ACUTE UPPER GASTROINTESTINAL BLEEDING

DIFFERENTIAL DIAGNOSIS

Cause	Associated Signs and Symptoms	Associated Conditions/Risk Factors	Endoscopic Findings
Ulcerative or Erosive			
Duodenal and/or gastric ulcer	- Upper abdominal pain - Pain associated with eating (worse when eating = gastric ulcer, better when eating = duodenal ulcer) - Dyspepsia	- H. pylori infection - CMV infection - HSV infection - NSAIDs - Stress ulcer (e.g., patients who are critically ill) - Excess gastric acid (ZES) - Idiopathic	Examination of the ulcer may reveal: - Active bleeding or oozing - Nonbleeding visible vessel - Adherent clot - Flat pigmented spot - Clean ulcer base
Esophagitis	- Dysphagia/odynophagia - Retrosternal pain - Food impaction	- Gastroesophageal reflux disease - Medications that cause pill esophagitis such as: bisphosphonates, clindamycin, doxycycline, erythromycin, iron supplements, NSAIDs, potassium chloride, quinidine, tetracycline, trimethoprim-sulfamethoxazole - Candida albicans infection - CMV infection - HIV infection - HSV infection	- Erythema, mucosal breaks, exudative lesions, superficial or deep ulcers, stenosis - **Peptic esophagitis:** ulcerations irregularly shaped or linear, multiple, and distal; may be accompanied by Barrett's esophagus - **Pill-induced:** ulcerations singular and deep, occurring at points of stasis (especially near the carina), with sparing of distal esophagus - **HSV** – discrete, superficial ulcers, with well-demarcated borders that tend to involve 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** – involves mid to distal esophagus, ulcers may be shallow or deep; may be large
Gastritis/gastropathy Duodenitis/ duodenopathy	- Dyspepsia	- H. pylori - NSAIDs - Excessive alcohol consumption - Radiation injury - Physiologic stress - Weight loss surgery - Bile reflux - Anticoagulant use	- Erythematous mucosa - Superficial erosions - Nodularity - Diffuse oozing
Complications of Portal Hypertension			
Esophagogastric varices	- Stigmata of chronic liver disease, in particular, signs of portal hypertension (splenomegaly, ascites, thrombocytopenia)	Portal hypertension from: - Cirrhosis - Portal vein thrombosis - Noncirrhotic portal hypertension	Vascular structures that protrude into esophageal and/or gastric lumen Findings associated with 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 may be categorized as small or large Gastric varices - GOV1: gastroesophageal varices along lesser curvature of the stomach - IGV1: isolated gastric varices in fundus - IGV2: isolated gastric varices at other loci in stomach
Ectopic varices			- Vascular structures that protrude into areas of the gastrointestinal tract lumen other than esophagus or stomach (e.g., small bowel, rectum)
Portal hypertensive gastropathy			- Mosaic-like pattern; “snakeskin” appearance of gastric mucosa - Mucosal changes most evident in fundus and body; in more severe cases, increased vascularity (angioma)s are often evident in fundus, body, and antrum
Vascular Lesions			
Angiodysplasia	- 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	- Patient may present with painless bleeding from unidentifiable source, can be brisk or self-limited and prone to recur	- Etiology unknown - Bleeding may be associated with NSAIDs, cardiovascular disease, hypertension, chronic kidney disease, diabetes, or alcohol abuse	- Usually located in proximal stomach along lesser curvature - May have active arterial spurting from the mucosa without associated ulcer or mass - If bleeding stopped, may be raised nipple or visible vessel without associated ulcer - Endoscopic ultrasound may confirm diagnosis
Gastric antral vascular ectasia (GAVE)	- May be associated with cirrhosis/ stigmata of chronic liver disease (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 pylorus into antrum that resemble stripes on watermelon

Cause	Associated Signs and Symptoms	Associated Conditions/Risk Factors	Endoscopic Findings
Vascular Lesions *(continued)*			
Blue rubber bleb nevus syndrome (Bean syndrome)	- Venous malformations and hemangiomas of any organ, including skin, central nervous system, liver, muscles, and lymphatics - Intussusception		- Blue or purple nodules, round or multi-lobular; may occur anywhere in the gastrointestinal tract
Traumatic or Iatrogenic			
Mallory-Weiss syndrome	- 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 area - 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	- Dysphagia - Odynophagia - Neck or abdominal pain - Choking - Hypersalivation - Retrosternal fullness	- 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, such as air in the mediastinum)
Post-surgical anastomotic bleeding ("marginal ulcers")	- 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	- 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		- Hiatal hernia - Reflux esophagitis	- Linear ulcers or erosions on the mucosal folds of a hiatal hernia at the diaphragmatic impression
Aortoenteric fistula	- Back pain - Fevers - Signs of sepsis - Pulsatile abdominal mass - Abdominal bruit	- Prosthetic aortic graft - Infectious aortitis (syphilis, tuberculosis) - 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 stie of an adherent clot, or an extrinsic pulsatile mass in the distal duodenum or esophagus - The diagnostic test of choice is CTA; angiography is usually not helpful
Tumors			
Upper GI tumors	- Weight loss, anorexia, early satiety - Nausea/vomiting - Epigastric pain - Dysphagia - Gastric outlet obstruction - Palpable mass - Paraneoplastic manifestations (diffuse seborrheic keratoses, acanthosis nigricans, membranous nephropathy, coagulopathy)	- Benign tumors (leiomyoma, lipoma, polyp) - 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
Miscellaneous			
Hemobilia	- 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 - Cross-sectional imaging or angiography is often required to confirm the diagnosis
Hemosuccus pancreaticus	- Abdominal pain - Past evidence of symptoms/signs of pancreatitis - Imaging evidence of pancreatitis - Elevated amylase and lipase	- Chronic pancreatitis - Pancreatic pseudocysts - Pancreatic tumors - Pancreatic pseudoaneurysm - Therapeutic endoscopy of the pancreas or pancreatic duct	- 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

HISTORY

Bleeding Manifestations
- Hematemesis
  - *Bright red bloody emesis, moderate to severe upper GI bleed*
- Coffee-ground emesis
  - *More limited upper GI bleed*
- Melena
  - *Black, tarry, foul-smelling stool, probable upper GI bleed, possible lower GI bleed*
- Hematochezia
  - *Bright red bloody stool, probable lower GI bleed, possible upper GI bleed*
- Maroon-colored stool
  - *Probable lower GI bleed, possible upper GI bleed*

Medical History
- Peptic ulcer disease
  - History of H. pylori infection
  - NSAID use
  - Antithrombotic use
  - Smoking
- Varices or portal hypertensive gastropathy
  - History of liver disease
  - Excess alcohol use
  - H. pylori infection
- Marginal ulcer (ulcer at anastomotic site)
  - Gastroenteric anastomosis
- Medications
  - Aspirin and NSAIDs (peptic ulcer formation)
  - Bisphosphonates, tetracyclines, iron, KCL (pill esophagitis)
  - Warfarin, DOACs, antiplatelet agents
  - SSRIs, Ca^{2+} channel blockers, aldosterone antagonists (associated with GI bleeding)
  - Bismuth, charcoal, licorice, iron (alter clinical presentation by turning stool black)

Symptom Assessment
- Severe bleeding
  - Orthostatic lightheadedness
  - Confusion
  - Angina
  - Severe palpitations
  - Cold/clammy extremities
- Peptic ulcer
  - Upper abdominal pain
- Esophageal ulcer
  - Odynophagia
  - Gastroesophageal reflux
  - Dysphagia
- Variceal hemorrhage or portal hypertensive gastropathy
  - Jaundice
  - Ascites
- Mallory-Weiss tear
  - Emesis
  - Retching
  - Coughing prior to hematemesis
- Malignancy
  - Dysphagia
  - Early satiety
  - Unintentional weight loss
  - Cachexia

PHYSICAL EXAM

Signs of Hypovolemia/Hemodynamic Instability
- Resting tachycardia → <15% of blood volume loss
- Orthostatic hypotension → >15% of blood volume loss
- Supine hypotension → >40% of blood volume loss

Initial Signs of Significant Blood Loss
- Tachycardia
- Low urine output
- Narrowed pulse pressure (pulse pressure <25% of systolic pressure)

Abdominal Exam
Inspection
- The patient should lie flat, knees bent
- Inspect for scars, rashes, lesions, or striae

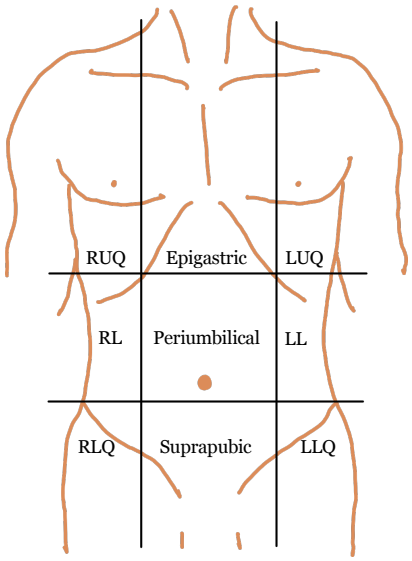
Auscultation
- Use the diaphragm to auscultate for bowel sounds prior to percussion or palpation
  - **Hyperactive, high-pitched bowel sounds** may be heard in early small bowel obstruction

Palpation
- Palpate with palmar finger surfaces
- Superficial palpation is done first to assess for tenderness, superficial masses, and muscular resistance
- Deep palpation is done second to assess for liver edge, kidneys, and abdominal masses

Liver Examination
- Place left hand behind patient's right 11th and 12th ribs and press upward
- Place right hand on right abdomen lateral to rectus muscle with fingertips well below the lower border of liver dullness
- Have patient exhale, then inhale, and feel liver edge as it descends

Guarding and Rebound Tenderness
- Guarding refers to contraction of abdominal muscles by patient
  - Involuntary guarding (rigidity) is a sign of an **acute abdomen**
  - Significant abdominal tenderness accompanied by signs of peritoneal irritation (e.g., involuntary guarding) suggests **perforation**
- Check for rebound tenderness by very slowly pressing deeply into patient's abdomen then rapidly withdrawing pressure
  - If withdrawal is more painful for patient, then rebound tenderness is present and is a sign of an **acute abdomen**

Rectal Exam
- Have patient lie on their side with knees bent towards their chest
- Insert a lubricated, gloved finger into rectum
  - Stool color on glove can help determine source of bleed (melena vs. hematochezia vs. brown)



MANAGEMENT

Risk Stratification and Triage
- High-Risk Features: hypotension, tachycardia, coagulopathy (INR > 1.5), AMS, syncope, age > 65, liver dx, CHF
- Risk Scores: Glasgow-Blatchford Score and ABC score recommended over AIMS65
- Triage: likely need for MICU if either BP < 90 and HR > 100 x2 30 min apart, Hgb < 7 regardless of vital signs with evidence of active significant bleed in past 12 hrs, >2L IVF or 2u pRBCs to prevent instability, or ATLS hemorrhagic shock class III (blood loss of 1.5-2L or 30-40% of blood volume)

Initial Workup
- ❑ CBC (q2-q8hr)
- ❑ CMP
- ❑ Coags
- ❑ Type & screen
- ❑ Rectal exam

Stabilization
- ❑ NPO
- ❑ Supplemental O_2 as needed (≥94% for patients w/o COPD)
- ❑ Intubation if large volume hematemesis or AMS (aspiration risks)
- ❑ ≥2 large bore peripheral IVs (16/18 gauge or larger)

Resuscitation
- ❑ Treat hypotension initially with **rapid, bolus infusions** of **isotonic crystalloid** (e.g., 500 – 1000mL NS or LR 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 ratio of pRBCs, plasma, and platelets, as for trauma patients
- For hemodynamic instability despite crystalloid resuscitation, transfuse 1-2u pRBCs
- For hemoglobin < 8 g/dL in high-risk patients (e.g. older adult, coronary artery disease), transfuse 1u pRBCs and reassess patient's clinical condition
- ❑ For **hemoglobin < 7 g/dL in low-risk patients**, transfuse 1u pRBCs and reassess the patient's clinical condition
  - Avoid over transfusion if possible esophageal varices (can increase portal pressures and worsen bleeding)
  - Hct drop lags 24-72hr from onset of bleeding
- Give PCC (lower volume, faster onset than FFP) for **coagulopathy** or **after transfusing 4u pRBCs**
- Give platelets for **thrombocytopenia** (platelets < 50,000) or platelet dysfunction (e.g., chronic aspirin therapy) or after transfusing 4u pRBCs
  - If uremic, consider ddAVP (0.3mcg/kg)
  - If ESLD, INR inaccurate so avoid FFP volume (can increase portal pressure)

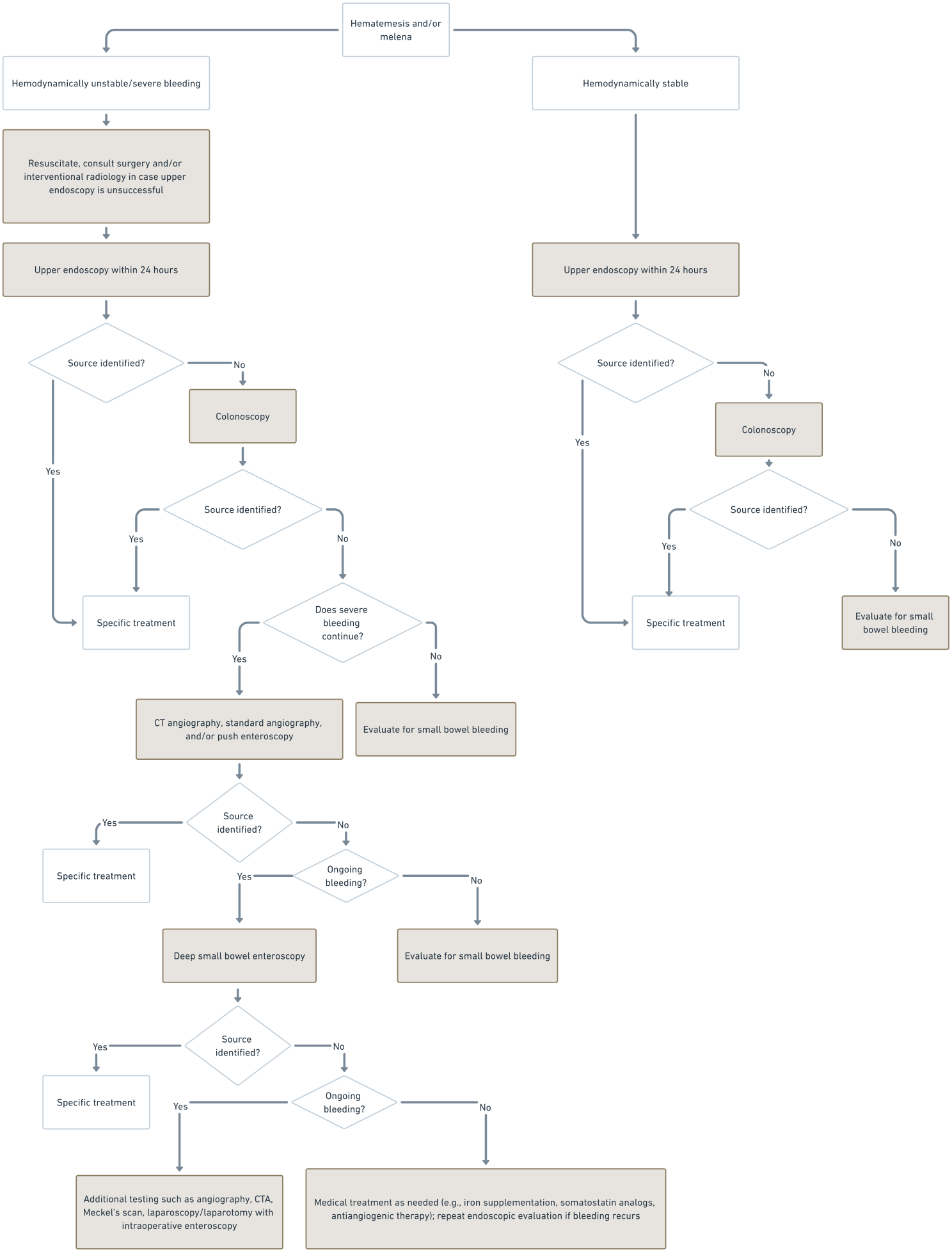
Consults
- ❑ **GI**
  - ❑ For EGD and/or colonoscopy
  - ❑ EGD within 24hrs (no change in outcomes if between 0-6hrs vs. 6-24hrs for non-variceal or non-HDUS bleeds)
- ❑ **Surgery/IR**
  - ❑ If hemodynamic instability or if endoscopy not preferred

Pre-EGD Pharmacotherapy & Management for Cirrhosis and Known/Suspected Esophageal Varices
- ❑ **IV octreotide** (somatostatin analog, decreases blood flow to GI/portal system) **50mcg bolus** (may repeat bolus in first hour if bleeding uncontrolled) followed by **octreotide gtt at 50mcg/hr for 3-5 days**
- ❑ **IV ceftriaxone (CTX) 1g q24hr x7 days** for ppx against bacterial infections and mortality benefit
- ❑ **Stop beta-blockers**

Pre-EGD Pharmacotherapy & Management for All Patients
- ❑ **IV PPI: pantoprazole 40mg BID** (neutralizing acid stabilizes clots); decreases high-risk lesions requiring endoscopic therapy but unclear clinical impact pre-EGD; if EGD delayed beyond 12 hours, give second doze 40mg IV
- ❑ **IV prokinetics: erythromycin 250mg** 30min prior to EGD to increase gut motility and visualization
- Other measures:
  - **NG lavage** may be helpful to clear clots, blood, and debris from stomach prior to EGD to improve visualization
  - **Balloon tamponade** may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (e.g., Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

Post-EGD Pharmacotherapy & Management
- Review GI procedure note for specific diet, PPI, and recommendations
- For high-risk PUD:
  - ❑ **IV pantoprazole 40mg BID x 72hrs**, decreases re-bleeds and need for repeat EGD. Switch to **PO PPI after 72hrs, discharge with BID dosing x2-8 weeks**
  - ❑ **Treat *H.pylori*** if positive
- For variceal bleed:
  - ❑ Consider **octreotide x2-5 days**
  - ❑ Continue **IV CTX 1g q24hr x7 days**
- For angiodysplasia: consider long-term **octreotide**, **bevacizumab,** or **thalidone** w/GI help
- If re-bleed: consider repeat EGD, angiography, surgical/IR consult. If variceal bleeding, consider balloon tamponade, TIPS, or BRTO

Anticoagulation/Antiplatelet Management
- **Warfarin: Hold** during bleed. For reversal, can consider PCC, but FFP or vit K NOT recommended. Resume after hemostasis (w/unfractionated heparin aka UFH bridge for ~48hrs if indicated). Decreases risk of thrombosis/death in AF if resumed within 7 days)
- **DOAC: Hold** during bleed. Reversal with idarucizumab, Andexanet alfa, or PCC NOT recommended. Resume within 72hrs in high thrombotic risk patients or within 7 days for low thrombotic risk patients
- **ASA: Continue** during bleed if low-moderate risk, hold if high risk (unless recent PCI/ACS). Resume ASA for secondary prevention after hemostasis endoscopically confirmed. Increased risk of 30d mortality if not resumed; if PUD, add PPI to decrease risk of re-bleeding
- **DAPT for PCI/ACS: Discuss with cardiologist.** Generally, if very recent (<30d PCI, <90d ACS), continue both unless life-threatening; if more distant, continue ASA but less risk with holding P2Y12i. Resume within 1-5 days pending course

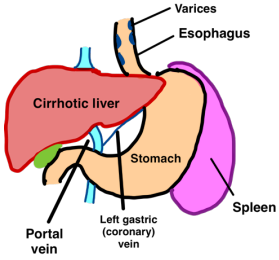

Hematemesis and/or melena
Hemodynamically unstable/severe bleeding
Hemodynamically stable
Resuscitate, consult surgery and/or interventional radiology in case upper endoscopy is unsuccessful
Upper endoscopy within 24 hours
Upper endoscopy within 24 hours
Source identified?
No
Yes
Colonoscopy
Source identified?
Yes
No
Specific treatment
Does severe bleeding continue?
Yes
No
CT angiography, standard angiography, and/or push enteroscopy
Evaluate for small bowel 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 (e.g., iron supplementation, somatostatin analogs, antiangiogenic therapy); repeat endoscopic evaluation if bleeding recurs
Source identified?
No
Yes
Colonoscopy
Source identified?
Yes
No
Specific treatment
Evaluate for small bowel bleeding

PATHOPHYSIOLOGY

Esophageal Varices

Cirrhosis → portal flow impeded → retrograde blood flow → varices

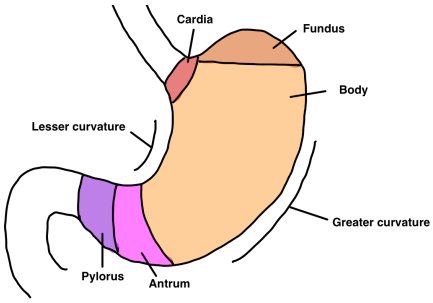
While the esophageal veins drain into the superior vena cava, distal veins in the submucosa connect to the left gastric vein (coronary vein), which normally drains into the portal vein. In cirrhosis, **restricted portal blood** flow causes **blood to back up under high pressure**, leading to the **formation of esophageal varices**, which are **vulnerable** to serious bleeding due to their increased **size, pressure, and surface exposure**.



Dieulafoy's Lesion

- A **congenital dilated tortuous artery**, typically on the **lesser curvature** of the stomach, that erodes the gastric lining without a primary ulcer, exposing it to gastric secretions and potentially causing massive upper GI bleeding
- **Endoscopy** typically shows **a small pinpoint defect** in normal-appearing gastric mucosa with a raised, visible vessel eroding through the lining
- Identified during endoscopy (when bleeding) and described as a visible vessel without an associated ulcer

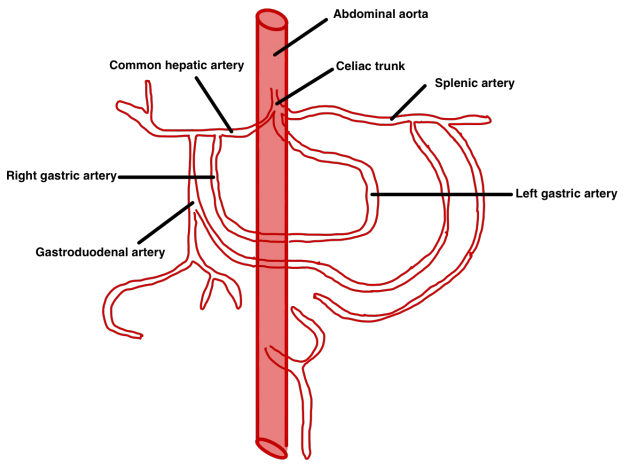
Sections of the Stomach



ANATOMY

The branches of the celiac trunk may be subject to erosion leading to **severe hemorrhage** if an ulcer penetrates through the gastrointestinal mucosa and into the vessel.

Branch of Celiac Trunk	Type of Ulcer	Location
Splenic artery	Gastric	Posterior wall of the stomach
Left gastric artery	Gastric	Lesser curvature of the stomach/gastric cardia
Gastroduodenal artery (off common hepatic artery)	Duodenal	Posterior wall of the first portion of duodenum



Acute and Chronic Gastritis

Acute gastritis is secondary to the dysfunction of mucosal defenses (**prostaglandins, bicarbonate, and somatostatin**) that are responsible for reducing the inflammatory effects that gastric acid can have on the gastric mucosa. Increased HCl secretion does NOT play a primary role.

- NSAIDs reduce production of prostaglandins and their protective mechanisms on the stomach lining
- Consumption of corrosive materials (e.g., household cleaners, pesticides, gasoline, cosmetics) can lead to acute gastritis

Chronic gastritis is long-term and divided into two types:

- Type A (fundus-dominant) linked to autoimmune disorders causing pernicious anemia and B12 deficiency
- Type B (antral-dominant) usually caused by H. pylori, leading to peptic ulcers and a higher risk of gastric cancer and MALT lymphoma

	Acute gastritis	Chronic gastritis
Erosive	Yes	No
Etiology	NSAID abuse, alcohol, steroids, uremia	Pernicious anemia, H. pylori infection
Pathogenesis	Decreased integrity of mucosal barrier	Inflammation related to autoantibodies or H. pylori infection

FACTOIDS

Obscure vs. Occult GI Bleeding
Obscure- obvious bleeding that is known to the patient but is hard to identify despite endoscopy
Occult- bleeding is not known to the patient, discovered either by fecal occult blood testing or by noting a microcytic iron deficiency anemia on blood testing

PCC vs. FFP
Both contain factors II, VII, and X (PCC can also have Factor IX in some formulations) but PCC contains less volume, can be administered quicker, and is more cost effective, making it preferable

Type and Screen vs. Type and Cross
Type- determines ABO and Rh status
Screen- identifies presence of alloantibodies in recipient's blood that may react with donor blood
Cross- recipient blood tested against donor packed cells to determine if clinically significant response occurs to antigens on donor's cells

Type and Screen- determines blood type and checks for antibodies, but *does not reserve* blood; use if likelihood of blood transfusion is low (elective surgery)

Type and Cross- matches specific blood units and *reserves blood* for transfusion; use if likelihood of needing blood is high

TRIALS & STUDIES

TRICC (1999)
In critically ill patients, restrictive transfusion (Hgb > 7 g/dL) is associated with better survival compared to liberal strategy (Hgb > 10).

Transfusion Strategies of Acute Upper Gastrointestinal Bleeding (2013)
Among patients with acute upper GI bleeding, a restrictive transfusion threshold (Hgb ≥ 7) was associated with reduced mortality at 45 days compared to a liberal transfusion threshold (Hgb ≥ 10).

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