigen typing performed using autologous cells treated with EGA  
**Antigen typing performed using autologous cells treated with CDP

Conclusions: Anti-Js<sup>b</sup>, -K, -Fy<sup>a</sup>, -Jk<sup>b</sup>, -S, -f, -C<sup>w</sup> and a warm autoantibody were identified using standard tube techniques. The physician informed the hospital blood bank that there was a 30% chance that a unit of blood would be needed during surgery and ordered 1 unit for standby. About 0.08% of Blacks are both Js(b-) and f-. African Americans make up less than 3% of blood donors. The hospital was informed that blood would have to be procured through the American Rare Donor Program (ARDP) and may be difficult to find. Recommendations that the patient’s siblings be tested, and that the patient consider donating autologous units were made. The patient was taken to surgery and no blood was needed.

References: