Objectives: define differences between upper and lower GI bleeds, decide need for acute intervention versus stable evaluation, describe pathologic causes of upper GI bleeds and an algorithmic approach to the UGIB patient

Introduction: When approaching a patient who is bleeding from their GI tract, it is important to consider 1) their airway, 2) where the bleeding is coming from, which involves determining if it is from the Upper GI tract or Lower GI tract. 

Upper: from mouth to ligament of Treitz. 
Lower: from the ligament of Treitz to anus. 

It is important to remember that UGIB are more common than LGIB.

Presentation:

UGIB: melena and hematemesis (either frank blood or coffee-ground material) 
LGIB: hematochezia, rarely hematemesis. 

90% of melena (dark tarry stool due to digested blood) originates from the upper GI tract (above the ligament of Treitz). However, hematochezia (bright red or maroon blood that is fresh) can come from an upper GI source if there is a massive bleed and blood is transiting the GI tract quickly. Of course, these patients would likely present hemodynamically unstable from the rapid blood loss.

Causes of UGIB in order of most prevalent: PUD/Gastritis, Varices, Mallory Weiss Tear, gastric or esophageal cancers.

Workup pathway for all suspected GI bleeding:

1. Hemodynamically stable or unstable? 
   -all GI bleeding patients need at least 2 large bore IVs for resuscitation. 
   -type and screen those early if strong suspicion or active blood in stool or hematemesis on presentation 
   -closely watch respiratory status - patient might need intubation to protect airway if significant hematemesis. 
2. Obtain an HPI (only if hemodynamically stable and the patient is able to verbalize!) 
   -how much bleeding, how long? 
   -pertinent positives: abdominal pain, nausea/vomiting, bowel movements 
   -vomiting: How many times? is it bilious? Bloody? If bloody, is it frankly bloody or coffee-ground? 
   -stool: diarrhea? Formed, loose, or just frank blood? Bright red blood or dark and tarry? Clots? Painful? 
3. Obtain PMH and social history (do not overlook this!) 
   -has this happened before? If so, was it treated? (60% of previous GI bleeders rebleed!) 
   -use of NSAIDs or Goody’s/BC powder, aspirin, corticosteroids, anticoagulants? 
   -history of H. pylori diagnosis? Were they treated for it? 
   -history of GI malignancy? 
   -history of abdominal aortic aneurysm or an aortic graft? 
   -use of alcohol? Look for signs of cirrhosis on exam. 
   -history of GI surgeries? 
4. Perform a focused physical exam 
   -recheck the vitals. Hypotension is a late finding in young patients but is very concerning. 
   -abdominal exam that demonstrates focal tenderness, guarding, rebound tenderness concerning for peritoneal irritation (suggests viscous perforation). 
   -rectal exam to determine if gross blood if no obvious hematemesis or obvious blood in stool on presentation. +/- hemoccult assessment for occult blood but this has been called into question as reliable and accurate. We do stool hemoccult cards on a case by case basis, but we do believe they have a minor role in GI bleeding workup. 
5. Order these tests for every GI bleeder: 
   -CBC, CMP (tells us if there are electrolyte losses as well as with suspected liver disease). 
   -Consider getting coagulation studies. In the real world you should only get these if the patient has a history of anticoagulant usage, liver disease/alcohol abuse, or inherited coagulopathy.

The history and physical exam findings will determine your future diagnostic pathway: 
In general, UGIB patients need EGD (endoscopy). This is the gold standard for finding, and potentially treating, an UGIB. 

Here is the general flow chart in most patients:
Hematemesis +/- melena and/or patient has strong history suggesting UGIB

Source found?  
EGD in 36 hours  
HDS  
HDUS  
Resuscitate with fluids, blood products

No?  
Yes? Specific therapy (Clipping, etc.)  
EGD as soon as HDS. If fails to find source, perform colonoscopy.

What about NG tubes? Minimal evidence supporting their usage. Many studies have agreed that they are about 66% accurate and even up to 15% of patients with an active UGIB will have a negative NG tube. We do not recommend their usage.

EGD: studies have shown it to be routinely 95% diagnostic, and 85% therapeutic.

The following is a more detailed algorithmic approach to the UGIB patient with particular attention to those unstable:

Patient actively having hematemesis or profuse dark melanotic stool

Unstable or Stable?

Unstable

- Emergency release blood products \(\rightarrow\) consider MTP
- Type and screen, CBC, CMP, coagulation studies
- Aggressive supportive care with blood and vasopressors
- IV PPI
- Intubate for airway protection and hypoxemia early
- Call GI and establish need for EGD early (<12 hours)
- Reverse anticoagulants in patients in extremis

Stable

- Full History and Physical (see page 1)
- Type and screen, CBC, CMP, coagulation studies
- Discuss inpatient vs outpatient follow up depending on results of testing and patient and patient presentation.
- Discussion of GI consultation and admission for EGD/colonoscopy.

Hematemesis with history of varices/cirrhosis or evidence of cirrhosis on exam?

No

Continue critical care management

Yes

IV Octreotide and Ceftriaxone

Consider Blakemore tube placement

Detailed description of causes in order:

Most commonly, a patient with UGIB will present with gastritis. Usually these patients can be managed as outpatient and do well, however if the damage is severe enough, hemorrhagic gastritis can occur, where there can be melena and coffee-ground emesis (very similar to ulcers).

Gastritis: irritation \(\rightarrow\) inflammation of gastric mucosa. Gastritis and peptic ulcer disease are on a spectrum, with gastritis being the more “mild” variant that can potentially lead to ulcer formation. Can be acute or chronic. The most common cause of chronic gastritis is H pylori.

Acute: “CANCUN gave me gastritis”

- Cushing ulcers- “stress” induced inflammation from any head trauma.
- Alcoholism
- NSAID use- the most common cause of acute gastritis
- Curling ulcers- multiple, small ulcers. Result of massive volume depletion seen in burn patients.
- Uremia- ammonia is erosive on the gastric mucosa
- Nonspecific stress. Can occur in any patient with severe organic disease (i.e. ICU)

Treatment: stop the offending agent, treat the underlying cause. PPI or H2-blocker trial can be given for 2 weeks.

Chronic gastritis: Most common cause is H. pylori. Acute management is same as acute gastritis with addition of triple therapy.

Peptic Ulcer Disease (PUD): ulcer formation in either the stomach or duodenum. Cause: H. pylori > NSAIDs.
Inflammation of gastric mucosa → Increased pH or loss of protective mucosa → epithelial injury

Major high yield acute complications of PUD:

1) Hemorrhage: most common complication. Often presents with coffee ground emesis or frank bloody emesis and melena. If hemorrhage severe enough, patients can have hematochezia.

2) Perforation: the ulcer ruptures through all layers of the stomach/duodenum, leading to air entering the abdominal cavity and gram-negative bacteria entering as well. Patients will present with signs of peritonitis and not really any hematemesis or melena.

**Mallory-Weiss Tear: Painful** incomplete tearing of the esophagus (only the mucosa and submucosa). The most common cause is due to repeated bouts of vomiting and retching → high amounts of gastric acid enters and damages the distal esophagus. The tear is **linear** in appearance.

The classic presentation are patients who are retching (dry heaving) after multiple episodes of vomiting, very common in alcoholics. There will be varying amounts of hematemesis. Some present with blood-tinged vomitus, others present with profuse bloody vomitus and require urgent EGD.

Treatment: observation and fluid rehydration for the majority. EGD can be needed high risk patients.

**Assuming the worst...**

**Esophageal varices: painless** esophageal bleeding with hematemesis due to portal hypertension. Increased pressure in the left gastric vein leads to blood pooling in the esophageal veins. When under stress they can burst.

Presentation: cirrhotics with portal hypertension. They will present with frank hematemesis that began suddenly often with no precipitant vomiting. This is **releantless** hematemesis, often necessitating airway protection.

Diagnosis: Endoscopy = tortuous veins under the mucosa in the esophagus. Very difficult to see if patient is actively bleeding.

Acute treatment: Airway protection → Resuscitation with large IVs → IV octreotide*, IV PPI, IV ceftriaxone** → EGD with banding or sclerotherapy → if failed, balloon inflation in the esophagus until surgical management can occur (e.g. TIPS).

*Octreotide works by reducing portal venous pressure. It might decrease mortality, but it has been shown to decrease transfusion requirements.

**Antibiotics are needed to reduce bacterial complications given location of bleeding and bacterial migration. Antibiotics do improve mortality and reduce risk of re-bleeding and infection.

**Aortoenteric fistula: patients that have history of AAA especially with graft repair can have compression of this major vessel against the surrounding gastrointestinal structures (most commonly the duodenum). A resulting fistula can form between the structures and as you can predict this is a recipe for disaster. CT angiography is indicated if patient is HDS. Surgery should be called very early on.

References: