ABID CASE #10, ANSWERS

Case study by Jim Perkins, M.D. (© 2008)

Indian Immunohematology Initiative

1. What is the identity of this antibody(ies)?

   Alloanti-P1, warm reactive

2. Is any further workup needed to prove it? Comment on the sensitivity of the gel and saline/tube techniques in this case.

   No further workup is needed to prove the identity of the antibody in this case. The gel and saline/tube panel reactivity is similar in strength. Four (4) P1-positive cells were non-reactive in gel, and the technologist probably ran the saline/tube panel hoping it would be more sensitive because of the immediate spin phase, a room temperature reaction. However, the inconsistent reactions in gel may have more to do with the strength of the P1 antigen on the panel cells than with the sensitivity of the test method to the antibody; P1 expression varies widely between different individuals. This is why it is preferable to have cells with strong P1 expression notated in the manufacturer's panel to help in ruling out anti-P1.

   Soluble P1 substance prepared from hydatid cyst fluid will neutralize anti-P1 and can be used in antibody identification in analogous fashion to Lewis substance as demonstrated in the previous problem (ABID #10).

3. What is the probable source of the immunizing stimulus in this case?

   Anti-P1 is typically a non-red cell immune ("naturally occurring") antibody but can be stimulated by close contact with pigeons and by infection with a number of parasitic worms. Red cell immune anti-P1 is uncommon.

4. Does this antibody cause hemolytic transfusion reactions (HTRs)?

   Anti-P1 that is reactive at 37^{o} or in the Coombs' test, as does this one, is considered potentially hemolytic. There are a handful of case reports of severe, even fatal IHTRs due to strong 37^{o} reactive anti-P1. Only rare DHTRs are reported.

5. Does this antibody cause hemolytic disease of the fetus and newborn?

   HDFN is not reported.

6. How would we select compatible blood for this patient? What percentage of donors is expected to be compatible with this recipient?

   For the uncommon warm-reactive anti-P1 such as this we would transfuse Coombs' crossmatch compatible RBCs demonstrated to be P1-negative by reagent typing sera. About 20 to 25% of Caucasians would be expected to be compatible with this antibody but the number would be lower in donors with African descent and much higher in Asian populations (as many as 70% of Japanese compatible).

7. What is the biochemical nature of the antigen? (Review the outline of the features of the relevant blood group system.)

   P1 and related antigens such as P^{k} and P are carried by oligosaccharides on glycosphingolipids of the RBC membrane bilayer synthesized by glycosyltransferases. An antigen termed P (now P1) was discovered by Landsteiner and Levine by injecting human RBCs into rabbits. What was once considered as one blood group system is now divided into 3 "systems' and one "collection", but at the hospital level one would not expect to encounter antibodies other than anti-P1 more than once or twice in an entire career. The two most important of these blood group systems are the "P1Pk system" (antigens P1, Pk, and NOR) and the "Globoside" system (P antigen). The P1 and P^{k} antigens are synthesized from different substrates by the same galactosyltransferase acting on different precursors, paragloboside and lactosylceramide respectively. In most individuals P^{k} is converted to the very high prevalence P antigen "globoside", the most common glycosphingolipid in the RBC membrane.

   Besides anti-P1, the important antibodies to be aware of in relation to these systems are autoanti-P, the so-called Donath-Landsteiner antibody, which causes paroxysmal cold hemoglobinuria, and anti-PP1P^{k} which causes severe IHTRs and recurrent abortions.