

Alpha Methyl dopa

[Afro Metal-dough-boy](#)

Various medications can precipitate the formation of warm agglutinin autoantibodies. These include alpha-methyl dopa, cephalosporins, diclofenac, ibuprofen, and levodopa. Some drugs stimulate the production of IgG autoantibodies, while others attach the RBC surface membrane and form haptens that induce IgG production.

Chronic Lymphocytic Leukemia (CLL)

[CaLL me Crone](#)

Some studies have shown that greater than 10% of patients with chronic lymphocytic leukemia also develop warm agglutinin autoimmune hemolytic anemia. CLL patients that are treated with purine analogs may be at an increased risk.

Systemic Lupus Erythematosus (SLE)

[Loopy-butterfly](#)

SLE is associated with warm agglutinin autoimmune hemolytic anemia. Patients may also have a positive Coombs test without any clinical evidence of hemolysis.

Diagnosis

Positive Coombs Test

[Positive Comb](#)

The Coombs test detects if a patient has formed autoantibodies to their RBCs. A Direct Coombs test involves mixing a patient's RBCs with test serum containing anti-IgG antibodies. If a patient has W-AIHA, their RBCs will be coated in IgG and anti-IgG in the test serum will agglutinate the RBCs; this reaction is referred to as a positive Coombs test.

Treatment

Corticosteroids

[Quarter-on-steroids](#)

Corticosteroids are used to treat many autoimmune conditions, including W-AIHA. Treatment helps to decrease antibody formation and increase hemoglobin. Approximately half of patients require long-term treatment with steroids.

Splenectomy

[Chopped-off Spleen](#)

Because much of the extravascular hemolysis in W-AIHA occurs in the spleen, splenectomy is the preferred second-line treatment for patients with W-AIHA refractory to corticosteroids.

Intravenous Immunoglobulin (IVIG)

[Ivy-gold-goblin](#)

IVIG, or intravenous immune globulin, has limited efficacy for treatment and is employed in refractory cases resistant to corticosteroids.

Rituximab

[Red-tux-mob](#)

Rituximab is a monoclonal antibody that works against B-cells, which produce autoantibodies. This drug is also a second-line treatment for patients who fail corticosteroids.