AIHA CASE #1, ANWERS

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

Indian Immunohematology Initiative

1. What antibody(ies) are present? Is this allo- or auto-antibody?

The patient appears to have a warm autoantibody in the serum and the eluate that acts as a pan-agglutinin, as well as an underlying alloanti-Jk^a. The warm autoantibody was presumably present when she was transfused at the other hospital 2 months earlier, but there was no mention of anti-Jk^a in her history from that institution. The presence of anti-Jk^a may be due to a primary or secondary (anamnestic) antibody response and may have caused a DHTR.

2. What does the auto-adsorption accomplish? Is this auto-adsorption result valid?

The autoadsorption removed the autoantibody from the patient plasma by adsorbing it onto the patient's own RBCs, "leaving behind" the underlying anti-Jk^a which was otherwise obscured by the panagglutinin.

Autoadsorption might not leave behind underlying alloantibody if there were still circulating Jk^a-positive donor RBCs from the previous transfusion. For this reason it is taught that autoadsorption is only valid if there is no recent transfusion, which is generally given as 3 months, slightly less than the maximum life of a red blood cell. Note however that such adsorption of alloantibody along with the autoantibody would only cause one to MISS an underlying alloantibody; if an alloantibody IS present in the autoadsorbed serum that is a valid finding. That is, the presence of donor cells admixed with the patient's own could theoretically cause a false negative result, not a false positive.

Moreover, anti-Jk^a is a highly hemolytic antibody, and likely had destroyed the donor RBCs before the two months had passed after transfusion. And even had the patient not developed anti-Jk^a the donor RBCs would have had a shortened survival due to accelerated destruction by the autoantibody.

3. No antigen phenotype(s) was done. What is the problem(s) for performing an antigen phenotype in such a case? Could one have been done? How?

At the time of this case commercial anti-Jk^a typing sera were polyclonal IgG of human origin, and required an AHG phase to determine a phenotype. Since the patient's cells were already coated with antibody, in this case autoantibody, it would have been difficult to type them. However, in many cases a gentle elution with EDTA-glycine reagent or chloroquine will remove autoantibody without damaging the RBCs so that they can be typed. Again there is a concern that donor cells might be circulating invalidating the phenotyping result, but this is unlikely for the same reasons cited above. In cases in which it is an important consideration, reticulocytes (patient only) can be isolated by centrifugation in capillary tubes and used as a target for the typing sera.

4. How would we select compatible RBCs for this patient?

We would select group O, Jk^a negative units and crossmatch them with autoadsorbed plasma.