COMMUNICATIONS

Unexpected activity with monoclonal anti-B reagents

To the Editor:

In 1989 Beck and colleagues observed that potent monoclonal anti-A reagents agglutinated the red cells of 1 percent of group B blood donors. This spurious activity was attributed to overlapping specificity of abnormally high levels of D-galactosyltransferase and has been referred to as the B(A) phenomenon. Since Greenwell et al. had demonstrated similar overlapping specificity of α-3-N-acetyl-D-galactosaminy transferase in vitro, we predicted that potent monoclonal anti-B reagents would crossreact with group B samples. Shortly after, Voak et al. described the A(B) phenomenon, in which scant B receptors were detected on group A red cells. These B receptors were assumed to arise through the transfer of D-galactose by the A-gene-specific transferase.

Another mechanism by which B antigens may develop on group A red blood cells is through deacytlation of N-acetyl-D-galactosamine residues: the acquired-B phenomenon. In this reference laboratory, we expect to encounter no more than one or two examples of acquired-B each year. In the past 8 months however, we have observed five cases of apparent acquired-B antigen. In each case the group A red cells reacted with Gamma-Clone monoclonal anti-B (Gamma Biologics, Inc., Houston, TX). Reactions ranged from +w to 2+ under normal ABO grouping conditions. The Gamma-Clone anti-B reagent is based on ES4, a clone known to react well with acquired-B. This clone also provides the source material for the monoclonal anti-B produced by Immucor (Immucor, Norcross, GA). We were not surprised, therefore, to observe similar, but weaker, reactions when these samples were tested with Immucor’s Monoclonal anti-B reagents. In no case was agglutination demonstrated with human polyclonal anti-B reagents.

Users of monoclonal anti-B reagents prepared from ES4 should expect to encounter an increased incidence of samples that react unexpectedly with these reagents. These samples may not be classified as acquired-B by conventional polyclonal anti-B reagents. In our series of five cases, three individuals had a medical history typically associated with the acquired-B state. Of the remaining two, one was a patient with no clinical signs of infection; the other was a normal blood donor. Lack of a typical history in the latter two cases may militate in favor of the A(B) phenomenon although the acquired-B condition has been described in apparently healthy individuals.

Monoclonal anti-B reagents composed of ES4 may cause unexpected reactions with group A individuals. This activity may be due to the acquired-B or the A(B) phenomena.

Malcolm L. Beck, FIMLS, MIBiol
Community Blood Center of
Greater Kansas City
4040 Main Street
Kansas City, MO 64111

Mary A. Kowalski, MT(ASCP)SBB,
Julie R. Kirkegaard, MT(ASCP)SBB,
and Joan L. Korth, MT(ASCP)
Community Blood Center of
Greater Kansas City
Kansas City, MO