A NEW PANAGGLUTININ IN A TRANSFUSION-DEPENDENT PATIENT

ANSWERS:

1. What is the probable identity of this antibody?

   *This patient’s panagglutinin most likely represents a warm autoantibody. Warm autoantibodies and immune hemolytic anemia are common in patient’s with CLL.*

2. Why did an alloadsorption have to be done, rather than an autoadsorption?

   *Because the patient was transfusion dependent, all of his circulating RBCs were of donor origin. If there was a new alloantibody underlying the presumed warm autoantibody, cells with the corresponding antigen might still be circulating, and could adsorb it out.*

3. Why was no blood group phenotype done?

   *The RBCs circulating in the patient were all of donor origin since he was transfusion dependent. They could be expected to have a variety of blood group phenotypes, and the result would not be useful in determining what alloantibodies he could make.*

4. Is there any other possibility? Could the findings represent an alloantibody? What are the criteria for proving the presence of an autoantibody?

   *Yes, these findings could represent an alloantibody directed against a high frequency antigen. In that case, most donor RBCs would have the antigen, and it would appear as a pan-agglutinin, as do most warm autoantibodies. Since it would be bound to all of the circulating [donor] RBCs, the DAT would be positive without any sign of a mixed field reaction as is usually seen in a primary immune response or delayed hemolytic reaction. Moreover, the eluate would again have what appeared to be a warm autoantibody, because it would react with virtually all donor RBCs.*

   *The criteria for proving that an antibody is an autoantibody include the fact that it is eluted off of RBCs that come from the person forming it, or that that persons own RBCs can adsorb it. But in a transfusion dependent patient, autologous RBCs are not available.*

5. Why wasn’t the tentative identification of anti-Lu3 accepted? What is the identity of this antibody?

   *NON-reactivity of the antibody with cord RBCs and resistance of the reactions to AET pre-treatment of the RBCs were felt to favor anti-AnWj. This specificity was then supported by the fact that the antibody failed to react with two AnWj negative RBC samples. It is not anti-Lu3 because it reacted with Lu(a-b-) RBCs of the autosomal recessive type, and because the Lutheran genotype performed at the NYBC was LU BB.*

6. Why are most Lu(a-b-) cells non-reactive?

   *Because AnWj antigen is suppressed (but not absent) along with Lutheran antigens in the most common form of the Lu(a-b-) phenotype, the so called “inhibitor” type. However, in individuals with the recessive form of the Lu(a-b-) phenotype, the Lutheran genes are deleted or inactivated, and AnWj expression is normal.*

7. How would you transfuse this patient?

   *Look for compatible RBCs from a sibling or from a rare donor registry. In this case in(Lu) units proved to survive longer than AnWj pos RBCs and enough were available to support him for a year. Screening for other alloantibodies can be (and was) done with panels of cord cells.*