

Upper GI Bleed

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The esophagus (swallowing tube), stomach, and duodenum (the first part of the small intestine, ending at the ligament of Treitz) are lined with a layer of mucus-secreting cells that also release bicarbonate (a base) to neutralize stomach acid and protect the tissue underneath. Just below this lining sits a rich network of blood vessels (the submucosal plexus) that supplies oxygen and nutrients to keep the tissue alive. The stomach also makes hydrochloric acid (a strong acid) in special cells called parietal cells to break down food, and pepsin (a protein-digesting enzyme) to help digest what you eat. Normally, the mucus barrier keeps the acid from burning the lining, and tiny injuries heal quickly because platelets (sticky blood cells) plug small leaks and clotting factors seal them shut.

BODY'S JOB

This upper GI tract moves food from your mouth to your intestines, breaks it down with acid and enzymes, and absorbs some nutrients. The mucus-bicarbonate layer acts like a shield so the stomach's own acid does not digest the stomach itself. The blood vessels underneath deliver oxygen and building blocks so the lining can repair daily wear and tear. When everything works, small scrapes seal themselves in hours and you never notice.

1. You swallow food → it slides down the esophagus (a muscular tube about 10 inches long) and passes through the lower esophageal sphincter (a ring of muscle that acts like a one-way gate) into the stomach.
2. The stomach (a J-shaped bag that holds about 1–1.5 liters) churns food with hydrochloric acid (pH around 1–2, strong enough to dissolve metal) and pepsin to break proteins into smaller pieces.
3. The stomach lining makes thick mucus (like clear snot) and pumps out bicarbonate (baking-soda-like base) to neutralize acid right at the surface, so the acid only touches food, not the stomach wall.
4. Blood flows into the stomach wall through the left gastric artery, celiac artery branches, and the gastroduodenal artery, forming a dense web of capillaries (tiny blood vessels) in the submucosa (the layer just under the lining) to feed the cells and carry away waste.
5. Partly digested food then moves into the duodenum (the first 10 inches of small intestine, shaped like a C around the pancreas), where more enzymes from the pancreas and bile from the liver finish breaking it down.
6. The ligament of Treitz (a band of muscle at the duodenojejunal junction) marks the end of the upper GI tract; anything above it is 'upper,' anything below is 'lower.'

What breaks

Something punches through, erodes, inflames, or overstretches the lining of the esophagus, stomach, or duodenum so that blood vessels underneath are exposed and start leaking blood into the gut.

CLINICAL TERM

Upper GI bleed (bleeding from the esophagus, stomach, or duodenum—above the ligament of Treitz). Causes split into non-variceal (not from swollen veins) and variceal (from burst veins backed up by liver disease).

How the body compensates

The body tries to defend blood pressure and oxygen delivery to the brain and heart by speeding up the heart, clamping down blood vessels in the skin and gut, and shifting fluid from tissues into the bloodstream—but these tricks only buy minutes to hours. Vital signs can look deceptively stable (normal blood pressure) even after losing 15–30% of blood volume, because young, healthy patients compensate hard. Then compensation fails suddenly, blood pressure crashes, and organs start dying from lack of oxygen. The job is to spot the hidden bleeding and act BEFORE the crash, because once the patient tips into shock, the window to save them narrows fast.

1. {'what': 'A fast heart rate (Tachycardia) (heart rate rises above 100 bpm, often the FIRST vital sign to change)', 'why': 'The baroreceptors (pressure sensors) in the carotid artery and aortic arch detect falling blood volume and send signals up the glossopharyngeal and vagus nerves to the brainstem (specifically the nucleus tractus solitarius). The brainstem tells the sympathetic nervous system to release norepinephrine (the body's adrenaline-like chemical), which makes the heart beat faster and harder to pump the remaining blood around the body more times per minute, keeping oxygen delivery up even though total blood is down.'}
2. {'what': 'Peripheral vasoconstriction (blood vessels in the skin, gut, and muscles clamp down; skin feels cool and clammy, capillary refill slows)', 'why': 'Sympathetic nerves release norepinephrine at alpha-1 receptors on smooth muscle cells in arterioles (tiny arteries) of non-essential organs, squeezing them tight to redirect the shrinking blood supply toward the brain, heart, and kidneys—the organs that will kill you fastest if they lose oxygen.'}
3. {'what': 'Fluid shift from interstitium (tissue spaces) into blood vessels', 'why': 'As blood volume drops, capillary hydrostatic pressure (the push of blood against vessel walls) falls, so the normal balance that pushes fluid out into tissues reverses—fluid is pulled back into the bloodstream by plasma proteins (oncotic pressure), temporarily boosting circulating volume by 10–15% over 30–60 minutes.'}
4. {'what': 'Increased respiratory rate (tachypnea)', 'why': 'The brainstem senses falling oxygen delivery and rising metabolic acid (lactic acid from tissues not getting enough oxygen), so it speeds up breathing to blow off CO₂ and try to keep blood pH normal (respiratory compensation for metabolic acidosis (acid building up in the blood)).'}
5. {'what': 'Orthostatic low blood pressure (hypotension) (blood pressure drops ≥ 20 mmHg systolic or ≥ 10 mmHg diastolic when standing, or heart rate rises ≥ 20 bpm)—appears after ~15–20% blood volume loss', 'why': 'When the patient sits or stands, gravity pools the remaining blood in the legs; there is not enough volume left to fill the vessels and maintain pressure in the brain, so dizziness, tunnel vision, or fainting (presyncope or fainting (syncope)) happens. This is a red flag that compensation is starting to fail.'}

6. {'what': 'Frank low blood pressure (hypotension) (systolic BP drops below 90 mmHg)—appears after ~30–40% blood volume loss (class III bleeding-driven (hemorrhagic) shock)', 'why': 'The sympathetic system has maxed out; there is simply not enough blood left to fill the vascular tree, so pressure collapses. Once BP falls, oxygen delivery to the brain, kidneys, and gut drops sharply—organs start dying within minutes to hours. This is the crash point.'}
7. {'what': 'Altered mental status (confusion, agitation, then lethargy)', 'why': 'The brain is exquisitely sensitive to low oxygen and low blood flow (cerebral poor blood flow (hypoperfusion)). Neurons in the cerebral cortex (thinking and awareness centers) malfunction when oxygen drops, causing confusion or combativeness; if it worsens, the reticular activating system (the brainstem network that keeps you awake) shuts down and the patient becomes unresponsive.'}
8. {'what': 'Low urine output (Oliguria) (urine output drops below 0.5 mL/kg/hour) or no urine output (anuria) (no urine)', 'why': 'The kidneys sense low blood flow and clamp down on urine production to save every drop of fluid (via renin-angiotensin-aldosterone system and ADH release). If shock persists, the kidneys themselves become blood-starved (ischemic) (starved of oxygen), and acute tubular tissue death (necrosis) (ATN, death of the filtering tubule cells) causes acute kidney injury.'}
9. {'what': 'DO NOT wait for low blood pressure (hypotension) to act—by the time BP drops, the patient has already lost 30–40% of blood volume and is in severe shock. Treat based on a fast heart rate (tachycardia), orthostasis, and clinical picture (hematemesis, black tarry stool (melena), fainting (syncope)), NOT just the blood pressure number.'}

Domino effect

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. — 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. — 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. — 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. — 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. — 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). — 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). — 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). — 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.

9. Hemoglobin (Hgb) and hematocrit (Hct) start to DROP—but this can lag 4–6 hours behind the actual blood loss, because the body has not yet pulled in enough tissue fluid to dilute the remaining red blood cells. — When you first bleed, you lose whole blood (red cells + plasma together), so the concentration of red cells in the remaining blood stays normal for a while—then, over hours, fluid shifts from tissues into vessels to restore volume, diluting the red cells and revealing the true anemia. DO NOT wait for the hemoglobin to drop before treating—treat the clinical picture (tachycardia, low blood pressure (hypotension), vomiting blood (hematemesis)).
10. If bleeding continues or resuscitation is delayed: systolic blood pressure crashes below 90 mmHg (hypotension), mental status deteriorates (confusion, then unresponsiveness), urine output stops (oliguria/no urine output (anuria)), and lactic acid rises (lactic too much acid in the blood (acidosis) from anaerobic metabolism in oxygen-starved tissues). — The heart is beating as fast as it can, vessels are clamped down maximally, but there is simply not enough blood left to fill the system and deliver oxygen—organs switch to anaerobic (without oxygen) metabolism, producing lactic acid and failing one by one. This is bleeding-driven (hemorrhagic) shock, class III–IV. Without immediate large-volume resuscitation (IV fluids, blood transfusion) and source control (endoscopy to stop the bleed), the patient will die from cardiovascular collapse or multi-organ failure.
11. Even after initial resuscitation, 10–20% of patients re-bleed within 72 hours, especially if the ulcer has a visible vessel on endoscopy or if varices are not banded. — The clot sealing the bleeding vessel is fragile—stomach acid, pepsin, and mechanical movement can dislodge it. Varices remain under high pressure from portal high blood pressure (hypertension), so even after banding one, another can rupture. Re-bleeding has high mortality, so close monitoring and repeat endoscopy (if needed) are essential.

Symptoms · what the patient feels, 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
- Belly pain or discomfort, especially burning in the upper stomach: An ulcer (a sore that has eaten through the stomach or intestine lining) releases stomach acid onto raw tissue and can bleed; the acid irritates the exposed nerves and causes pain
- Spider-like red blood vessels on the skin (spider angiomas), swollen belly (ascites), red palms (palmar redness (erythema)), or large belly veins (caput medusae): These are signs of liver disease — a scarred liver (cirrhosis) can't handle blood flow properly, so pressure builds up in veins around the esophagus and stomach, and those veins can rupture and bleed (variceal bleed)
- Confusion, sleepiness, or trouble waking up: Not enough oxygen is reaching the brain because blood volume or blood pressure has dropped too low from the bleeding

Focused exam

- A fast heart rate (Tachycardia) (heart rate > 100 bpm) and orthostatic low blood pressure (hypotension) (systolic BP drops ≥ 20 mmHg or diastolic drops ≥ 10 mmHg when standing, or dizziness on standing): The body is losing blood volume fast. The heart speeds up to try to keep oxygen moving to the brain and organs. When you stand, gravity pulls blood down, and if there is not enough blood left, pressure drops. This is your first warning that the body is struggling to keep up with blood loss.
- Vomiting blood (Hematemesis) (vomiting bright red blood or dark blood) or coffee-ground vomiting (emesis) (vomit that looks like wet coffee grounds): This tells you blood is coming from the esophagus, stomach, or top of the small intestine — that is the definition of upper GI bleed. Coffee grounds mean the blood sat in the stomach long enough for stomach acid to turn it brown and grainy. Bright red means the bleeding is fast and fresh.
- Black tarry stool (Melena) (black, sticky, tar-like stool with a strong smell): Black tarry stool means blood traveled through the whole stomach and intestines. It takes at least 8 hours for blood to turn black as bacteria break it down. This points to upper GI bleed. If stool is bright red and bleeding is very fast, blood rushed through before it had time to turn black.

- Stigmata of chronic liver disease: spider angiomas (small red spider-shaped blood vessels on chest or face), fluid buildup in the belly (ascites) (swollen belly from fluid), caput medusae (bulging veins around the belly button), yellowing of the skin and eyes (jaundice) (yellow skin), or palmar redness (erythema) (red palms): These signs tell you the liver is scarred (cirrhosis). A scarred liver blocks blood flow through the liver, so pressure builds up in veins that drain the esophagus and stomach. Those veins swell into varices (bulging weak veins) and can rupture, causing life-threatening upper GI bleed.
- Low blood pressure (Hypotension) (systolic blood pressure < 90 mmHg or mean arterial pressure < 65 mmHg) that comes AFTER a fast heart rate (tachycardia): The body ran out of tricks to keep blood pressure normal. This means a lot of blood is already gone — often more than 30% of total blood volume. The body is heading toward shock. This is a red flag that the upper GI bleed is severe and the patient can crash fast.
- Altered mental status (confusion, sleepiness, slow to respond): The brain is not getting enough oxygen because not enough blood is left to carry it. This means the upper GI bleed has caused significant blood loss and the body is in shock. The brain is one of the first places you see trouble.

Diagnostics · every test answers a question

- Complete blood count (CBC) with hemoglobin and hematocrit → How much blood has the body lost, and is it still losing blood?
- Blood urea nitrogen (BUN) and creatinine ratio → Is this blood loss happening in the stomach or gut?
- Prothrombin time (PT/INR) and partial thromboplastin time (PTT) → Is the blood able to clot normally, or is something making it too thin?
- Lactate level → Is the body in shock from too little oxygen reaching the tissues?
- Type and crossmatch (or type and screen) → What is the patient's blood type, and is blood ready if we need to transfuse?
- Liver function tests (ALT, AST, bilirubin, albumin) → Is the liver damaged or scarred, pointing to varices as the cause?
- Upper endoscopy (esophagogastroduodenoscopy, EGD) → Where exactly is the bleeding coming from, and can we stop it right now?
- Platelet count → Does the body have enough platelets (the sticky cells that plug bleeding holes) to form a clot?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.
- Nasogastric (NG) tube placement is NOT routine and should only be used if the diagnosis is unclear or to clear the stomach before endoscopy → Can confirm upper GI bleed if you see blood or coffee grounds come up the tube. Can suction out blood clots so the endoscopy camera has a clear view.

- Interventional radiology (angiography with embolization) or surgery if endoscopy fails to stop the bleeding or bleeding restarts despite two tries → Radiology threads a catheter into the bleeding artery and plugs it with coils or glue. Surgery can tie off the vessel (ligation) or remove part of the stomach. These are rescue options when the camera approach does not work.

Medications

Crystalloid IV fluids (normal saline or lactated Ringer's) · Volume expander

Replaces lost blood volume quickly to keep blood pressure stable and organs fed. These are salt-water solutions that go straight into the veins through two thick IV tubes (large-bore) so a lot of fluid can move fast. The goal is balanced resuscitation: give enough to keep the body running but not so much that you trigger more bleeding or overload the lungs with fluid.

1. Blood is lost from the upper GI tract (esophagus, stomach, or duodenum), so circulating volume drops.
2. Less volume means lower blood pressure and less oxygen delivery to vital organs.
3. Crystalloid fluids are infused rapidly through large-bore IVs to restore volume and stabilize blood pressure.
4. This buys time until the bleeding source is found and stopped, and until blood transfusion can be given if needed.

SAFETY WATCH

Too much fluid too fast can overload the lungs and cause pulmonary edema (fluid flooding the lungs) (fluid in the lungs, making breathing hard). · In variceal bleeding (bleeding from swollen veins in the esophagus due to liver disease), permissive low blood pressure (hypotension) is safer: keep systolic blood pressure around 90–100 instead of pushing it higher, because higher pressure can pop the varices open wider. · Monitor urine output, breathing, and blood pressure closely during resuscitation.

Packed red blood cells (pRBCs) · Blood product transfusion

Restores the oxygen-carrying capacity of the blood by replacing lost red blood cells. A restrictive transfusion strategy is used: transfuse to a hemoglobin target of 7 g/dL in stable patients, or 8 g/dL if the patient has blood-starved (ischemic) heart disease (not enough blood feeding the heart muscle) or other serious heart or lung problems. Over-transfusing can

increase portal pressure (pressure in the liver's blood vessels) in variceal bleeding and worsen outcomes.

1. Bleeding depletes red blood cells, so hemoglobin (the oxygen-carrying protein inside red cells) drops.
2. Low hemoglobin means less oxygen reaches tissues, causing fatigue, confusion, chest pain, or organ damage.
3. Packed red blood cells are transfused to raise hemoglobin to the safe target (7 or 8 g/dL).
4. This restores oxygen delivery and prevents blood-starved (ischemic) injury while the bleeding is controlled.

SAFETY WATCH

Transfusion reactions (fever, chills, allergic reactions; rare but serious). · Transfusion-related acute lung injury (TRALI): sudden breathing trouble within 6 hours of transfusion. · Over-transfusion in variceal bleeding can raise portal pressure and trigger re-bleeding. · Always confirm correct blood type and cross-match before transfusion.

Proton pump inhibitor (PPI): IV pantoprazole or esomeprazole · Proton pump inhibitor

Blocks the stomach's acid-making pumps (the proton pumps on the cells lining the stomach) so the stomach becomes less acidic. A clot forming over a bleeding ulcer is fragile; acid can dissolve it and restart bleeding. By raising the stomach's pH above 6, PPIs protect the clot and help it seal the ulcer. Given as an IV bolus (fast push) followed by a continuous infusion (slow drip) for 72 hours in high-risk ulcer bleeding, then switched to oral PPI once the patient can swallow.

1. Parietal cells in the stomach normally pump hydrogen ions (H^+) into the stomach to make hydrochloric acid.
2. The PPI enters the parietal cell and permanently blocks the proton pump.
3. Acid production drops sharply, raising the stomach pH above 6.
4. At higher pH, pepsin (a digestive enzyme activated by acid) is inactive and clots are stable, so re-bleeding risk falls and healing can start.
5. The effect lasts 24–48 hours per dose because the pump is permanently blocked; new pumps must be made by the cell.

SAFETY WATCH

Long-term PPI use (months to years) is linked to low magnesium, low vitamin B12, bone fractures, and rarely kidney disease—but short-term use (days to weeks) in acute bleeding is safe and guideline-recommended. · May slightly increase risk of *Clostridioides difficile* (C. diff) infection in the colon. · Interacts with clopidogrel (Plavix): PPIs can reduce clopidogrel's anti-clotting effect, but current guidelines say the benefit of PPI in GI bleed outweighs this risk.

Octreotide · Somatostatin analog (synthetic copy of a natural hormone)

Mimics somatostatin, a natural hormone that constricts (tightens) blood vessels in the gut and lowers portal pressure (the pressure in the veins draining the intestines and spleen into the liver). High portal pressure makes varices (swollen, fragile veins in the esophagus) more likely to bleed. Octreotide is used when varices are suspected or confirmed, started as soon as possible and continued for 2–5 days alongside antibiotics and endoscopy.

1. Liver cirrhosis (scarring) blocks blood flow through the liver, so pressure builds up in the portal vein.
2. High portal pressure forces blood backward into fragile veins in the esophagus (varices), which can rupture and bleed massively.
3. Octreotide binds to somatostatin receptors on splanchnic arteries, causing them to constrict.
4. Less blood flows into the portal system, so portal pressure drops by 20–30%.
5. Lower pressure reduces the force stretching the varices, helping bleeding stop and preventing re-bleeding until endoscopy can tie them off.

SAFETY WATCH

Nausea, abdominal cramps, and diarrhea are common but usually mild. · Can cause high or low blood sugar; monitor glucose, especially in diabetics. · Rarely causes a slow heart rate (bradycardia) (slow heart rate) or arrhythmias (irregular heartbeat). · Gallstones and sludge can form with long-term use (weeks), but short-term use (days) is generally safe.

Ceftriaxone · Third-generation cephalosporin antibiotic

Kills bacteria that commonly infect patients with cirrhosis and variceal bleeding, especially bacteria that sneak from the gut into the bloodstream (spontaneous bacterial peritonitis and bacteremia). Infection worsens outcomes and increases re-bleeding risk. Ceftriaxone is started immediately when varices are suspected, even before infection is proven, and continued for up

to 7 days. This preventive (prophylactic) (preventive) antibiotic reduces death rates and infection rates in variceal bleeding.

1. Cirrhosis weakens the immune system and makes the gut wall leaky, so bacteria cross into the blood and peritoneal space.
2. Infection triggers inflammation, which raises portal pressure and clotting problems, making bleeding worse.
3. Ceftriaxone enters bacteria and blocks their cell-wall-building enzymes (penicillin-binding proteins).
4. Without a strong wall, bacteria burst and die.
5. Preventing infection keeps the patient stable, lowers re-bleeding risk, and improves survival.

SAFETY WATCH

Allergic reactions, including rash, hives, or anaphylaxis (rare but life-threatening); ask about penicillin or cephalosporin allergy before giving. · Diarrhea and risk of *C. difficile* colitis (severe gut infection) with prolonged use. · Can displace bilirubin from albumin in newborns (never use in neonates with yellowing of the skin and eyes (jaundice)), but safe in adults. · Do NOT mix with calcium-containing IV solutions (can form precipitates in the line).

Reversal agents for anticoagulants (specific to the drug the patient is taking) · Varies by anticoagulant: vitamin K antagonist reversal, direct oral anticoagulant (DOAC) reversal, heparin reversal

Stops or reverses the blood-thinning effect of anticoagulant medications so clots can form and bleeding can stop. The choice of reversal agent depends on which anticoagulant the patient is on and how severe the bleeding is. Examples: (1) Warfarin: give IV vitamin K and four-factor prothrombin complex concentrate (4F-PCC) or fresh frozen plasma (FFP). (2) Dabigatran (Pradaxa): give idarucizumab (Praxbind), a monoclonal antibody that binds and inactivates dabigatran. (3) Rivaroxaban, apixaban, edoxaban (Xa inhibitors): give andexanet alfa (Andexxa) or 4F-PCC. (4) Heparin: stop the infusion; if urgent, give protamine sulfate. The goal is NOT to make the blood hypercoagulable, only to normalize clotting enough to stop life-threatening bleeding.

1. Anticoagulants block one or more steps in the clotting cascade, so clots form slowly or not at all.
2. In major bleeding, this delay is dangerous—blood loss continues unchecked.

3. The reversal agent either replaces the missing clotting factors (PCC, FFP), inactivates the anticoagulant drug (idarucizumab, andexanet alfa), or supplies the vitamin (vitamin K) needed to make new factors.
4. Clotting function is restored within minutes to hours, allowing a stable clot to form at the bleeding site.
5. Once bleeding is controlled, the team decides whether to restart anticoagulation (depending on stroke, clot, or valve risk).

SAFETY WATCH

Reversal increases clotting risk: stroke, heart attack, a clot in a deep leg vein (deep vein thrombosis), or pulmonary embolism (a clot lodging in a lung artery) can occur, especially in patients with mechanical heart valves, atrial fibrillation (an irregular, quivering heartbeat), or recent clots. · Prothrombin complex concentrates and andexanet alfa carry thrombotic risk; use the lowest effective dose. · Protamine (for heparin) can cause severe allergic reactions, low blood pressure, and a slow heart rate (bradycardia); give slowly. · Vitamin K takes 12–24 hours to work, so it must be combined with PCC or FFP for urgent reversal. · Always coordinate reversal with hematology, cardiology, or neurology if the patient has high clot risk.

Platelet transfusion (if platelets < 50,000/μL and active bleeding) · Blood product transfusion

Replaces platelets, the tiny cell fragments that plug holes in blood vessels and help clots form. If the platelet count is very low (thrombocytopenia), clots are weak or don't form at all, and bleeding continues. Transfusing platelets raises the count temporarily so endoscopy can be performed safely and bleeding can be controlled. Guideline targets vary, but generally platelets are transfused to keep the count above 50,000/μL during active bleeding or before high-risk procedures.

1. Bleeding or certain diseases (liver disease, bone marrow failure, immune destruction) lower the platelet count.
2. Without enough platelets, the first step of clotting—plugging the hole—fails, so bleeding continues.
3. Donor platelets are transfused, raising the count within minutes to hours.
4. The new platelets migrate to the bleeding site, stick together, and form a temporary plug while the clotting cascade builds a fibrin mesh over it.

SAFETY WATCH

Transfusion reactions (fever, chills, allergic rash; rarely severe). · Transfusion-related acute lung injury (TRALI). · Platelet refractoriness: if the patient has antibodies against donor platelets, the count may not rise; HLA-matched platelets may be needed. · Risk of bacterial contamination (platelets are stored at room temperature); fever after transfusion should prompt blood cultures.

Fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC) (if INR elevated and active bleeding) · Clotting factor replacement (blood product or plasma-derived concentrate)

Replaces clotting factors (proteins made by the liver that work together in a cascade to form a clot). Patients on warfarin, or those with liver disease, often have low levels of factors II, VII, IX, and X, so their INR (international normalized ratio, a measure of clotting time) is high and clots form slowly. FFP contains all clotting factors; PCC is a concentrated dose of factors II, VII, IX, and X. PCC works faster and with less volume than FFP, so it is preferred in urgent bleeding. Both are given IV to normalize the INR quickly (goal < 1.5 in major bleeding) so a stable clot can form.

1. Warfarin or liver disease depletes vitamin K-dependent clotting factors (II, VII, IX, X).
2. Without these factors, the clotting cascade stalls and the INR climbs (normal is ~1.0; warfarin patients often run 2–3, liver disease can push it higher).
3. High INR means slow or absent clot formation, so bleeding continues.
4. FFP or PCC is transfused, delivering a bolus of clotting factors into the blood.
5. The cascade is restored, fibrin forms, and a stable clot seals the bleeding vessel within minutes to hours.
6. Vitamin K is given alongside (IV or PO) so the liver can start making its own factors again (takes 12–24 hours).

SAFETY WATCH

Volume overload and pulmonary edema (fluid flooding the lungs) with FFP (it takes 4–6 units to reverse warfarin, each unit is 200–250 mL). · Thrombotic risk (stroke, MI, DVT, PE) with PCC—use the lowest effective dose and avoid in patients with active clotting unless bleeding is life-threatening. · Transfusion reactions (fever, allergic reactions, rarely TRALI). · Viral transmission risk is extremely low with modern screening but not zero. · Always give with vitamin K to sustain reversal; factors from FFP/PCC are consumed and degraded within hours.

Red flags

- 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)

Nursing priorities

1. Establish two large-bore IV lines (18-gauge or larger) and start balanced crystalloid fluids.
— 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. — 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. — 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.
4. Keep patient NPO (nothing by mouth) and place on aspiration precautions with head of bed elevated 30 to 45 degrees. — Vomiting blood (Hematemesis) (vomiting blood) increases aspiration risk (breathing stomach contents into lungs) causing chemical pneumonitis (lung inflammation) or ARDS (acute respiratory distress syndrome); NPO prepares patient for urgent endoscopy requiring sedation.
5. Administer IV proton pump inhibitor bolus followed by continuous infusion as ordered. — PPI (proton pump inhibitor) raises stomach pH above 6, which stabilizes clots on bleeding ulcers by preventing pepsin (a stomach enzyme) from dissolving the platelet plug; infusion maintains this effect until endoscopy.
6. Start octreotide infusion and ceftriaxone if stigmata of liver disease are present or variceal bleed is suspected. — Octreotide reduces splanchnic blood flow (blood going to the intestines), lowering portal pressure (pressure in liver veins) and variceal bleeding; ceftriaxone prevents spontaneous bacterial peritonitis (infection of belly fluid) which worsens outcomes in cirrhosis patients.

7. Maintain hemoglobin goal of 7 to 8 g/dL with packed red blood cells, avoiding over-transfusion. — Restrictive transfusion strategy prevents rebleeding because high blood pressure from over-transfusion dislodges fragile clots; hemoglobin of 7 to 8 g/dL ensures adequate oxygen delivery without increasing portal pressure in variceal bleeds.
8. Prepare for urgent endoscopy and notify gastroenterology immediately if massive bleeding or hemodynamic instability occurs. — Endoscopy within 24 hours allows direct visualization and treatment with clips, bands, or cautery (burning tissue to stop bleeding); massive bleeding needs endoscopy within 12 hours or interventional radiology if endoscopy fails to control the source.

Sources

- ACG Clinical Guideline: Upper Gastrointestinal Bleeding (2021)
- AASLD Practice Guidance: Variceal Hemorrhage (2020)
- AGA Institute Guideline on Management of Acute Lower GI Bleeding (2016)
- Cochrane systematic reviews on peptic ulcer and variceal bleeding

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