

Hemolytic Anemia

Destroying red blood cells (Hemolytic) anemia means red blood cells are breaking apart before their time. Your job is to find out why.

THE ONE-LINE TRUTH

Red blood cells are dying too fast — look for high LDH, low haptoglobin, high indirect bilirubin, and lots of young red cells (reticulocytes) trying to replace them.

The chain — how it unfolds

1. Red blood cells start breaking apart too early. — why: Whether from immune attack, mechanical shearing, or intrinsic fragility, red cells are dying in days or hours instead of 120 days. Hemoglobin spills out into the blood, and the marrow gets the alarm signal that red cell count is dropping.
2. Hemoglobin floods the blood and uses up haptoglobin. — why: Free hemoglobin is toxic if it stays loose. Haptoglobin tries to grab it and deliver it safely to the liver — but when red cells break in huge numbers, haptoglobin runs out. Lab result: haptoglobin drops to near zero.
3. Free hemoglobin breaks down into bilirubin, causing yellowing of the skin and eyes (jaundice). — why: Once hemoglobin is broken, the heme part becomes bilirubin (a yellow pigment). The liver can only process so much at a time, so bilirubin piles up in the blood (indirect or unconjugated type) and stains your skin and eyes yellow.
4. LDH spills out of broken red cells, shooting the lab value way up. — why: LDH is an enzyme inside red cells. When the cells pop open, LDH floods into the blood. A very high LDH (often over 1000) is a hallmark of red blood cell destruction (hemolysis) and tells you cells are being destroyed fast.
5. Bone marrow cranks out reticulocytes to replace lost red cells. — why: Your marrow detects the drop in red cells and goes into overdrive, releasing young red cells (reticulocytes) into the blood. A high reticulocyte count (often over 2-3%) proves the marrow is working and trying to compensate — this is actually a good sign that the marrow is healthy.
6. You get anemia, and your body shifts into oxygen-saving mode. — why: Fewer red cells means less oxygen delivery. You feel tired, weak, short of breath, and pale. Your heart beats faster to pump more blood per minute and make up the difference.

7. Spleen swells as it works overtime to clear damaged red cells. — why: The spleen is the main filter for abnormal red cells. When it's processing huge numbers of damaged cells, it enlarges (splenomegaly). You may feel fullness or pain in your left upper belly.
8. Free hemoglobin spills into urine, turning it dark red or brown. — why: Once haptoglobin is used up, free hemoglobin travels through the kidneys and into the urine. This is hemoglobinuria — it looks like cola or tea and is a red flag that red blood cell destruction (hemolysis) is severe. If it goes on too long, the hemoglobin can damage the kidney tubules and cause acute kidney injury.

Spot it — what you see, and why

- Pale skin and nail beds: Red blood cells are breaking apart faster than the bone marrow can replace them, so there aren't enough cells left to carry oxygen and give skin its pink color.
- Feeling very tired and weak: With fewer red blood cells, less oxygen reaches the muscles and brain, so the body runs low on energy—like a phone battery that won't charge all the way.
- Yellow color to the skin or whites of the eyes (jaundice): When red blood cells break apart, they spill a yellow pigment called bilirubin (the leftover from hemoglobin), and too much of it stains the skin and eyes yellow.
- Dark or tea-colored urine: The kidneys filter out extra hemoglobin and bilirubin from all the broken red cells, and these waste products turn the urine brown or red-brown.
- Fast heartbeat (tachycardia): The heart pumps faster to push what little oxygen is left around the body more quickly, trying to make up for the shortage of red blood cells.
- Enlarged spleen (splenomegaly): The spleen filters and destroys old or damaged red blood cells; when it has to work overtime clearing away all the broken cells, it swells up.

Red flags — act now

- 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

Treat it — in this order

1. Identify the cause: use DAT (Coombs test), blood smear, LDH, haptoglobin, and reticulocyte count to find whether red blood cell destruction (hemolysis) is immune, mechanical, or from an inherited red blood cell defect → Finding the specific upstream cause tells you exactly which treatment to use. Without knowing the cause, you cannot fix the destruction and only treat symptoms blindly.
2. Warm autoimmune destroying red blood cells (hemolytic) anemia (AIHA): start corticosteroids (prednisone 1 mg/kg/day); Cold AIHA: avoid cold exposure and give rituximab (375 mg/m² weekly for 4 weeks); Drug-induced: stop the offending drug immediately → Corticosteroids suppress the immune system so it stops making antibodies that attack red blood cells in warm AIHA. Rituximab kills B cells (the immune cells that make antibodies) in cold AIHA. Stopping the drug removes the trigger in drug-induced red blood cell destruction (hemolysis). Each treatment shuts down the immune attack.
3. Thrombotic thrombocytopenic purple bleeding blotches (purpura) (TTP): urgent plasma exchange (plasmapheresis) daily until platelets normalize; Atypical destroying red blood cells (hemolytic) uremic syndrome (HUS): eculizumab (complement C5 blocker, 900 mg IV weekly); Disseminated intravascular coagulation (DIC): treat the underlying trigger (infection, cancer, trauma); Paroxysmal nocturnal hemoglobinuria (PNH): eculizumab → Plasma exchange in TTP removes the antibody (against ADAMTS13) causing tiny clots that shred red blood cells. Eculizumab in atypical HUS and PNH blocks complement proteins (part of the immune system) from punching holes in red blood cell membranes. Treating the trigger in DIC stops the clotting cascade. Each stops the mechanical or complement-driven destruction.
4. G6PD deficiency: avoid oxidative triggers (sulfa drugs, nitrofurantoin, dapsone, fava beans, naphthalene mothballs); Sickle cell disease: hydroxyurea (15–35 mg/kg/day) to increase fetal hemoglobin; Thalassemia: regular red blood cell transfusions and iron chelation (deferasirox or deferoxamine) → Avoiding triggers in G6PD deficiency prevents red blood cells from breaking under oxidative stress. Hydroxyurea increases fetal hemoglobin, which does not sickle, reducing painful crises and red blood cell destruction (hemolysis) in sickle cell disease. Transfusion in thalassemia maintains oxygen delivery, and chelation removes excess iron from repeated transfusions to prevent organ damage.
5. Transfuse packed red blood cells if hemoglobin drops below 7 g/dL or if the patient has chest pain, shortness of breath, confusion, or rapid heart rate despite higher hemoglobin; use crossmatch-compatible units → Transfusion immediately restores oxygen-carrying capacity and stabilizes blood pressure and heart rate while the body or treatment stops destroying red blood cells. Crossmatching prevents the immune system from attacking the transfused cells.

Your top priorities at the bedside

1. 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 — why: Severe anemia reduces oxygen-carrying capacity, forcing the heart to pump faster to deliver enough oxygen to organs; red blood cell destruction (hemolysis) can drop hemoglobin quickly, and patients can develop a fast heart rate (tachycardia) (fast heart rate), low blood pressure (hypotension) (low blood pressure), and organ damage if red blood cell destruction outpaces the bone marrow's ability to make new cells
2. 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) — why: Free hemoglobin released during red blood cell breakdown is filtered by the kidneys and can block kidney tubules (the tiny tubes in kidneys that make urine), causing acute kidney injury; dark urine signals active red blood cell destruction (hemolysis) and kidney risk, requiring IV fluids to maintain urine flow above 0.5 mL/kg/hour
3. Administer oxygen to keep oxygen saturation above 92% and position the patient upright if they report shortness of breath — why: Low red blood cell counts mean less hemoglobin is available to carry oxygen from the lungs to tissues; supplemental oxygen increases the amount of oxygen dissolved in blood plasma (the liquid part of blood) and bound to the remaining hemoglobin, preventing tissue low oxygen (hypoxia) (low oxygen in organs)