ABID CASE #18, ANSWERS

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1. As a start to interpreting this case summarize the findings in the antibody detection tests (gel and saline tube methods) , and in each of the panels.

   The *gel antibody screen is positive with both screening cells. The cold antibody screen by the saline/tube technique suggests the presence of a cold autoantibody detected only at 4°C (although a 15° incubation was not performed); neither I/i nor H specificity is suggested since the group O screening cells react at the same strength as the umbilical cord blood cells and the patient's own group A cells, respectively.*

   The two gel panels show reactivity with most panel cells which is variable but relatively strong with some panel cells. Certain alloantibodies appear to be ruled out based on the two non-reactive cells in the first gel panel including anti-c, -e, -k, -Fy^b, -Jk^b, - Le^b, -S, -M, and -P1, and inspection of the pattern of strong and weak reactions does not suggest an alloantibody specificity. Note that the first panel suggested that the patient had anti-s, but in the follow-up gel panel the serum reacted with all cells. In addition, the patient was shown to express the s antigen, so if the antibody were anti-s it would be an auto-antibody. However, the DAT was negative, which is against an auto-anti-s.

   The LISS/tube panel is completely non-reactive , but the PEG/tube panel shows an alloantibody of anti-Fya specificity.

2. What antibody(ies) do you think are likely present? Is(are) this(these) allo- or auto-antibody? Is your interpretation proven?

   Alloanti-Fy^a appears to be proven by the PEG/tube results (7 of 7 Fy^{a+} cells reacting; all 4 Fy^{a-} cells and the autocontrol not reacting; other alloantibodies ruled out per protocol with the exception of anti-Le^a; the patient is Fy^{a-}). The lack of other reactivity in the LISS/tube and PEG/tube panels suggests that the non-specific positive reactions in the gel panels are an artifact of the gel method, or due to a so-called "gel-dependant antibody, as seen in cases #16 and #17.

   There also appears to be a weak cold autoantibody as mentioned above, but this is within the limits of what could be seen in any patient or blood donor. The cold autoantibody could be proven by demonstrating that it is removed by cold autoadsorption.

3. Comment on the sensitivity and specificity of the different techniques used for the panels. Had the autocontrol been performed with the gel panels would it have changed your answer? What else might you have done to substantiate your interpretation?

   This case is presented as another example of the approach to nonspecific reactivity encountered in gel testing and is analogous to how one might investigate such reactions detected by solid phase techniques. Had an autocontrol been performed with the initial gel panel and been interpreted as positive, one might have been tempted to explain the reactivity as due to cold autoantibody. But if the reactivity in gel is artifact, perhaps that artifact would have affected the autocontrol as well. One way to resolve this distinction would have been to perform a cold autoadsorption to remove autoantibody and then to test the autoadsorbed plasma against one of the same panels by the gel method. If all of the gel reactivity (except the anti-Fy^a) disappeared one could say that it was due to extreme sensitivity of the method to cold autoantibodies. If it did not disappear it would be consistent with a gel-dependent antibody. These same considerations would apply to case #17.

   This problem demonstrates the utility of having multiple IAT techniques available even if gel or solid phase is the primary method.

4. How would you select compatible blood for this patient?

   Standard protocol would be to crossmatch Fy^a-negative RBCs by the routine IAT method (in our case at the time 4 drops plasma, 1 drop donor RBC suspension). Some experienced serologists might add an IAT crossmatch using the method that showed the antibody, in this case PEG enhancement.