Upper GI bleeding

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Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis (vomiting of blood or coffee-ground-like material) and/or melena (black, tarry stools).

The initial evaluation of patients with acute upper GI bleeding involves an assessment of hemodynamic stability and resuscitation if necessary.

Diagnostic studies (usually endoscopy) follow, with the goals of diagnosis, and when possible, treatment of the specific disorder.

The initial evaluation of a patient with a suspected clinically significant acute upper GI bleed includes a history, physical examination, and laboratory tests.

Potential bleeding sources suggested by a patient's past medical history include:

- Varices or portal hypertensive gastropathy
- Angiodysplasia in a patient with renal disease, aortic stenosis, or hereditary hemorrhagic telangiectasia
- Peptic ulcer disease in a patient with a history of *Helicobacter pylori* (*H. pylori*) infection, nonsteroidal anti-inflammatory drug (NSAIDs) use, antithrombotic use, or smoking
- Malignancy
- Marginal ulcers

Comorbid illnesses may influence patient management in the setting of an acute upper GI bleed

- Make patients more susceptible to adverse effects of anemia (eg, coronary artery disease, pulmonary disease). Such patients may need to be maintained at higher hemoglobin levels than patients without these disorders
- Predispose patients to volume overload in the setting of vigorous fluid resuscitation or blood transfusions (eg, renal disease, heart failure). Such patients may need more invasive monitoring during resuscitation.
- Result in bleeding that is more difficult to control (eg, coagulopathies, thrombocytopenia, significant hepatic dysfunction). Such patients may need additional hemostatic therapies.
- Predispose to aspiration of GI contents into the lungs (eg, dementia, hepatic encephalopathy). Endotracheal intubation should be considered in such patients.

Medication history

- Predispose to peptic ulcer formation, such as aspirin and other NSAIDs
- Are associated with pill esophagitis
- Increase risk of bleeding, such as anticoagulants (including warfarin and the direct oral anticoagulants) and antiplatelet agents
- Have been associated with GI bleeding, including selective serotonin reuptake inhibitors (SSRI), calcium channel blockers, and aldosterone antagonists.
- May alter the clinical presentation, such as bismuth, charcoal, licorice, and iron, which can turn the stool black.

Symptom assessment

- Peptic ulcer – Upper abdominal pain
- Esophageal ulcer – Odynophagia, gastroesophageal reflux, dysphagia
- Mallory-Weiss tear – Emesis, retching, or coughing prior to hematemesis
- Variceal hemorrhage or portal hypertensive gastropathy: Jaundice, abdominal distention (ascites)
- Malignancy – Dysphagia, early satiety, involuntary weight loss, cachexia

Physical examination

- Mild to moderate hypovolemia (less than 15 percent of blood volume lost) – Resting tachycardia.

- Blood volume loss of at least 15 percent – Orthostatic hypotension (a decrease in the systolic blood pressure of more than 20 mmHg and/or an increase in heart rate of 20 beats per minute when moving from recumbency to standing).

- Blood volume loss of at least 40 percent – Supine hypotension.

The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding, raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy.

Laboratory data

Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies.

In addition, serial electrocardiograms and cardiac enzymes may be indicated in patients who are at risk for a myocardial infarction, such as older adults, patients with a history of coronary artery disease, or patients with symptoms such as chest pain or dyspnea.

The initial hemoglobin level in patients with acute upper GI bleeding may be at the patient's baseline because the patient is losing whole blood.

The hemoglobin level should initially be monitored every two to eight hours, depending upon the severity of the bleed.

Because blood is absorbed as it passes through the small bowel and patients may have decreased renal perfusion, patients with acute upper GI bleeding typically have an elevated blood urea nitrogen (BUN)-to-creatinine or urea-to-creatinine ratio. Values >30:1 or >100:1, respectively, suggest upper GI bleeding. The higher the ratio, the more likely the bleeding is from an upper GI source.

NGT lavage may be used when it is unclear if a patient has ongoing bleeding and thus might benefit from an early endoscopy.

In addition, NGT lavage can be used to remove particulate matter, fresh blood, and clots from the stomach to facilitate endoscopy.

An alternative to NGT lavage in this situation is to use a prokinetic such as erythromycin.

GENERAL MANAGEMENT
Hemodynamically unstable patients

Intravenous access
Fluid resuscitation
Transfusion

- For patients with active/brisk bleeding and hypovolemia, transfusion should be guided by hemodynamic parameters (eg, pulse and blood pressure), the pace of the bleeding, estimated blood loss, and the ability to stop the bleeding, rather than by serial hemoglobin measurements. If the initial hemoglobin level is low (<7 g/dL) transfusions should be initiated.
- In an acutely hemorrhaging patient, however, transfusion support should not be delayed while awaiting laboratory test results.
- The approach to medications (eg, proton pump inhibitors) and endoscopy are similar for patients with hemodynamic instability compared with patients who are hemodynamically stable. It is particularly important to ensure that these patients are adequately resuscitated prior to undergoing upper endoscopy.

All patients with hemodynamic instability or active bleeding (manifested by hematemesis, bright red blood per nasogastric tube, or hematochezia) should be admitted to an intensive care unit for resuscitation and close observation with automated blood pressure monitoring, electrocardiographic monitoring, and pulse oximetry.

Elective endotracheal intubation in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration.

Adequate resuscitation and hemodynamic stabilization is essential prior to endoscopy to minimize treatment-associated complications.

Patients with active bleeding should receive intravenous fluids (eg, 500 mL of normal saline or lactated Ringer's solution over 30 minutes) while being typed and crossmatched for blood transfusion.

If the blood pressure fails to respond to initial resuscitation efforts, the rate of fluid administration should be increased. In some patients, temporary support with vasopressor drugs may be required.

for patients at increased risk of adverse events in the setting of significant anemia, such as those with coronary artery disease or in those with evidence of ongoing active bleeding, our goal is to maintain the hemoglobin at a level of ≥8 g/dL.

It is important to avoid overtransfusion in patients with suspected variceal bleeding. In patients with variceal bleeding, we transfuse once the hemoglobin is <7 g/dL, with the goal of increasing the hemoglobin to ≥7 g/dL. We do not use a higher transfusion threshold (eg, <9 g/dL), as transfusion can precipitate worsening of the bleeding.

Patients who require massive transfusion (defined by institutional protocols, often >3 units of RBCs in an hour or 10 units of RBCs in 24 hours) may also need replacement of coagulation factors and/or platelets.

Patients with critical or life-threatening bleeding and a low platelet count (<50,000/microL) should be transfused with platelets

Limited data suggest that proceeding with upper endoscopy in patients with thrombocytopenia is generally safe.

perform an upper endoscopy if the platelet count is >20,000/microL, though if the patient is suspected to have active bleeding, we attempt to raise the platelet count to >50,000/microL prior to endoscopy.

In the past, platelet transfusions were considered in non-thrombocytopenic or mildly thrombocytopenic patients with life-threatening bleeding who had been taking antiplatelet agents such as aspirin or clopidogrel .
However, high-quality evidence regarding the benefit of platelet transfusion is lacking, and some evidence suggests that platelet transfusion may be deleterious.

For most patients, endoscopy should not be delayed because of anticoagulant or antiplatelet agent use.

Typically, we try to perform endoscopy on patients with suspected ongoing active bleeding after resuscitation within 12 hours.

Medications

Acid suppression

Giving an IV PPI every 12 hours or starting a continuous infusion

Oral formulations (eg, esomeprazole 40 mg orally twice daily) are a reasonable alternative if IV formulations are not available.

Oral and intravenous PPI therapy also decrease the length of hospital stay, rebleeding rate, and need for blood transfusion in patients with high-risk ulcers treated with endoscopic therapy.

PPIs may also promote hemostasis in patients with lesions other than ulcers. This likely occurs because neutralization of gastric acid leads to the stabilization of blood clots

Prokinetics

Both erythromycin and metoclopramide have been studied in patients with acute upper GI bleeding. The goal of using a prokinetic agent is to improve gastric visualization at the time of endoscopy by clearing the stomach of blood, clots, and food residue

Vasoactive medications

Somatostatin, its analog octreotide, is used in the treatment of variceal bleeding and may also reduce the risk of bleeding due to nonvariceal causes. In patients with suspected variceal bleeding, octreotide is given as an intravenous bolus of 50 mcg, followed by a continuous infusion at a rate of 50 mcg per hour.

Octreotide is not recommended for routine use in patients with acute nonvariceal upper GI bleeding, but it can be used as adjunctive therapy in some cases.

Antibiotics for patients with cirrhosis

Bacterial infections are present in up to 20 percent of patients with cirrhosis who are hospitalized with gastrointestinal bleeding

Antibiotics may also reduce the risk of recurrent bleeding in hospitalized patients who bled from esophageal varices.

The randomized trial and meta-analysis suggest that there is no role for tranexamic acid in the treatment of upper GI bleeding.

The decision to obtain surgical and interventional radiology consultations prior to endoscopy should be based upon the likelihood of persistent or recurrent bleeding, or risks/complications stemming from endoscopic therapy (perforation, precipitation of massive bleeding).

Early endoscopy (within 24 hours) is recommended for most patients with acute upper GI bleeding.
For patients with suspected variceal bleeding, we perform endoscopy within 12 hours of presentation.

Findings include spurting hemorrhage (class Ia), oozing hemorrhage (class Ib), a nonbleeding visible vessel (class IIa) , an adherent clot (class IIb) ,a flat pigmented spot (class IIc) , and a clean ulcer base (class III)

In patients in whom blood obscures the source of bleeding, a second endoscopy may be required to establish a diagnosis and to potentially apply therapy, but routine second-look endoscopy is not recommended.

that patients do not need to have a normal hematocrit in order to safely undergo endoscopy. In addition, endoscopy appears to be safe in patients who are mildly to moderately anticoagulated.

Other diagnostic tests for acute upper GI bleeding include CT angiography and angiography, which can detect active bleeding , deep small bowel enteroscopy, and rarely, intraoperative enteroscopy .

Upper GI barium studies are **contraindicated** in the setting of acute upper GI bleeding because they will interfere with subsequent endoscopy, angiography, or surgery.

Factors associated with rebleeding identified in a meta-analysis included:

- Hemodynamic instability (systolic blood pressure less than 100 mmHg, heart rate greater than 100 beats per minute)
- Hemoglobin less than 10 g/L
- Active bleeding at the time of endoscopy
- Large ulcer size (greater than 1 to 3 cm in various studies)
- Ulcer location (posterior duodenal bulb or high lesser gastric curvature)

An increase in the blood urea nitrogen (BUN) level at 24 hours compared with baseline may be another predictor of poor outcomes.

Two commonly cited scoring systems are the Rockall score and the Blatchford score

The Rockall score which is calculated after endoscopy is based upon age, the presence of shock, comorbidity, diagnosis, and endoscopic stigmata of recent Hemorrhage

Glasgow Blatchford score (GBS) is based upon the blood urea nitrogen, hemoglobin, systolic blood pressure, pulse, and the presence of melena, syncope, hepatic disease, and/or cardiac failure.

AIMS65 is another scoring system that uses data available prior to endoscopy (serum albumin, INR, presence of altered mental status, systolic blood pressure, and age)

As a general rule, we discharge patients following endoscopy if they have a likely bleeding source identified on upper endoscopy that is not associated with a high risk of rebleeding provided they:

- have no comorbidities
- Have stable vital signs
- Have a normal hemoglobin level

High-risk bleeding sources include variceal bleeding, active bleeding, bleeding from a Dieulafoy's lesion, or an ulcer bleeding with high-risk stigmata

Most patients who have received endoscopic treatment for high-risk stigmata should be hospitalized for 72 hours to monitor for rebleeding, since most rebleeding occurs during this time.

Upper GI bleeding in adults: Rapid overview of emergency management

Major causes*
Peptic ulcer, esophagogastric varices, arteriovenous malformation, tumor, esophageal (Mallory-Weiss) tear
Clinical features
History
Use of: NSAIDs, aspirin, anticoagulants, antiplatelet agents
Alcohol abuse, previous GI bleed, liver disease, coagulopathy
Symptoms and signs: Abdominal pain, hematemesis or "coffee ground" emesis, passing melena/tarry stool (stool may be frankly bloody or maroon with massive or brisk upper GI bleeding)
Examination
Tachycardia; orthostatic blood pressure changes suggest moderate to severe blood loss; hypotension suggests life-threatening blood loss (hypotension may be late finding in healthy younger adult)
Rectal examination is performed to assess stool color (melena versus hematochezia versus brown)
Significant abdominal tenderness accompanied by signs of peritoneal irritation (eg, involuntary guarding) suggests perforation
Diagnostic testing
Obtain type and crossmatch for hemodynamic instability, severe bleeding, or high-risk patient; obtain type and screen for hemodynamically stable patient without signs of severe bleeding
Obtain hemoglobin concentration (note that measurement may be inaccurate with acute severe hemorrhage), platelet count, coagulation studies (prothrombin time with INR), liver enzymes (AST, ALT), albumin, BUN, and creatinine
Nasogastric lavage may be helpful if the source of bleeding is unclear (upper or lower GI tract) or to clean the stomach prior to endoscopy

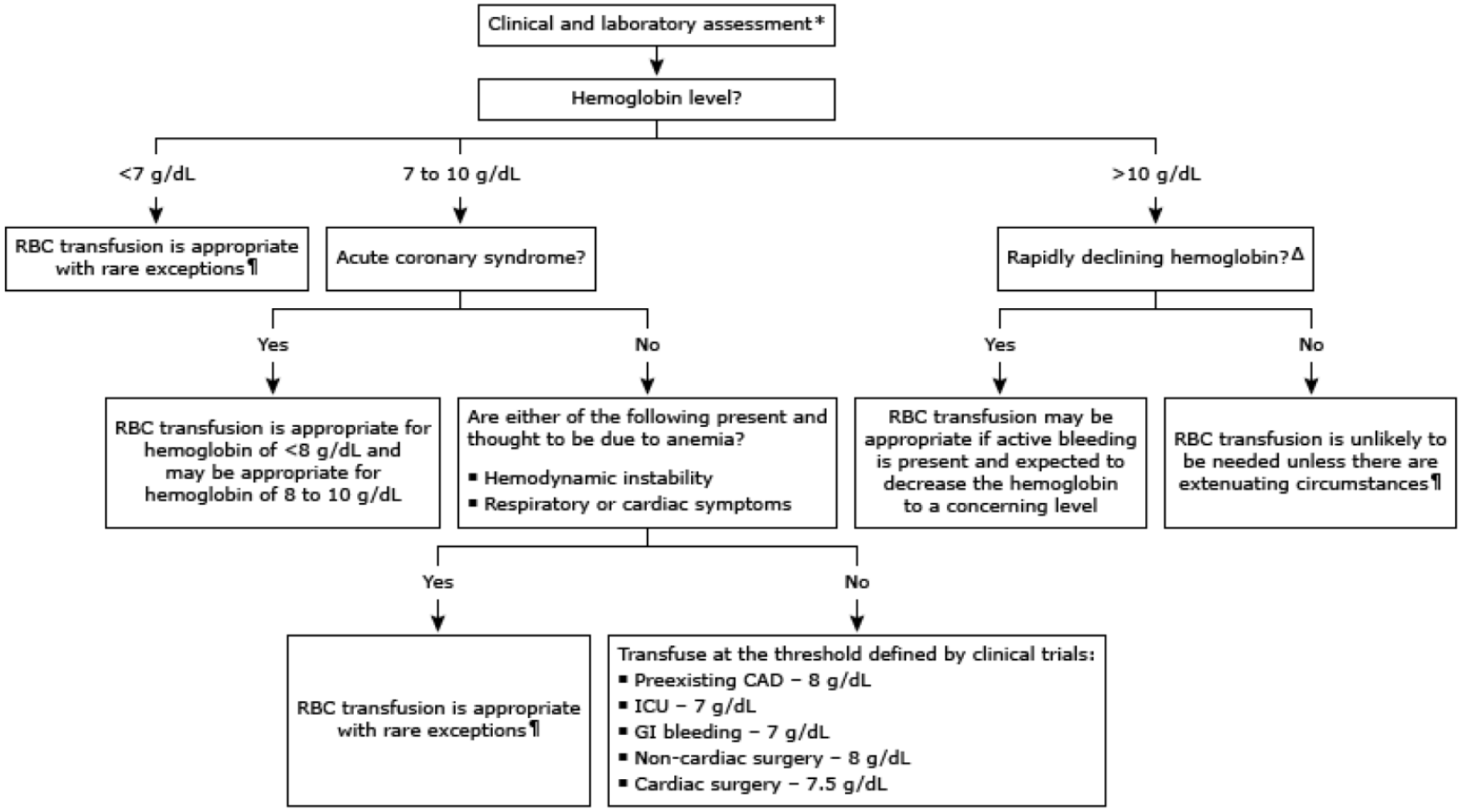
Treatment
Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place)
Do **NOT** give patient anything by mouth
Establish two large bore IV lines (16 gauge or larger)
Provide supplemental oxygen (goal oxygen saturation ≥94% for patients without COPD)
Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid (eg, 500 to 1000 mL per bolus; use smaller boluses and lower total volumes for patients with compromised cardiac function)
Transfusion:
For severe, ongoing bleeding, immediately transfuse blood products in 1:1:1 ration of RBCs, plasma, and platelets, as for trauma patients
For hemodynamic instability despite crystalloid resuscitation, transfuse 1 to 2 units RBCs
For hemoglobin <8 g/dL (80 g/L) in high-risk patients (eg, older adult, coronary artery disease), transfuse 1 unit RBCs and reassess the patient's clinical condition
For hemoglobin <7 g/dL (70 g/L) in low-risk patients, transfuse 1 units RBCs and reassess the patient's clinical condition
Avoid over-transfusion with possible variceal bleeding
Give plasma for coagulopathy or after transfusing four units of RBCs; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin therapy) or after transfusing four units of RBCs

Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding ¶
Pharmacotherapy for all patients with suspected or known severe bleeding:
Give a proton pump inhibitor:
Evidence of active bleeding (eg, hematemesis, hemodynamic instability), give esomeprazole or pantoprazole, 80 mg IV
No evidence of active bleeding, give esomeprazole or pantoprazole, 40 mg IV
Endoscopy delayed beyond 12 hours, give second dose of esomeprazole or pantoprazole, 40 mg IV
Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:
Give somatostatin or an analogue (eg, octreotide 50 mcg IV bolus followed by 50 mcg/hour continuous IV infusion)
Give an IV antibiotic (eg, ceftriaxone or fluoroquinolone)
Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

Thresholds for red blood cell transfusion in adults

Condition	Hemoglobin threshold for transfusion
Symptomatic patient (eg, myocardial ischemia, hemodynamic instability)	10 g/dL* [1]
Hospitalized patient	
Preexisting coronary artery disease	8 g/dL*
Acute coronary syndromes, including acute MI	8 to 10 g/dL ¶[2]
ICU (hemodynamically stable)	7 g/dL* [3,4]
Gastrointestinal bleeding (hemodynamically stable)	7 g/dL* [5,6]
Orthopedic surgery	8 g/dL* [1]
Cardiac surgery	7.5 g/dL* [7,8]
Ambulatory outpatient	
Oncology patient in treatment	7 to 8 g/dL ¶
Palliative care setting	As needed for symptoms; hospice benefits may vary

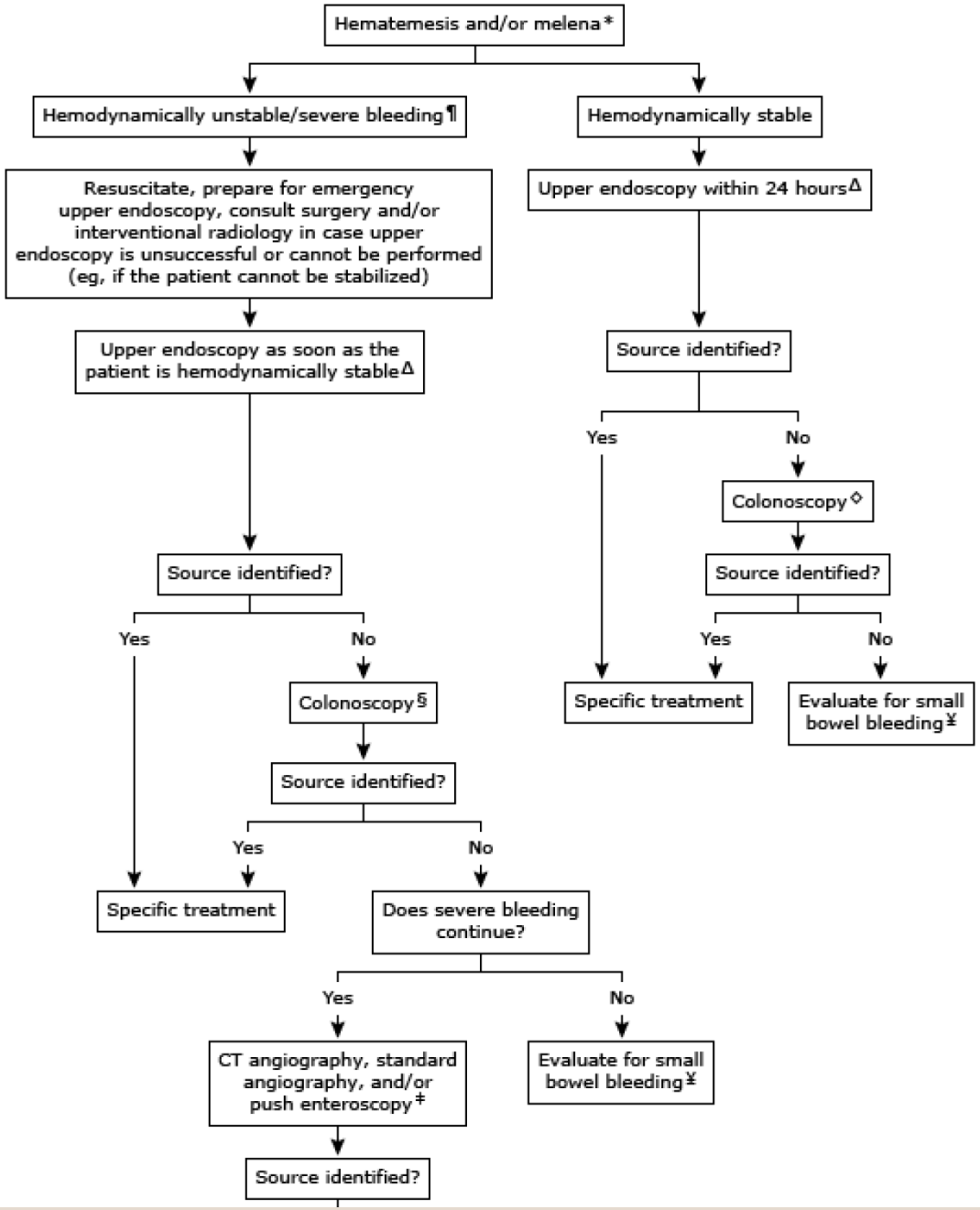
Red blood cell (RBC) transfusion decisions in adults

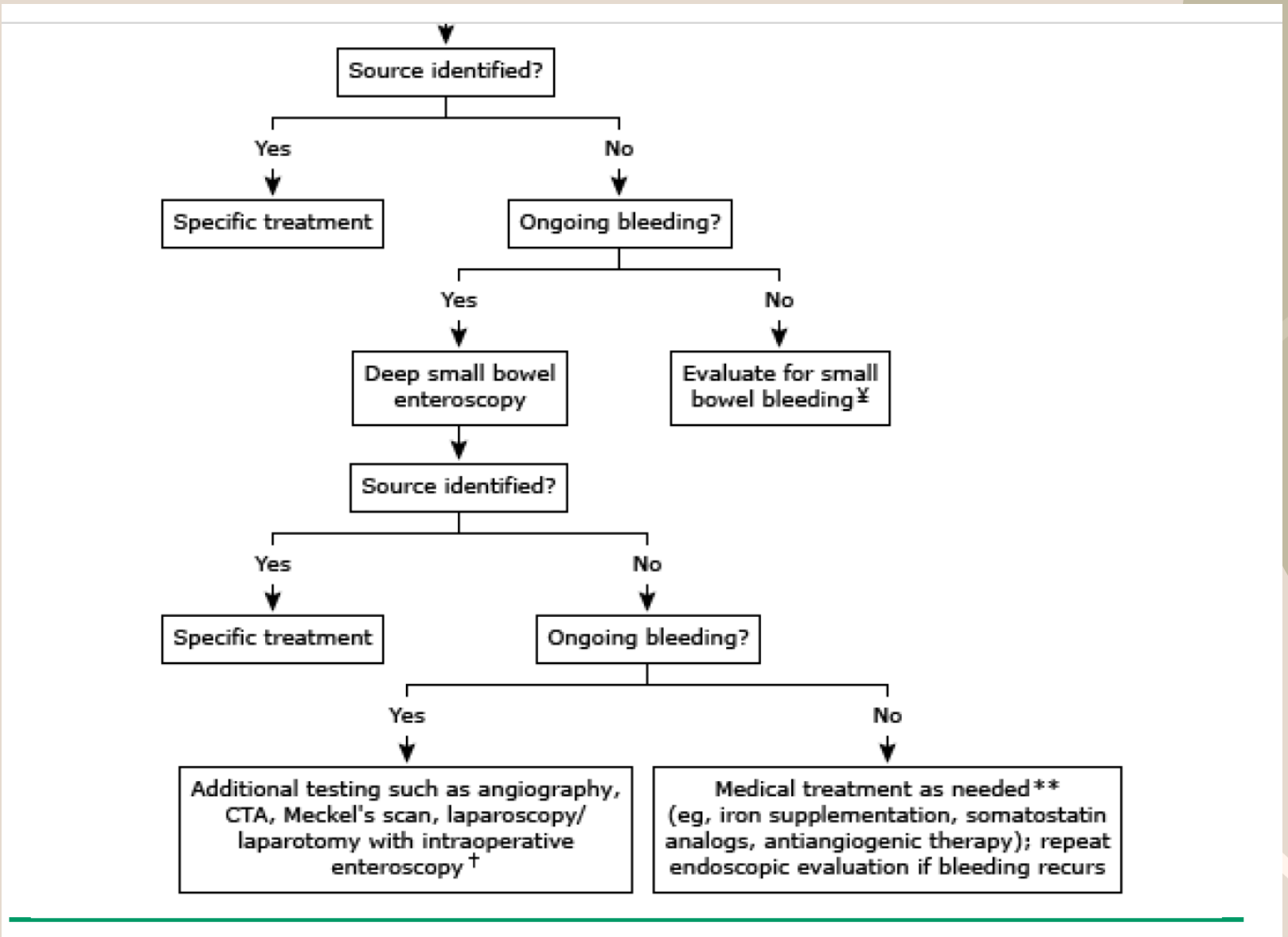


Endoscopic predictors of recurrent peptic ulcer hemorrhage [1,2]

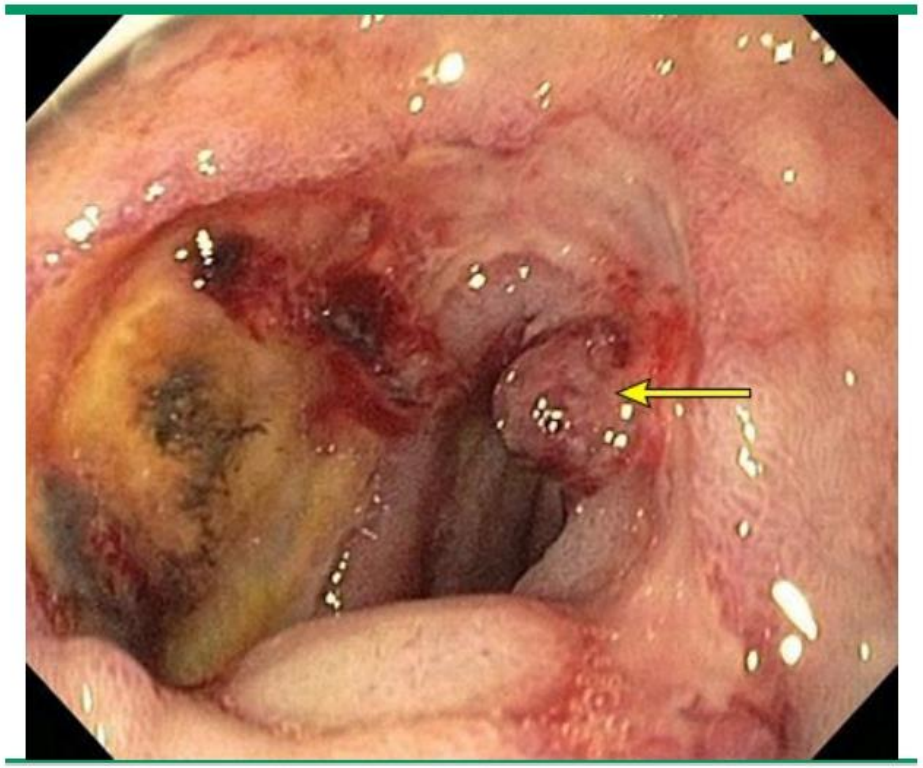
Endoscopic stigmata of recent hemorrhage	Prevalence, percent	Risk of rebleeding on medical management, percent
Active arterial bleeding (Forrest Ia)	12% (arterial bleeding + oozing)	55 (arterial bleeding + oozing)
Oozing without visible vessel (Forrest Ib)		
Non-bleeding visible vessel (Forrest IIa)	8	43
Adherent clot (Forrest IIb)	8	22
Flat spot (Forrest IIc)	16	10
Clean ulcer base (Forrest III)	55	5

Evaluation of suspected upper gastrointestinal bleeding




Source identified?
Yes
No
Specific treatment
Ongoing bleeding?
Yes
No
Deep small bowel enteroscopy
Evaluate for small bowel bleeding¥
Source identified?
Yes
No
Specific treatment
Ongoing bleeding?
Yes
No
Additional testing such as angiography, CTA, Meckel's scan, laparoscopy/ laparotomy with intraoperative enteroscopy†
Medical treatment as needed** (eg, iron supplementation, somatostatin analogs, antiangiogenic therapy); repeat endoscopic evaluation if bleeding recurs

Duodenal ulcer with visible vessel

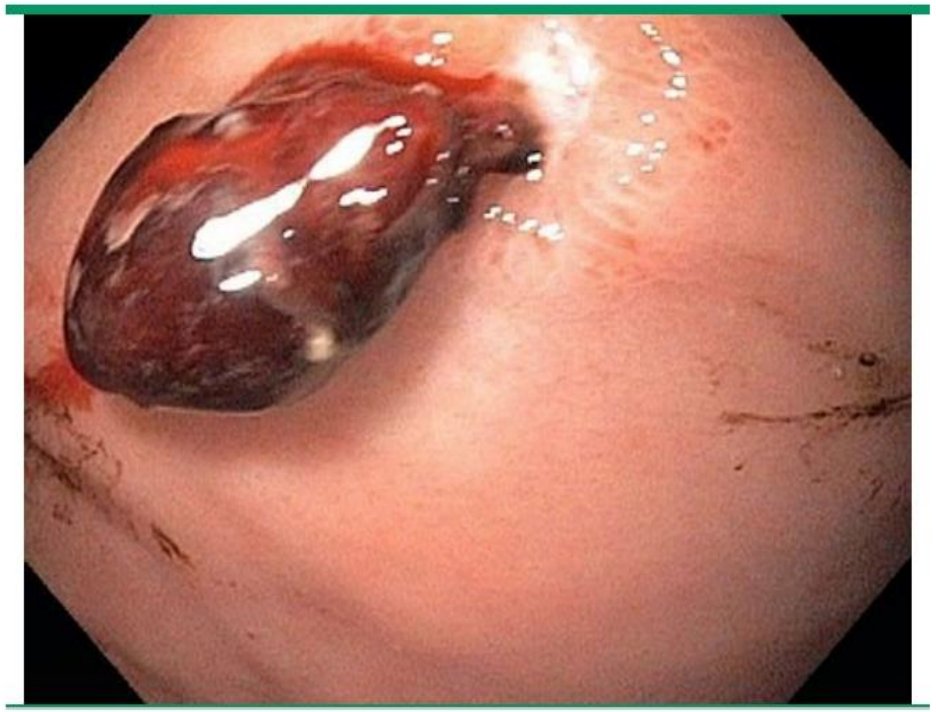


Upper endoscopy showing a duodenal ulcer with a nonbleeding visible vessel (arrow) in a large circumferential ulcer (Forrest classification IIa).

Courtesy of Rome Jutabha.

Graphic 54960 Version 4.0

Gastric ulcer with adherent clot

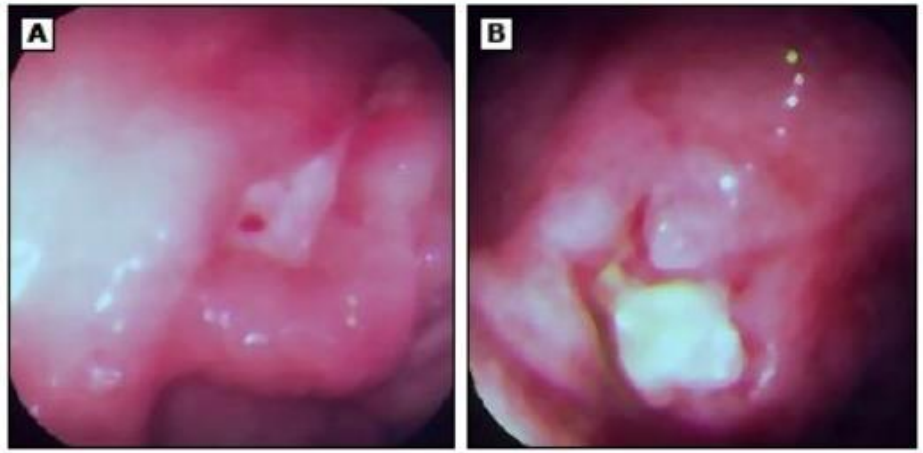


Upper endoscopy showing a gastric ulcer with an adherent clot (Forrest classification IIb).

Courtesy of Rome Jutabha, MD.

Graphic 76246 Version 1.0

Peptic ulcers at low risk for rebleeding



Ulcers with a flat pigmented spot (Forrest classification IIc; panel A) or a clean base (Forrest classification III, panel B) are at low risk for rebleeding and do not need to be treated endoscopically.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 52497 Version 2.0

bleeding peptic ulcers
Forrest classification

- Class Ia – Spurting hemorrhage
- Class Ib – Oozing hemorrhage
- Class IIa – Nonbleeding visible vessel
- Class IIb – Adherent clot
- Class IIc – Flat pigmented spot
- Class III – Clean ulcer base

patients with active bleeding (spurting or oozing), a nonbleeding visible vessel, or an adherent clot are generally considered to be at high risk for recurrent bleeding.

Most patients with high-risk stigmata require endoscopic therapy to decrease the risk of recurrent bleeding.

PPIs given for the treatment of bleeding peptic ulcers are often given as high-dose continuous infusions.

High-dose continuous PPI infusions decrease rebleeding and mortality rates compared with H2RAs or placebo, whereas non-high dose PPIs decrease rebleeding rates compared with H2RAs or placebo (but have not been proven to decrease mortality rates)

While direct comparisons of high-dose continuous PPI infusions with non-high dose PPIs have not shown a difference in the risk of rebleeding or mortality, the International Consensus Group guideline favors high-dose continuous PPI infusions in patients with bleeding ulcers with high-risk stigmata who have undergone successful endoscopic therapy, since indirect comparisons suggest that high-dose continuous PPI infusions may be superior to non-high dose PPIs when it comes to mortality.

Somatostatin and its long-acting analogue octreotide (commonly used in the management of variceal bleeding) have a theoretical benefit in bleeding ulcer disease because they reduce splanchnic blood flow, inhibit gastric acid secretion, and may have gastric cytoprotective effects.

Second-look endoscopy refers to the practice of performing a planned follow-up endoscopy, generally within 24 hours of the initial endoscopy. If there is active bleeding or a nonbleeding visible vessel, endoscopic therapy is performed

In addition to failure of endoscopic therapy, other indications for surgery for peptic ulcer hemorrhage include:

- Hemodynamic instability despite vigorous resuscitation (more than a three unit transfusion)
- Shock associated with recurrent hemorrhage
- Perforation

Secondary or relative indications include rare blood type, difficult crossmatch, refusal of transfusion, shock on presentation, advanced age, severe comorbid disease, and chronic gastric ulcer as the origin of hemorrhage.

In addition, surgery may be appropriate for older adult patients who are unlikely to tolerate prolonged attempts at resuscitation, large volume transfusions, or periods of hypotension

Angiography with transarterial embolization (TAE) for persistent or recurrent peptic ulcer bleeding is a less invasive alternative to surgery.

Patients with lesions that are low risk for recurrent bleeding (eg, an ulcer with clean base or flat pigmented spot) may resume oral intake with a normal diet within 24 hours.

All patients with bleeding peptic ulcers need to be evaluated for factors that predispose to ulcer formation (eg, *H. pylori*) and treated as appropriate.

Acute management of severe upper gastrointestinal bleeding

1. Resuscitation and stabilization, initiation of medical therapy with an intravenous proton pump inhibitor
2. Assessment of onset and severity of bleeding
3. Risk stratification using validated prognostic scale
4. Diagnostic endoscopy
    - Preparation for emergent upper endoscopy
    - Localization and identification of the bleeding site
    - Identification of stigmata of recent hemorrhage
    - Stratification of the risk for rebleeding
5. Therapeutic endoscopy
    - Control of active bleeding or high-risk lesions
    - Minimization of treatment-related complications
    - Treatment of persistent or recurrent bleeding

Disorders that cause upper GI bleeding in adults

Cause	Bleeding manifestations	Associated signs and symptoms	Associated conditions or risk factors	Endoscopic findings*
Ulcerative or erosive				
Duodenal and/or gastric ulcer	Hematemesis Melena Hematochezia (indicates brisk bleeding) Occult blood loss	Upper abdominal pain Pain associated with eating (worse when eating suggests gastric ulcer, improvement with eating suggests duodenal ulcer) Dyspepsia¶	Infections: ▪ *Helicobacter pylori* ▪ CMV ▪ HSV NSAIDs Stress ulcer (eg, in patients who are critically ill) Excess gastric acid production (ZES) Idiopathic	Ulcer with smooth, regular, rounded edges; ulcer base often filled with exudate Examination of the ulcer may reveal: ▪ Active bleeding or oozing ▪ Nonbleeding visible vessel ▪ Adherent clot ▪ Flat pigmented spot ▪ Clean ulcer base

Esophagitis	Hematemesis Melena Occult blood loss	Dysphagia/odynophagia Retrosternal pain Food impaction	Gastroesophageal reflux disease Medications that may cause "pill esophagitis": ▪ Erythromycin ▪ Tetracycline ▪ Doxycycline ▪ Clindamycin ▪ Trimethoprim-sulfamethoxazole ▪ NSAIDs ▪ Oral bisphosphonates ▪ Potassium chloride ▪ Quinidine ▪ Iron supplements Infections: ▪ HSV ▪ CMV ▪ *Candida albicans* ▪ HIV	Erythema, mucosal breaks/erosions, exudative lesions, superficial or deep ulcers, stenosis, scarring Peptic esophagitis: The ulcerations are usually irregularly shaped or linear, multiple, and distal; may be accompanied by Barrett's esophagus Pill-induced: Ulcerations are usually singular and deep, occurring at points of stasis (especially near the carina), with sparing of the distal esophagus Infectious esophagitis: ▪ HSV – Discrete, superficial ulcers, with well-demarcated borders that tend to involve the upper or mid-esophagus; vesicles may be seen ▪ CMV – Ulcers range from small and shallow to large (>1 cm) and deep; most patients have multiple lesions ▪ *Candida* – Diffuse white plaques ▪ HIV – Tends to involve the mid to distal esophagus, ulcers may be shallow or deep, and may be large

Gastritis/gastropathy Duodenitis/duodenopathy	Occult blood loss Hematemesis Melena	Dyspepsia ¶	Risk factors: ▪ *H. pylori* ▪ NSAIDs ▪ Excessive alcohol consumption ▪ Radiation injury ▪ Physiologic stress ▪ Weight loss surgery ▪ Bile reflux Risk factors for bleeding: ▪ Anticoagulant use	Erythematous mucosa Superficial erosions Nodularity Diffuse oozing

Complications of portal hypertension				
Esophagogastric varices	Hematemesis Melena Hematochezia (indicates brisk bleeding)	Stigmata of chronic liver disease[Δ], in particular, signs of portal hypertension (splenomegaly, ascites, thrombocytopenia)	Portal hypertension from: ▪ Cirrhosis ▪ Portal vein thrombosis ▪ Noncirrhotic portal hypertension	Vascular structures that protrude into the esophageal and/or gastric lumen Findings associated with an increased risk of hemorrhage: ▪ Longitudinal red streaks on the varices (red wale marks) ▪ Cherry-colored spots that are flat and overlie varices ▪ Raised, discrete red spots (hematocystic spots) Esophageal varices: ▪ F1: Small, straight varices ▪ F2: Enlarged, tortuous varices that occupy less than one-third of the lumen ▪ F3: Large, coil-shaped varices that occupy more than one-third of the lumen Gastric varices:
				▪ GOV1: Gastroesophageal varices along the lesser curvature of the stomach ▪ GOV2: Gastroesophageal varices along the greater curvature of the stomach ▪ IGV1: Isolated gastric varices in the fundus ▪ IGV2: Isolated gastric varices at other loci in the stomach

Ectopic varices	Hematemesis Melena Hematochezia (indicates brisk bleeding)	Stigmata of chronic liver disease[Δ], in particular, signs of portal hypertension (splenomegaly, ascites, thrombocytopenia)	Portal hypertension from: ▪ Cirrhosis ▪ Portal vein thrombosis ▪ Noncirrhotic portal hypertension	Vascular structures that protrude into areas of the gastrointestinal tract lumen other than the esophagus or stomach (eg, small bowel, rectum)
Portal hypertensive gastropathy	Occult blood loss Hematemesis Melena Hematochezia (indicates brisk bleeding)	Stigmata of chronic liver disease[Δ], in particular, signs of portal hypertension (splenomegaly, ascites, thrombocytopenia)	Portal hypertension from: ▪ Cirrhosis ▪ Portal vein thrombosis ▪ Noncirrhotic portal hypertension	Mosaic-like pattern that gives the gastric mucosa a "snakeskin" appearance Mucosal changes are usually most evident in the fundus and body; in more severe cases, oozing, bleeding, subepithelial hemorrhages, and increased vascularity similar to angiomas are evident, often involving the gastric fundus, gastric body, and antrum

Vascular lesions				
Angiodysplasia	Hematemesis Melena Hematochezia Occult blood loss May have brisk bleeding	Cutaneous angiodysplasia in patients with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)	End-stage kidney disease Aortic stenosis Left ventricular assist device Hereditary hemorrhagic telangiectasia von Willebrand disease Radiation therapy Idiopathic	Small (5 to 10 mm), flat, cherry-red lesions, often with a fern-like pattern of arborizing, ectatic blood vessels radiating from a central vessel
Dieulafoy's lesion	Hematemesis Melena Hematochezia (indicates brisk bleeding; bleeding is often particularly brisk)		Etiology unknown Bleeding may be associated with NSAIDs, cardiovascular disease, hypertension, chronic kidney disease, diabetes, or alcohol abuse	Usually located in the proximal stomach (within 6 cm of the esophagogastric junction) along the lesser curvature (although can be found anywhere in the GI tract) May have active arterial spurting from the mucosa without an associated ulcer or mass If the bleeding has stopped, there may be a raised nipple or visible vessel without an associated ulcer Endoscopic ultrasound may help confirm the diagnosis
Gastric antral vascular ectasia (GAVE)	Hematemesis Melena Hematochezia (indicates brisk bleeding) Occult blood loss	In patients with cirrhosis, there may be stigmata of chronic liver disease[Δ], in particular, signs of portal hypertension (splenomegaly, ascites, thrombocytopenia)	Idiopathic Cirrhosis with portal hypertension Kidney disease/transplantation Diabetes mellitus Systemic sclerosis (scleroderma) Bone marrow transplantation	Longitudinal rows of flat, reddish stripes radiating from the pylorus into the antrum that resemble the stripes on a watermelon

Blue rubber bleb nevus syndrome (Bean syndrome)	Hematemesis Melena Hematochezia (indicates brisk bleeding) Occult blood loss	Venous malformations and hemangiomas of any organ, including: ▪ Skin ▪ Central nervous system ▪ Liver ▪ Muscles ▪ Lymphatics Intussusception		Blue or purple nodules, round or multilobular; may occur anywhere in the gastrointestinal tract
Traumatic or iatrogenic				
Mallory-Weiss syndrome	Hematemesis following an increase in intra-abdominal pressure Melena Hematochezia (indicates brisk bleeding)	Epigastric pain Back pain	Vomiting/retching (often related to alcohol consumption) Straining at stool or lifting Coughing Seizures Blunt abdominal trauma Hiatal hernia may increase the risk of developing a tear	Tear in the esophagogastric junction Usually singular and longitudinal, but may be multiple Visualization may require retroflexion of the gastroscope in the cardia of the stomach The tear may be covered by an adherent clot
Foreign body ingestion	Hematemesis Melena Hematochezia (indicates brisk bleeding) Occult blood loss	Dysphagia Odynophagia Neck or abdominal pain Choking Hypersalivation	Psychiatric disorders Altered mental status (toxin induced, dementia, etc) Loose dentures	Visualization of the foreign body endoscopically (plain radiographs of the neck, chest, and abdomen may reveal a radiopaque foreign body or signs of perforation)
		Retrosternal fullness		
Post-surgical anastomotic bleeding ("marginal ulcers")	Occult blood loss Hematemesis Melena Hematochezia (indicates brisk bleeding)	Epigastric pain Nausea	Billroth II surgery Gastric bypass surgery NSAID use *H. pylori* infection Smoking	Ulceration/friable mucosa at an anastomotic site

Post-polypectomy/endoscopic resection/endoscopic sphincterotomy	Hematemesis Melena Hematochezia (indicates brisk bleeding)	Past history of instrumentation (may be as long as three weeks prior to presentation)	Large lesions	Bleeding at resection site; ulceration at the site may be seen
Cameron lesions	Occult blood loss Hematemesis Melena Hematochezia (indicates brisk bleeding)		Hiatal hernia Reflux esophagitis	Linear ulcers or erosions on the mucosal folds of a hiatal hernia at the diaphragmatic impression
Aortoenteric fistula	Hematemesis Melena Hematochezia (indicates brisk bleeding) May have a "herald" bleed followed by massive bleeding	Back pain Fever Signs of sepsis Pulsatile abdominal mass Abdominal bruit	Infectious aortitis (syphilis, tuberculosis) Prosthetic aortic graft Atherosclerotic aortic aneurysm Penetrating ulcers Tumor invasion Trauma Radiation injury Foreign body perforation	Endoscopy is important, primarily to exclude other, more common causes of acute upper GI bleeding Endoscopy with an enteroscope or side-viewing duodenoscope may reveal a graft, an ulcer or erosion at the site of an adherent clot, or an extrinsic pulsatile mass in the distal duodenum or esophagus
Tumors				
Upper GI tumors	Hematemesis Melena Hematochezia (indicates brisk bleeding) Occult blood loss	Weight loss Anorexia Nausea/vomiting Early satiety Epigastric pain Dysphagia (for tumors in the esophagus or proximal stomach) Gastric outlet obstruction Palpable mass Paraneoplastic manifestations: ▪ Diffuse seborrheic keratoses ▪ Acanthosis nigricans	Virtually any tumor type may bleed Benign tumors: ▪ Leiomyoma ▪ Lipoma ▪ Polyp (hyperplastic, adenomatous, hamartomatous, inflammatory) Malignant tumors: ▪ Adenocarcinoma ▪ GI stromal tumors ▪ Lymphoma ▪ Kaposi sarcoma ▪ Carcinoid ▪ Melanoma ▪ Metastatic tumors	Ulcerated mass in the esophagus, stomach, or duodenum In gastric malignancies, the folds surrounding the ulcer crater may be nodular, clubbed, fused, or stop short of the ulcer margin; the margins may be overhanging, irregular, or thickened Bleeding lymphoma may appear as an ulcerated mass or polypoid lesion or as a gastric ulcer
		▪ Membranous nephropathy ▪ Coagulopathy		

Miscellaneous				
Hemobilia	Hematemesis Melena Hematochezia (indicates brisk bleeding)	Biliary colic Jaundice (obstructive) Sepsis (biliary)	Past history of liver or biliary tract instrumentation and/or injury, including the following: ▪ Liver biopsy ▪ Cholecystectomy ▪ Endoscopic biliary biopsies or stenting ▪ TIPS placement ▪ Angioembolization ▪ Blunt or penetrating abdominal trauma ▪ Gallstones ▪ Cholecystitis ▪ Hepatic or bile duct tumors ▪ Intrahepatic stents ▪ Hepatic artery aneurysms ▪ Hepatic abscesses	Blood or clot emanating from the ampulla (a side-viewing duodenoscope may be required to visualize the ampulla) If a clot has formed in the bile duct, bleeding may not be appreciated until the clot is removed ERCP may reveal a filling defect in the bile duct
Hemosuccus pancreaticus	Hematemesis Melena Hematochezia (indicates brisk bleeding)	Abdominal pain Past evidence of symptoms/signs of pancreatitis Imaging evidence of pancreatitis (current or in the past) Elevated amylase and lipase (current or in the past)	Chronic pancreatitis Pancreatic pseudocysts Pancreatic tumors Pancreatic pseudoaneurysm Therapeutic endoscopy of the pancreas or pancreatic duct: ▪ Pancreatic stone removal ▪ Pancreatic duct sphincterotomy ▪ Pseudocyst drainage ▪ Pancreatic duct stenting	Blood or clot emanating from the ampulla (a side-viewing duodenoscope may be required to visualize the ampulla) Cross-sectional imaging or angiography is often required to confirm the diagnosis

Thanks for your attention