AHD: Upper GI Bleeding (UGIB)
Facilitator Guide

CASE 1

Mr. Pud is a 68 y/o Caucasian male with obesity, chronic back pain, and tobacco use who presents to the ED for 3 days of abdominal pain and dark sticky stools. He has had epigastric abdominal pain for years but notes worsening pain over the past 5 days.

1. What are this patient's risk factors for peptic ulcer disease? Does he have any risk factors for a poor outcome?

PUD risk factors:

- *Obesity = increased intraabdominal pressures – increased risk of hiatal hernias, acid reflux, dyspepsia*
- *Smoking = increased inflammatory cytokines, irritation of mucosal lining, slowed healing and reduced mucous secretion; potentiate gastritis, increases risk of ulcerations, and impairs response to therapy*
- *Chronic back pain: consider chronic NSAID use*
- *Dark stool = need to determine if it is actually melena = black stool, sticky –dark 2/2 hemoglobin oxidation in the ileum and colon, most often a sign of UGIB due to the longer transit time. Hematochezia can occur in UGIB but is less common and indicates brisk UGIB. Patient less likely to be hemodynamically stable and asymptomatic.* ***Review what upper vs lower GIB means*** *(cutoff = ligament of Treitz)*
- *Epigastric burning = sign of dyspepsia, GERD or PUD*

Prognostic factors:

- In this patient, age >65

2. What other questions do you want to ask the patient and why?

*- **Meds** = NSAIDs, ASA/plavix, anticoagulation; steroids and SSRIs also increase risk of UGIB, especially when taken with NSAIDs.*

***SSRI mechanism** = platelet serotonin depletion reduces hemostatic response to vascular injury*

***NSAID mechanism =** directly (cell injury) and indirectly (inhibiting Cox-1/Cox-2 pathway; Cox-1 inhibits prostaglandin production which protects the surface mucosal barrier)*

*- **Hx** of prior GIB, H pylori (and treatment)*

*- **Details about melena** (frequency, amount, any BRBPR)*

*- **Other symptoms** = syncope, fatigue, lightheadedness, orthostasis out of concern for severe blood loss*

*- **EtOH/Drug Use, Hx of cirrhosis** or evidence of liver disease*

*- **Diet? No -** Some food/beverages associated with dyspepsia, but no conclusive evidence of association between ulcer formation and specific diet*

The ED obtains basic labs seen below. You speak to the patient and obtain more information. Your physical exam is as follows.

More HPI *(students have this information on the next page but I'd like them to ask these history points before seeing it)*

Pt started taking baby aspirin 8wks prior after friend had a heart attack. He uses extra strength ibuprofen 2-4x daily for his lower back pain for the last three months. No other med use. He is having 4-8 episodes of melena per day for 5 days. He endorses intermittent lightheadedness but no syncope. He has had no prior episodes of GIB; no prior colonoscopy or EGD in the past. Drinks one beer every 1-2 weeks.

Physical exam

VS: T 98.7, RR 14 satting 98% on RA, HR 111bpm, BP 120/80 mmHg laying 110/75 mmHg standing
GEN – AOx4, appears mildly anxious
HEENT – Normocephalic. PERRL, conjunctival pallor.
HEART – sinus tachycardia, regular rhythm, no murmurs, rubs or gallops.
CHEST – lungs are clear to auscultation, no rales
ABD – round, central adiposity, hyperactive sounds, soft but moderately tender to palpation at epigastrium
RECTAL– black tarry stool. No hemorrhoids, fissures.

Labs

WBC 13,000	Hb 9.1 mg/dL	Hct 27%	Plt 195,000	MCV 85
Na 137	Cl 10	BUN 45	Glucose 129	
K 4.1	HCO3 22	Cr 1.0		

3. What are the most important features of the exam and labs? Do any of these help you diagnostically? (Consider which findings have the best positive LRs.)

- *Comment specifically on tachycardia, pallor, hemoglobin of 9.1, elevated BUN, discuss why BUN is elevated in upper GIB, digestion of proteins from hb). Ideally would like to know what his baseline Hb is.*
- *EBM refresher. Pull up Bayes nomogram and review these LRs!*
    - *Positive LR of patient-reported melena: 5-6*
    - *Positive LR of BUN:Cr >30 is 7.5 for UGIB*
    - *Positive LR of observed melena in stool or on DRE is 25*
- *note Less than 15% of blood volume lost will usually have resting tachycardia, Greater than 15% usually has orthostatic hypotension, greater than 40% has supine hypotension*

4. Is there any utility for sending the stool to the lab at this point for FOBT or FIT testing in this case?

NO! Lets talk about testing stool for the presence of blood. What are the tests available?

- *FOBT: aka guaiac testing. "heme present in the sample reacts with a hydrogen peroxide developer to oxidize guaiac, producing a blue color"*
    - *High incidence of false positive results (type I error)*
    - *"False-positive FOBT results can occur from ingested blood via extra-intestinal sources (eg, epistaxis, gingival bleeding, pharyngitis, hemoptysis), or in conditions with intestinal mucosal inflammation (eg, esophagitis, gastritis, inflammatory bowel disease), GI blood loss induced by medications (eg, aspirin, nonsteroidal anti-inflammatory drugs), alcohol, or by ingestion of meats, fruits, or vegetables containing peroxidase (eg, broccoli, cauliflower)."*
- *FIT testing: uses anti-globin antibodies to detect the presence of blood in the stool*
    - *Subject to false negatives bc most hemoglobin from UGIBs is digested in the small bowel thus the globin protein is not present in the stool*
- *These tests are not validated in the setting acute GIB, and several studies have shown that they do not alter management in this situation. Use your clinical history and exam!*

5. What is your most likely diagnosis?

- Leading diagnosis likely UGIB, specifically PUD in setting of NSAIDs, ASA, time course.

Let's discuss some other differentials for this case as well as clinical features of each diagnosis.
(Italicized is NOT shown on their handout)

Cirrhotic patient with esophageal variceal bleed	*Abdominal distention, hematemesis, melena, hematochezia if severe, possible history of alcohol abuse or HCV; exam with spider angiomata, caput medusa, palmer erythema, gynecomastia*
Mallory-Weiss tear	Multiple episodes of retching followed by hematemesis; longitudinal mucosal tear seen on EGD
Aortoenteric fistula	Prosthetic abdominal aortic vascular graft; may be associated with a herald bleed, often associated with graft infection
Dieulafoy lesion	Dilated aberrant submucosal artery, characterized by self-limited (but often recurrent) episodes of bleeding
AVM of the small intestine	Elderly, persistent occult blood loss anemia with a negative colonoscopy; may require angiography or capsule endoscopy for diagnosis.
Pill-induced esophagitis	Chest pain worsened by swallowing, PO antibiotic use (especially doxycycline), increased incidence in those with left atrial enlargement
GERD	*Heartburn, water brash, chest pain, cough*
Gastric cancer	Middle-aged smoker with long-standing untreated heartburn who presents with weight loss, early satiety.
GAVE (gastric antral vascular ectasia)	A cause of acute or chronic gastrointestinal blood loss in the elderly and endoscopically described as 'watermelon stripes.' No clear etiology; some association with cirrhosis.

6. Discuss how you would assess and treat this patient.

- ***Hemodynamic assessment***
  - *If severe hematemesis, may need intubation for airway protection, larger catheter for massive transfusion*
- ***Risk Assessment***
  - *The article discussed using **Glasgow-Blatchford** in clinic/ED to determine need for admission versus outpatient follow-up. Have them look this up on MDCalc (patient is higher risk at 12; studies have shown that patient with scores of 0-1 (or 2 if <70yo) can likely be followed as an outpatient. Low risk = Score of 0. Any score higher than 0 is high risk for needing intervention: transfusion, endoscopy, or surgery. Sensitive, not Specific). The **AIMS65 score** is another risk assessment tool that was found to be better at predicting in-hospital mortality, need for ICU mortality, and LOS; however, the GBS is superior at predicting need for transfusion. Note that most patients meet criteria for admission based on GBS*

  - *Based on his clinical presentation patient requires admission; discuss floor v step down v MICU*
- ***NG or OG lavage**? Observational evidence does NOT show a clinical benefit in UGIB. Even if NG Aspiration is negative patient could still be having a bleed*
- ***Call GI** for consult and EGD! Have the team discuss how they would go about doing this and what information they would provide the GI Fellow*
- ***Orders***
  - ***Labs**: Coags, "Type and Cross," **trend H&H how frequently?***
  - ***2 large bore IV's** – ask them what they consider large bore and why is it better than say a PICC!*
  - ***Med Rec**: hold NSAIDs and ASA, anticoagulation*
  - *DVT ppx?*
- ***PPI and the data:***
  - *PPI IV 80 mg bolus followed by 8 mg/h infusion may be considered to decrease proportion of patients who have higher risk stigmata of hemorrhage at endoscopy (active bleeding, non-bleeding visible vessel, adherent clot) and who receive endoscopic therapy. Some evidence that intermittent dosing (IV BID) is non-inferior to bolus + continuous infusion but these studies are done AFTER initial EGD*
- *Resuscitation; do they need **PRBC or IVF? What's your threshold?***

  NEJM 2013: Transfusion strategies for acute upper GI bleeding (referenced briefly in their reading).

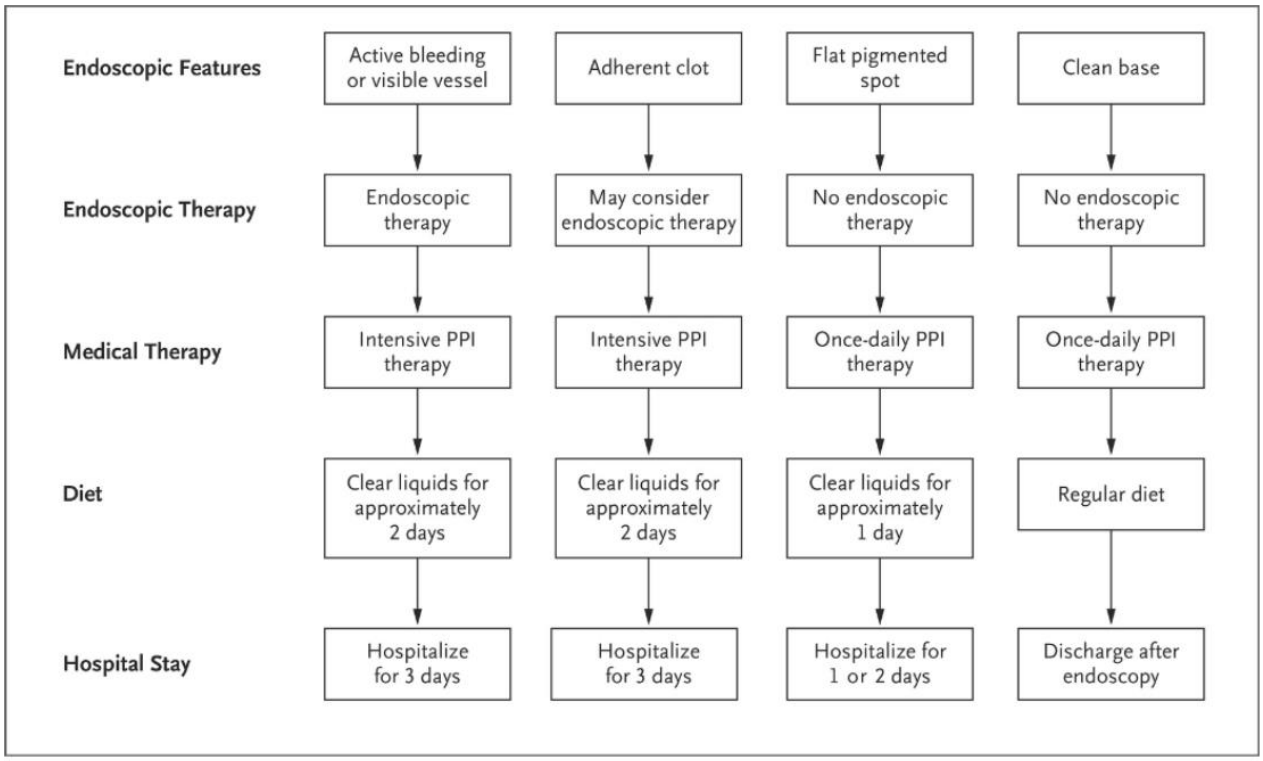
  Restrictive – Hgb threshold 7 g/dL (goal 7-9 g/dL post-PRBC)

  Liberal – Hgb threshold 9 g/dL (goal 9-11 g/dL post-PRBC)

  <u>Restrictive strategy significantly improved outcomes in patients with UGIB compared to liberal strategy</u>!!

The GI fellow pages you s/p EGD. What do you plan to ask about the procedure and why?

*- **EGD findings**: determines further Rx (most high risk for rebleeding to least)*



- Actively bleeding or nonbleeding vessel = need endoscopic treatment, intensive <u>PPI (IV x 72 hrs)</u>
rebleed risk of 43-55% with mortality of 11%

- *Adherent Clot = intensive PPI (<u>IV x 72 hrs</u>) +/- endoscopic treatment*
  - *Rebleed risk 22% with mortality of 7%*
- *Flat pigmented spot, clean based ulcer = no endoscopic intervention<u>, oral PPI</u>*
  - *Rebleed risk 5-10% and mortality 2-3%*

*- **Biopsy** (for malignancy, especially if gastric ulcer, and for H pylori!)*

- During endoscopy, biopsies for histology with rapid urease testing (RUT) – sensitivity of 77% and specificity of 100%. RUT is affected by PPI use in the last 1-2 weeks, antibiotics, blood, or bismuth use in last 4 weeks. Some guidelines therefore recommend repeat testing if patient negative on biopsy and high suspicion 2/2 decreased sensitivity during active GIB and PPI. Can also discuss urease breath test and fecal antigen testing in outpatient setting and lack of usefulness of Ab testing.

7. You are ready to discharge the patient 72 hrs later. What are key patient instructions and actions?

1) *STOP NSAID's – sounds obvious but it is often MISSED! Med rec and patient education is key! If the MUST be on NSAIDs. Like failed everything else. Use COX-2 Inhibitors WITH PPI*

2) *Do you Stop ASA? – The choice of whether to discontinue or withhold antiplatelet agents in patients with clear indications for treatment should balance the risk of rebleeding with the risk of thrombosis. Aspirin **reduces** mortality rates **tenfold** over 30 days while **increasing rebleeding** rates **only twofold**.*

   a. *If primary prevention, likely yes after conversation with the patient (NNT to prevent one MI, stroke or CV death is 1745 – risk of rebleed likely outweighs benefit).*

   b. *BUT continue if on ASA for secondary prevention (NNT is 67) so restart within 3-5 days or as soon as possible when patient has achieved homeostasis and is hemodynamically stable. If patient is on Plavix too, discuss with cardiology.*

3) *SMOKING CESSATION*

4) *WEIGHT LOSS*

****Bonus question**

What if the patient is on anticoagulation? (Note, this question was removed from the 2020 learner guide but can be discussed if there is time).

Patients with high thrombotic risk should receive re-anticoagulation but only after evaluating thromboembolic risk against rebleeding risk. Criteria for high thrombotic risk are chronic atrial fibrillation with a previous embolic event, CHADS2 score of 3 or greater, recent acute coronary syndrome, mechanical heart valve, deep venous thrombosis, pulmonary embolism, or hypercoagulable state. There are limited data for resuming anticoagulation after major GIB, particularly for patients with **endoscopically treated high-risk ulcers**; however, available data support beginning with (1) a bridging treatment (such as low-molecular-weight or unfractionated heparin) and careful observation or (2) beginning oral anticoagulation 7 days after the bleeding event. The latter approach, according to a retrospective study, reduces mortality and risk of thromboembolism (in patients with atrial fibrillation) without increasing GIB risk compared with withholding anticoagulation for 30 days.

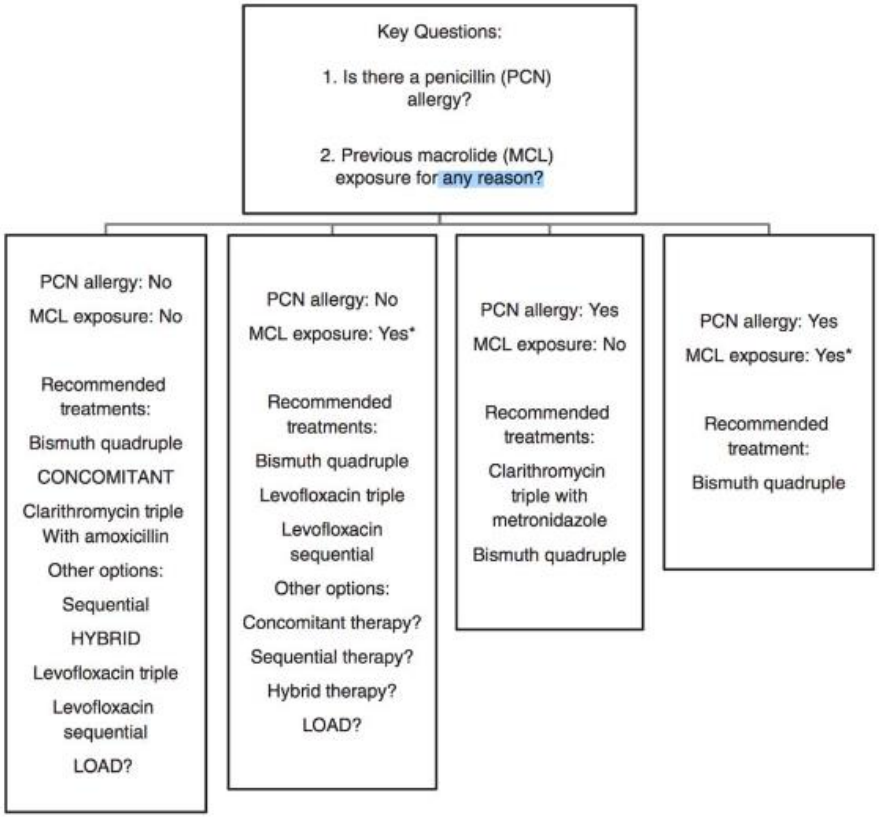
8. Biopsy results come back after patient is discharged and are positive for H pylori. You create a phone encounter in Epic, call the patient, and write a brief note. What do you need to ask the patient before prescribing and what meds do you send to the pharmacy? What do you tell the patient?

ACG guidelines for H pylori treatment provided. They need to know if the patient has a PCN allergy!

ACG guidelines for H pylori treatment: Meds
Clarithromycin triple therapy consisting of a PPI, clarithromycin, and amoxicillin or metronidazole for 14 days remains a recommended treatment in regions where *H. pylori* clarithromycin resistance is known to be <15% and in patients with no previous history of macrolide exposure for any reason
Bismuth quadruple therapy consisting of a PPI, bismuth, tetracycline, and a nitroimidazole for 10–14 days is a recommended first-line treatment option. Bismuth quadruple therapy is particularly attractive in patients with any **previous macrolide exposure** or who are **allergic to penicillin**

*The main **determinants of successful H. pylori eradication** are the choice of regimen, the patient's **adherence** to a multi-drug regimen with **frequent side-effects**, and the sensitivity of the H. pylori strain to the combination of antibiotics administered.*

*Whenever H. pylori infection is identified and treated, testing **to prove eradication** should be performed using a urea breath test, fecal antigen test or biopsy-based testing at least **4 weeks after the completion of antibiotic** therapy and **after PPI therapy has been withheld for 1–2 weeks.***



Possible expert question vs discussion in group: When do you stop the PPI after all this?

Case 2:

Mr. C. Roses is a 55 y/o m with DM2 (HbA1c 8), untreated HCV, cirrhosis decompensated by ascites and HE. Has never had a GI bleed but had varices on his last EGD 2 months prior. Patient is being admitted for altered mental status. You go see him in his room and witness him vomiting about ½ a cup of bright red blood into bedside basin!

Physical Exam:

VS: T99, HR 122 bpm, BP 98/52, RR 18, SPO2 96%

GEN – appears anxious, diaphoretic

HEENT – Normocephalic. PERRL, conjunctival pallor.

HEART – sinus tachycardia, regular rhythm, no murmurs
CHEST – lungs are clear to auscultation, no rales
ABD – round, mildly distended
SKIN – +spider angiomata, palmar erythema
NEURO – AOx4, no asterixis

Labs:
WBC 11.9 Hgb 7.1 (10.1 two months ago), Hct 20, Plt 98
Na 130, Cl 89, HCO3 28, BUN 42, Cr 1.3, glucose 70
Bili 2.1, Alb 2.9, INR 1.9

1. Write down your problem representation for this patient and compare answers in your group. As a reminder, a problem representation is a 1-2 sentence summary of the case using semantic qualifiers (tempo/duration of illness, characteristics of symptoms) that will help frame your thinking about the patient.

Example problem representation:

- *55 yo M with DM2, HCV and cirrhosis decompensated by HE and ascites with varices on prior EGD having acute onset large volume hematemesis.*
- *If time, can engage in some discussion here about building a good problem representation. Should have the background (cirrhosis, known varices) and the foreground (acute onset, large volume hematemesis). You could consider creating a problem representation for the patient in case 1 and comparing the two side by side.*

2. What is your working diagnosis?

Variceal hemorrhage

3. Cirrhosis refresher: What is your initial treatment plan for this patient?

- *Hemodynamic assessment*
- *Repeat labs – CBC, CMP, coags, type and cross if not yet done*
    - *No data to support initially correcting an INR with FFP (not a reliable indicator of coagulation status in cirrhotic)*
- *Ensure good access – 2 large bore IVs*
- *Consider emergent intubation to secure the airway*
- ***Call GI asap** to make them aware of possible variceal hemorrhage – may need EVL (endoscopic variceal ligation)*

- *Resuscitation –PRBC – goal Hgb 7-8mg/dL (PRBC > crystalloid – aggressive volume resuscitation with IVF with caution – worse outcomes seen in patients overly resuscitated, increases portal pressures and increases pressure in the varix)*
- ***Ceftriaxone 1g IV q24h for 7days** – shown to decrease rate of infection (esp SBP), recurrent hemorrhage, and mortality! For all cirrhotics, not only those with ascites.*
    - *Note: this is for any GI bleed in cirrhotic patients (even lower GIBs)*
- ***Octreotide** 50mg IV bolus followed by 50mg/h (to be continued for 3-5 d)*
    - ***Mechanism:** a somatostatin analogue and causes splanchnic vasoconstriction and decreased portal pressure (vasopressin and terlipressin are others); decreases mortality and transfusion requirements*
- ***IV PPI** - ligation causes superficial ulceration – continue with PO when stable for short term (time frame debatable <2-4wks)*

3. Patient arrives to MSD and has at least 500 cc of hematemesis with clots. What is your next plan of action?

Move him to ICU, monitor airway closely, repeat H&H although initial value likely not reflective of true value (patients bleed whole blood so this number doesn't drop immediately – takes hrs to equilibrate as intravascular volume replenished with IV/interstitial fluid), ensure blood has been "prepared," call GI with status update to see if they will scope sooner, likely transfuse now depending on his hemodynamic status and prior Hb of 7.1

Case 3:

70 y/o male patient with a history of CKD, CAD, COPD and GI bleed presents from nursing facility due to fatigue and streaks of blood in his stool. Patient was recently hospitalized for a lower GI Bleed. During that stay patient received a blood transfusion and his Hgb stabilized. As an outpatient he had both upper and lower endoscopy which did not show a potential source of bleeding.

Physical exam

VS: T 98.7, RR 14 satting 98% on RA, HR 98bpm, BP 125/80 mmHg

GEN – AOx4, appears comfortable

HEENT – Normocephalic. PERRL, no conjunctival pallor.

HEART – sinus tachycardia, regular rhythm, no murmurs, rubs or gallops.

CHEST – lungs are clear to auscultation, no rales

ABD – soft non-tender to palpation

RECTAL– Brown stool with some dark blood mixed in. No hemorrhoids, fissures.

Labs

WBC 9,000 Hb 6.5 mg/dL Hct 27% Plt 195,000 MCV 85

Na 137 Cl 10 BUN 45 Glucose 129
K 4.2 HCO3 25 Cr 1.8 (baseline 1.5)

1. What are the important parts of this patients HPI? What else do you want to know about this patient? What do you make of the previous History of GI bleeding? What is on your differential?

Just like in the previous cases it's important to ask for more information pertaining to past medical history including medications, social history, surgeries etc. Majority of lower GI bleeding resolves on its own such as in his previous episode but rebleeding is common.

2. What do you do next? What studies or procedures would you recommend?

Most important thing to do is stabilize the patient and monitor for further bleeding. This patient should receive a blood transfusion for a goal Hgb >7. They should also be made NPO if concern for continuing bleeding until stable and plan for diagnostic studies are finalized. In terms of further work up GI should be consulted to evaluate for repeat upper and lower endoscopy if stable before proceeding to other studies such as angiography, pill-cam, push enteroscopy.

3. You consult GI a upper and lower endoscopy are negative. Overnight you are called to bedside due to tachycardia. When you arrive, patient complains of lightheadedness and shortness of breath. You note a pool of blood in the bed. What do you do next?

STABILIZE the patient!! Begin fluids, prepare do give blood, get STAT labs. Next or while this is going on contact GI Fellow and update on change in status. If patient easily stabilized may recommend repeat endoscopy. However, given the recent negative studies and severity of bleeding CT angiography and possible embolization is likely most appropriate option. Call IR!