

Warm Autoimmune Hemolytic Anemia Case Study

A pretransfusion sample is received in your laboratory from a 61-year-old Caucasian female admitted for anemia. Patient history reveals a diagnosis of systemic lupus erythematosus (SLE) and no transfusions since receiving two units of blood two years earlier.

Laboratory findings are:

Hgb = 6.9 g/dL
Hct = 21%
Retic = 7%
Total Bilirubin 2.2 mg/dL
LDH = 450 U/L

Expected Results

ABO/Rh = O Pos

DAT = Positive (IgG only)

Antibody Screen = Positive with all cells tested (2-3+ IAGT)

Antibody identification panel = Positive with all cells tested (2-3+ IAGT) Autologous

control = Positive (2-3+ IAGT)

Some causes of positive DAT and/or positive autologous control:

- Transfusion Reaction
- Hemolytic Disease of the Fetus and Newborn (HDFN)
- Drug Induced Immune Hemolytic Anemia (DIIHA)
- Warm Autoimmune Hemolytic Anemia (WAIHA)

When considered along with the lab results presented as well as the patient history of SLE; Warm Autoimmune Hemolytic Anemia (WAIHA) is the most likely cause of the positive DAT and autologous control in the case presented. The presence of a warm autoantibody also explains the positive antibody screen and identification results. The warm autoantibody is coating the patient's red blood cells (positive DAT) and is also present in the patient's serum (antibody screen/antibody ID reactions).

Warm Autoimmune Hemolytic Anemia Discussion

To diagnose autoimmune hemolytic anemia (AHA), evidence of shortened red blood cell survival caused by autoantibodies directed against autologous RBCs is required. Approximately 80 percent of patients with AHA have warm-reactive autoantibodies while the remainder has cold-reactive autoantibodies.

Warm Autoimmune Hemolytic Anemia Case Study (cont.)

A typical presentation of AHA includes anemia, reticulocytosis, presence of spherocytes and polychromasia on the peripheral blood smear and a positive direct antiglobulin test (DAT). Increased indirect serum bilirubin, urinary urobilinogen and serum lactate dehydrogenase (LDH), and decreased serum haptoglobin are not required for the diagnosis but are often present.

Patients with AHA and no underlying associated disease are said to have primary or idiopathic AHA. AHA in patients with associated autoimmune disease (i.e. SLE) and certain malignant or infectious diseases is classified as secondary. The etiology of AHA is unknown.

Adsorptions

In a WAIHA case, it is important to detect and identify any underlying alloantibodies should transfusion be required. Since warm autoantibodies are typically panreactive in the antibody screens and panels, the detection and identification of any underlying alloantibodies can be difficult. An adsorption procedure can be performed to remove the autoantibodies by adding target antigens (RBCs) and allowing the antibody to bind to the antigen. Autologous red blood cells can be used for adsorption in patients who have not been transfused recently (in the last three months). For patients who have been transfused recently, an allogeneic adsorption procedure can be performed. The alloadsorption requires donor red blood cells of three different phenotypes be used to remove the autoantibody. For patient samples with very high titers of autoantibodies, subsequent adsorptions may be required. Once the plasma no longer reacts when tested with autologous red blood cells, an antibody screen and/or antibody identification panel can be performed to detect and identify any underlying alloantibodies.

Providing Blood for Transfusion

If an underlying alloantibody is identified, antigen-negative blood must be provided. Due to the panreactivity of the autoantibody present, all units tested will be incompatible. The patient's plasma, not adsorbed plasma, should be used for crossmatch testing and an autocontrol should be performed with each crossmatch. Donor units which show reactivity equal to or less than the autocontrol (i.e. least incompatible) may be selected for transfusion.

Reference

Roback JD, Coombs MR, et. al. *Technical Manual*. 16th ed. Bethesda MD: AABB; 2008.