soluble A and/or B Ags and ensuing inflammation and platelet activation.

Acute hemolytic reactions may present with hypotension, tachypnea, tachycardia, fever (+1–2°C), chills, chest and back pain, hemoglobinuria, and hemoglobinemia. In the most severe cases, DIC, acute renal failure, shock, and death may occur.

Delayed hemolytic reactions, with icterus and persisting or worsening anemia as the main clinical manifestations, result from an anamnestic response. Such reactions may occur in patients previously sensitized to RBC Ags who have a negative alloAb screen at the time of transfusion due to low Ab levels. The alloAb is detectable 1–2 weeks after the transfusion.

Diagnosis of transfusion-associated hemolysis relies on persistent and/or worsening anemia, depleted plasma haptoglobin levels, hemoglobinemia and hemoglobinuria, as well as elevated plasma lactate dehydrogenase and unconjugated bilirubin. The direct antiglobulin test (DAT, or direct Coombs test) that detects immunoglobulin, and possibly complement (C3d), on the surface of the recipient's RBC will most often be positive **(Fig. 113-2)**. Similarly, a positive indirect antiglobulin test (IAT, or indirect Coombs test) that detects anti-RBC alloAb in the serum will also be positive. An elution of the Ab on the surface of the RBC may allow for the identification of the culprit alloAb.