AIHA CASE #5 ANSWERS

Case study by Jim Perkins, M.D. (© 2009)

Indian Immunohematology Initiative

1. What is the specificity of this patient's antibodies? Are they allo- or autoantibodies or both? Would an adsorption procedure help determine this?

The antibody shows anti-D+C+E-like specificity in gel. With PEG enhancement the plasma reacts with all cells, but there is still a hint of specificity. A panagglutinin is recovered in the eluate, but that does not indicate that the plasma reactivity is due to autoantibody, since a large fraction of the patient's circulating RBCs must be of donor origin and could adsorb alloantibody(s). Similarly an autoadsorption can't be done for the same reason.

The pattern of seeing Rh specificity in the plasma but a panagglutinin in the eluate is common with autoantibodies showing specificity as illustrated in AIHA case #3 above. Although we did not have a history of the patient's previous serologic findings from the other hospital, at the very least we can assume he is D positive. This strongly suggests that, to the extent his antibodies show D specificity, they are autoantibody. (Other rare possibilities include the patient having a partial D antigen.)

One could perform alloadsorptions. Since we do not know the patient's phenotype, we would have to use multiple adsorbing cells in an to reveal one or more alloantibody specificities. If time permitted we could determine his genotype in order to direct selection of the adsorbing cells. Alloadsorption can't rule out an alloantibody directed against a high frequency antigen (see "A New Panagglutinin in a Transfusion-Dependent Patient" in the AIHA clinical case studies), but such alloantibodies are uncommon.

Note that the plasma antibody is reacting only weakly by the gel and PEG techniques which are very sensitive to warm autoantibodies, but that the plasma antibody(s) don't react in a LISS enhanced IAT. Although this technique is less sensitive to both autoantibodies and some alloantibodies, it was the method of choice for antibody detection and identification for years, and is strong evidence that there is not a clinically significant alloantibody.

2. Is the extended Rh phenotype valid? What does it show? How could you determine a true Rh phenotype for the patient?

The phenotype is not valid since the patient has recently been transfused. In fact, the strong mixed field reactions indicate that most of his RBCs are of donor origin as discussed above. A phenotype performed on a subsequent sample after a 4 month transfusion hiatus showed him to be D pos, C neg, c pos, E pos, e pos. Today the phenotype could be predicted by DNA testing of the patient's white cells, but such tests were not generally available when this patient presented.

3. How would you provide safe RBC transfusion for this patient?

Based on the negative LISS screen our laboratory would select RBCs that were compatible by a saline/tube IAT that was in use for the crossmatch at that time. Once we had the patient's Rh phenotype one might select phenotype-specific RBCs.