

Hemolytic Anemia

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, red blood cells live about 120 days, quietly carrying oxygen around your body. When they get old, your spleen gently takes them out of circulation and recycles their parts. But in destroying red blood cells (hemolytic) anemia, red cells are dying way too early — sometimes in days or even hours. Three things can cause this early death: your immune system can attack them by mistake (called autoimmune destroying red blood cells anemia, which can be warm or cold type, or triggered by a drug); something can physically smash them (like a leaky heart valve, clots in tiny vessels in conditions like TTP or HUS, or widespread clotting from DIC); or the red cells themselves can be fragile from birth (like in G6PD deficiency, sickle cell disease, thalassemia, or PNH). No matter the cause, the result is the same: your body is losing red cells faster than your bone marrow can make new ones. You get tired and pale (anemia). Your skin and eyes may turn yellow (jaundice) because broken red cells dump a pigment called bilirubin into your blood. Your spleen may swell because it's working overtime. Your urine can turn dark red or brown if free hemoglobin spills through your kidneys. And your bone marrow revs up, flooding your blood with young red cells (reticulocytes) to try to keep up. Every one of these signs is your body doing its best to cope — not the disease itself. The key is to trace everything back to the one upstream break and fix it.

What you might feel

- Pale skin and nail beds
- Feeling very tired and weak
- Yellow color to the skin or whites of the eyes (jaundice)
- Dark or tea-colored urine
- Fast heartbeat (tachycardia)
- Enlarged spleen (splenomegaly)
- Shortness of breath, especially with activity
- Chest pain or confusion (in severe cases)

Call the doctor right away if...

- Sudden, severe chest pain or trouble breathing—may mean the heart or lungs are in danger from too little oxygen
- Confusion, slurred speech, or fainting—signs the brain isn't getting enough oxygen
- Very dark or cola-colored urine with fever and back pain—may signal acute, life-threatening red blood cell destruction (hemolysis) or kidney damage
- Fast, pounding heartbeat that won't slow down, or new heart-failure symptoms (swollen legs, fluid in the lungs)—the heart is struggling to keep up

What the care team will do

- Monitor vital signs every 2 to 4 hours, focusing on heart rate and blood pressure, and assess for signs of shock like confusion or decreased urine output
- Check urine color every void and measure urine output hourly if dark urine is present or patient shows signs of severe red blood cell destruction (hemolysis)
- Administer oxygen to keep oxygen saturation above 92% and position the patient upright if they report shortness of breath
- Ensure type-and-crossmatch blood sample is drawn immediately and confirm that crossmatched blood is available at bedside before any transfusion
- Hold all new medications until the care team reviews the medication list, and immediately notify the provider if any drug known to cause red blood cell destruction (hemolysis) is identified
- Keep the patient warm with blankets and ensure room temperature is comfortable if cold agglutinin disease is suspected based on history or lab results
- Watch for and immediately report signs of thrombotic thrombocytopenic purple bleeding blotches (purpura) (TTP) including fever, confusion, pinpoint bleeding spots (petechiae) (tiny red spots), and low platelet count on labs
- Assess skin and eyes for yellowing of the skin and eyes (jaundice) (yellow color) every shift and monitor bilirubin trends on laboratory results

Medicines you may be given — and why

Prednisone (or methylprednisolone)

Prednisone calms the immune attack in warm autoimmune destroying red blood cells (hemolytic) anemia (warm AIHA). In warm AIHA, the body's own antibodies stick to red blood cells and flag them as invaders; the spleen then traps and destroys those cells. Prednisone tells the immune system to dial down — it shrinks the number of antibody-making immune cells and

slows antibody production. This means fewer antibodies coat the red cells, so fewer are destroyed. It works throughout the immune system (blood, lymph nodes, spleen, bone marrow).

Rituximab

Rituximab targets B cells, the immune cells that make antibodies. In both warm and cold AIHA, rogue B cells churn out antibodies that attack red blood cells. Rituximab locks onto a marker (CD20) on the surface of B cells and flags them for destruction. The immune system then clears those B cells away. With fewer B cells around, antibody levels drop, red blood cell destruction (hemolysis) slows, and red-cell counts climb. Rituximab is often used when steroids fail or to let patients taper steroids faster.

Folic acid

Folic acid (vitamin B9) is a building block for making new DNA. When red blood cells are being destroyed quickly (hemolysis), the bone marrow works overtime to churn out replacements. That high-speed factory burns through folic acid faster than normal. If stores run low, the marrow cannot keep up and anemia worsens. Folic acid supplements keep the supply full so the marrow can keep making healthy new red cells at top speed.

Eculizumab (or ravulizumab)

Eculizumab blocks part of the complement cascade, a chain reaction of immune proteins that punches holes in cells. In paroxysmal nocturnal hemoglobinuria (PNH), red cells lack a shield (CD55 and CD59) that normally stops complement from attacking; complement then blasts the cells open (intravascular red blood cell destruction (hemolysis)). In atypical destroying red blood cells (hemolytic) uremic syndrome (aHUS), runaway complement damages the kidney's tiny blood vessels, shredding red cells as they squeeze through. Eculizumab locks onto C5 (the protein that starts the final attack steps) and stops the hole-punching. Red cells survive longer, hemoglobin rises, and clots in small vessels (thrombotic microangiopathy) ease.

Hydroxyurea

Hydroxyurea treats sickle cell disease by boosting fetal hemoglobin (HbF), a version of hemoglobin babies make before birth. HbF does not sickle, so red cells with more HbF stay flexible and flow smoothly instead of forming rigid crescents that jam in capillaries. Hydroxyurea gently stresses the bone marrow, nudging it to turn fetal hemoglobin genes back on. Over weeks, HbF rises, sickling episodes drop, pain crises ease, and organ damage slows.

Plasma exchange (plasmapheresis)

Plasma exchange physically removes the liquid part of blood (plasma) and replaces it with fresh donor plasma or albumin. In thrombotic thrombocytopenic purple bleeding blotches (purpura) (TTP), a missing or blocked enzyme (ADAMTS13) lets huge sticky clumps of von Willebrand factor pile up in the bloodstream; platelets glom onto these clumps, forming

microscopic clots that shred red cells and starve organs. Plasma exchange yanks out the toxic clumps and autoantibodies (if present), and the replacement plasma brings in working ADAMTS13 enzyme. Clots dissolve, red blood cell destruction (hemolysis) slows, platelet count rises, and the brain and kidneys start to recover.

Supportive care (hydration, transfusion, avoid triggers)

Many destroying red blood cells (hemolytic) anemias are managed by supporting the body while it heals or by removing the trigger, rather than giving a specific drug. In G6PD deficiency, red cells lack an enzyme shield against oxidative damage, so avoiding oxidant drugs (sulfa antibiotics, antimalarials, fava beans) stops new red blood cell destruction (hemolysis); the body replaces destroyed cells on its own. In drug-induced immune destroying red blood cells anemia, stopping the culprit drug (penicillin, cephalosporins, methyldopa) lets the immune attack fade. In typical (Shiga-toxin) HUS, the toxin from *E. coli* O157 damages kidneys and shreds red cells; IV fluids protect kidneys, and the body clears the toxin naturally—antibiotics can make it worse, and plasma exchange does not help. In DIC (disseminated intravascular coagulation), treating the underlying trigger (sepsis, trauma, cancer) stops the clotting storm; transfusions of platelets, clotting factors, and red cells keep the patient alive while the trigger is controlled. Transfusion (packed red cells) is used across all destroying red blood cells anemias when hemoglobin drops dangerously low, but must be given carefully—in autoimmune cases, crossmatching is harder because antibodies attack many donor cells, and in chronic red blood cell destruction, repeated transfusions risk iron overload.

Where this information comes from

- American Society of Hematology (ASH) guidelines on hemolytic anemia
- UpToDate/NEJM clinical reviews on hemolysis
- Blood journal (American Society of Hematology)
- Cochrane reviews on diagnosis and management of hemolytic disorders

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