

Upper GI Bleed

Blood is coming up from the esophagus, stomach, or top of the small intestine—give two big IV lines and fluids first to keep the pressure up, call GI for an urgent scope to find the leak, and stop it before the patient runs out of blood.

THE ONE-LINE TRUTH

Bleeding from the esophagus, stomach, or the first part of the small intestine—above the ligament of Treitz (the bend where the small intestine starts)—floods the gut with blood and drops the body's total blood volume fast.

The chain — how it unfolds

1. An ulcer crater in the duodenum erodes into the gastroduodenal artery, a varix in the esophagus ruptures from high portal pressure, or a Mallory-Weiss tear rips open submucosal vessels—blood pours into the esophagus, stomach, or duodenum at 50-500 mL/minute. — why: The protective mucus-bicarbonate shield is gone, and the bleeding vessel has nothing to clamp it shut—platelets try to plug it, but arterial pressure or the sheer volume of blood overwhelms the clot, so bleeding continues.
2. Total blood volume drops (normal adult has ~5 liters; losing 500-1000 mL is ~10-20% loss)—the body detects this instantly via baroreceptors in the carotid artery and aortic arch. — why: These pressure sensors send urgent signals up the glossopharyngeal and vagus nerves to the brainstem, triggering a full-body alarm: 'We are running out of blood—prioritize the brain and heart, sacrifice everything else.'
3. Heart rate jumps to 100-120 bpm (tachycardia)—this is usually the FIRST vital sign you see change, often before the patient feels anything. — why: The brainstem's sympathetic command center (in the medulla) fires norepinephrine down sympathetic nerves to the sinoatrial (SA) node in the heart, telling it to beat faster—more beats per minute means more oxygen delivered per minute, even with less blood per beat.
4. Skin turns pale, cool, and sweaty (diaphoresis); capillary refill slows to >2 seconds. — why: Sympathetic nerves squeeze arterioles in the skin and gut shut (vasoconstriction), shunting blood away from the surface toward the core (brain, heart, kidneys). Sweat glands are also triggered by sympathetic activity, so the patient feels clammy—this is the body in fight-or-flight mode.

5. Patient feels dizzy, lightheaded, or faints when standing up (orthostatic low blood pressure (hypotension)) after losing ~15–20% blood volume. — why: Normally, when you stand, leg veins and reflexes squeeze blood back up to the heart to keep brain pressure steady. With low blood volume, there is not enough blood to refill the system when gravity pulls it down, so brain blood flow (perfusion) drops and you gray out or pass out.
6. Vomiting blood (hematemesis)—either bright red (frank blood, means brisk active bleeding) or dark brown 'coffee-ground' (means blood has sat in the stomach long enough for gastric acid to denature hemoglobin into brown hematin). — why: The stomach fills with blood from the upstream bleed; the stomach tries to empty itself by triggering the vomiting reflex (signals from the stomach to the vomiting center in the medulla). If bleeding is fast, the blood is still red; if it oozes slowly, acid turns it brown.
7. Black, tarry, sticky, foul-smelling stool (melena) appears 8–24 hours after the bleed starts (requires ≥ 50 –100 mL of blood in the upper GI tract). — why: Blood travels from the stomach through the small intestine and colon. Bacteria and digestive enzymes break down the red blood cells, turning hemoglobin into hematin and other black pigments. The iron in the blood also oxidizes, making the stool sticky and tar-like with a distinctive smell—this tells you the bleeding source is above the ligament of Treitz (upper GI). Bright red blood in stool (hematochezia) usually means lower GI bleeding, but can occur with massive, rapid upper GI bleed (≥ 1000 mL) that rushes through so fast the blood does not turn black.
8. Blood urea nitrogen (BUN) rises out of proportion to creatinine (BUN:Cr ratio $>20:1$, sometimes $>30:1$). — why: Blood sitting in the gut is digested like food—proteins in the blood are broken down into amino acids, which are absorbed in the small intestine, converted to urea in the liver, and dumped into the bloodstream. The kidneys try to reabsorb urea to conserve volume (because of low blood flow and high ADH), so BUN climbs. Creatinine (a muscle waste product) is not reabsorbed, so it stays normal or rises only slightly from kidney poor blood flow (hypoperfusion)—this creates the high BUN:Cr ratio, a clue that bleeding happened in the GI tract, not just kidney failure.

Spot it — what you see, and why

- Throwing up blood (hematemesis) — can look bright red or like coffee grounds: Blood is leaking from a tear, ulcer, or ruptured vein in the esophagus or stomach, and the stomach pushes it back up through the mouth
- Black, tarry, sticky stools (melena) that smell very bad: Blood from high in the gut (stomach or upper small intestine) gets broken down by stomach acid and digestive enzymes, turning it black and tar-like by the time it comes out
- Bright red blood in the stool (hematochezia) when the bleed is very fast: So much blood is pouring out so quickly that it rushes through the intestines before it has time to turn black, so it stays red

- Feeling dizzy, lightheaded, or fainting when standing up: Blood loss lowers the total volume in the blood vessels, so less oxygen-rich blood reaches the brain — especially when you stand and gravity pulls blood down to your legs
- Heart racing (tachycardia) or pounding: The heart beats faster to try to pump the smaller amount of blood around the body fast enough to keep organs fed with oxygen
- Pale, cool, clammy skin: The body clamps down blood vessels in the skin to save blood for the brain, heart, and kidneys — like turning off water to the lawn to keep the house pipes full

Red flags — act now

- Throwing up bright red blood or large clots
- Feeling faint, very dizzy, or passing out
- Heart racing over 100 beats per minute at rest, or blood pressure dropping below 90 on top
- Confused, hard to wake, or very drowsy
- Bright red blood pouring from the rectum with an upper source (means massive, fast bleed)

Treat it — in this order

1. Two large-bore IV lines (18-gauge or bigger) and start isotonic crystalloid (normal saline or lactated Ringer's) resuscitation with a restrictive transfusion strategy (target hemoglobin 7–9 g/dL in most patients, 7–8 g/dL if no heart disease) → Replaces lost blood volume so the heart can pump enough blood to deliver oxygen to the brain, heart, and kidneys. Stops the body's panic signals: a fast heart rate (tachycardia), orthostasis, and low blood pressure (hypotension). Restrictive transfusion (not over-transfusing) prevents re-bleeding by avoiding high pressure that can pop off a fresh clot.
2. Intravenous proton pump inhibitor (PPI) bolus (80 mg) then continuous infusion (8 mg/hour) if non-variceal bleed is suspected (ulcer, gastritis, or erosion) → Stops the stomach from making acid. Acid dissolves clots and keeps ulcers open. PPI raises stomach pH above 6, so a clot can form and stick at the ulcer site. This cuts re-bleeding risk in half.
3. Upper endoscopy (EGD) within 24 hours (or sooner if the patient is unstable or has high-risk features like ongoing vomiting blood (hematemesis), shock, or suspected variceal bleed) → Lets the doctor see exactly where the blood is coming from—ulcer, varix, tear, gastritis, or cancer—and treat it on the spot. The doctor can place clips, bands, inject epinephrine (a medicine that squeezes blood vessels shut), or use heat (cautery) to seal the vessel. This stops the bleeding at the source.

4. Octreotide bolus (50 mcg IV) then continuous infusion (50 mcg/hour) PLUS ceftriaxone (1 g IV daily) if variceal bleeding is suspected or confirmed (patient has cirrhosis signs) → Octreotide (a synthetic hormone like somatostatin) shrinks blood flow to the splanchnic circulation (the vessels feeding the stomach and esophagus), so pressure drops in varices and they stop bleeding. Ceftriaxone (an antibiotic) prevents spontaneous bacterial peritonitis (a deadly gut infection) in cirrhotic patients — infection risk goes up during variceal bleeds. Together they cut death risk.
5. Reverse anticoagulation if the patient takes blood thinners: vitamin K (10 mg IV) for warfarin, prothrombin complex concentrate (PCC) or fresh frozen plasma (FFP) for urgent reversal, idarucizumab for dabigatran, andexanet alfa for apixaban or rivaroxaban → Stops the medicine from keeping blood too thin, so a clot can form at the bleeding site. This is critical if INR is elevated or the patient is on a direct oral anticoagulant (DOAC). Reversal must happen fast.

Your top priorities at the bedside

1. Establish two large-bore IV lines (18-gauge or larger) and start balanced crystalloid fluids. — why: Large IVs allow rapid volume replacement because hypovolemia (low blood volume) causes shock and organ damage; balanced fluids like lactated Ringer prevent hyperchloremic too much acid in the blood (acidosis) (too much chloride and acid in blood) that normal saline causes.
2. Place patient on continuous cardiac monitoring and pulse oximetry, measure blood pressure every 15 minutes. — why: Acute blood loss causes a fast heart rate (tachycardia) (fast heart rate) first, then low blood pressure (hypotension) (low blood pressure) and arrhythmias (irregular heartbeat); early detection allows you to intensify resuscitation before organs fail from poor blood flow (hypoperfusion) (inadequate blood flow).
3. Type and crossmatch blood, draw CBC (complete blood count), PT/INR (clotting times), liver panel, and lactate immediately. — why: Hemoglobin shows severity of anemia (low red blood cells); INR shows if blood thinners need reversal; lactate above 2 mmol/L indicates tissue low oxygen (hypoxia) (not enough oxygen) from poor blood flow (perfusion); liver tests identify cirrhosis causing varices.