Gastrointestinal bleeding

Objectives:

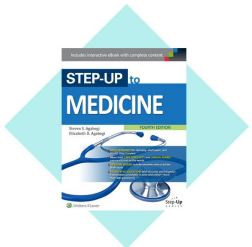
- Identify presenting features of patients with suspected upper GI bleeding
- Appropriate identification and risk stratify patients with suspected upper GI bleeding.
- Appropriate resuscitation and initial management of patients.
- Appropriate medical management of patients with upper gastrointestinal bleeding and the appropriate alternatives to endoscopy based on the clinical situation.

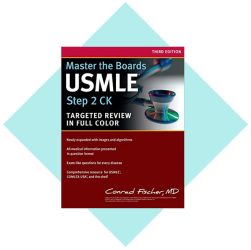
[Color index : Important | Notes | Extra]

[Editing file | Feedback | Share your notes | Shared notes | Twitter]

Resources:

- 435 slides



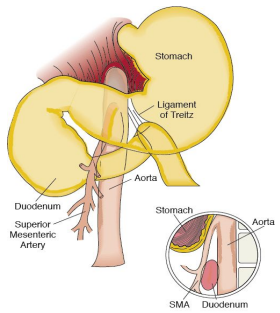


Check out this mind map here

- Done by: Jwaher Alharbi | Gassan almogbel | Mohammed aldegaither
- Team sub-leader: Jwaher Alharbi
- Team leaders: Khawla AlAmmari & Fahad AlAbdullatif
- Revised by: Ahmad Alyahya

Upper VS. lower GI bleeding

❖ Anatomical landmarks and location of gastrointestinal bleeding :



- Upper GI bleeding : a source of bleeding above the **ligament of Treitz** (suspensory muscle of duodenum).
- Lower GI bleeding : bleeding below the **ligament of Treitz**

❖ Acute upper gastrointestinal bleeding :

(Acute UGIB is a common medical emergency that has 11% hospital mortality rate)

The cardinal features are **haematemesis** (the vomiting of blood) and **melaena** (the passage of black tarry stools, the black colour being due to blood altered by passage through the gut). Melaena can occur with bleeding from any lesion *proximal to the right colon.* Rarely, melaena can also result from bleeding from the right colon. Following a bleed from the upper gastrointestinal tract, unaltered blood can appear per rectum, but **the bleeding must be massive** and is almost always accompanied by shock. The passage of dark blood and clots without shock is always due to lower gastrointestinal bleeding.

❖ Acute lower gastrointestinal bleeding :

Massive bleeding from the lower gastrointestinal tract is rare and is usually due to **diverticular disease** or ischaemic colitis. Common causes of small bleeds are haemorrhoids and anal fissures.

❖ Etiology:

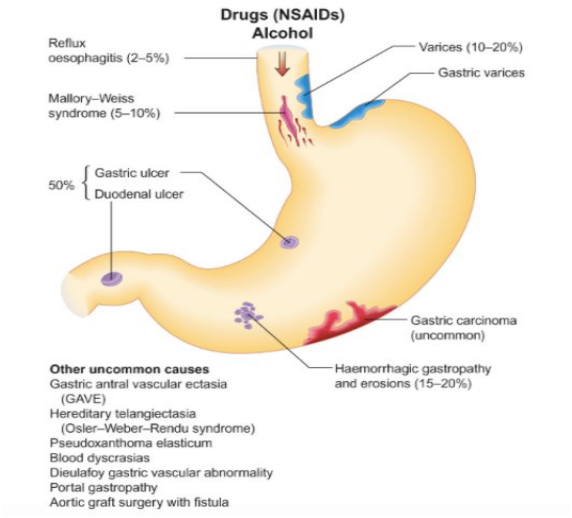
- The most common cause of **upper GI bleeding** is **peptic ulcer disease**
- The most common cause of **lower GI bleeding** is **Diverticulosis**[1]

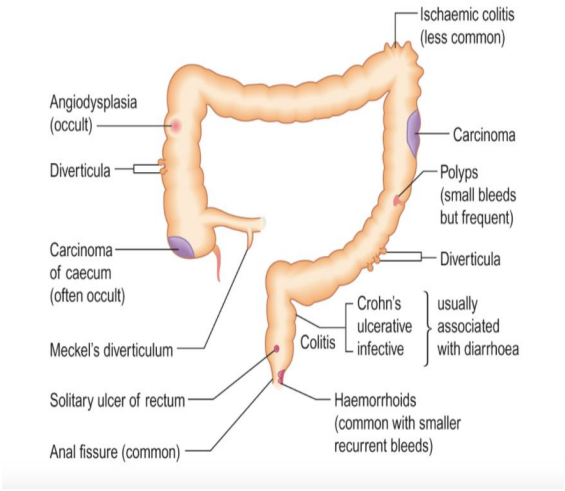
Upper GI bleeding	Lower GI bleeding
1. **Peptic ulcer disease** (duodenal ulcers 25% and gastric ulcer 20%). Most common 2. Esophagitis / Gastritis / duodenitis 3. Variceal bleeding 4. Mallory weiss tear and its severe form: Boerhaave syndrome (usually lethal) **Other uncommon causes:** 1. Arteriovenous malformation 2. Gastric antral vascular ectasia 3. Dieulafoy's lesion[2] (vessel that bleed and disappear) 4. Haemobilia[3] 5. Aortoenteric fistula after aortic surgery 6. Malignancy usually not an emergency	1. **Diverticular disease** (40%) most common source of GI bleeding in patients over age of 60, usually painless 2. Angiodysplasia (AVM) (40%) second most common source in patients over age of 60 3. IBD (UC, Crohn's disease) 4. Colorectal carcinoma 5. Colorectal adenomatous polyps 6. Ischemic colitis 7. Haemorrhoids, anal fissures 8. Small intestinal bleeding diagnosed by excluding upper GI and colonic bleeding

[1] Diverticulosis is the condition of having multiple pouches (diverticula) in the colon that are not inflamed. These are outpockets of the colonic mucosa and submucosa through weaknesses of muscle layers in the colon wall.They typically cause no symptoms.Diverticular disease occurs when diverticula become inflamed, known as diverticulitis, or bleed.

[2] Submucosal dilated arterial lesions that can cause massive GI bleeding.

[3] bleeding into the biliary tree





❖ Vericeal UGIB :

Defined as bleeding from the esophagus, stomach, or duodenum. Mortality decreased from 40 % to 15%. Fatality rate from 2.1 to 2.5% to 10%.

Portal HTN :

- Pre-hepatic abnormalities (eg, portal or splenic vein thrombosis).
- Post-hepatic abnormalities (eg, Budd–Chiari syndrome).
- Intrahepatic noncirrhotic causes (eg, schistosomiasis, sinusoidal obstruction syndrome) or cirrhotic liver disease.
- Portosystemic gradient (hepatic venous pressure gradient (HVPG)):
    - wedged hepatic venous pressure (a measure of sinusoidal hepatic pressure) - the free hepatic venous pressure
    - normal HVPG is 3 to 5 mmHg
    - HVPG > than 5 mm Hg defines portal hypertension
    - HVPG > 10 mmHg higher risk of : (varices, clinical decompensation “i.e., development of ascites, variceal hemorrhage, and hepatic encephalopathy”, hepatocellular carcinoma).

Clinically significant portal hypertension (CSPH):

- presence of collaterals on imaging studies.
- gastroesophageal varices on endoscopy.
- the presence of ascites patient with decompensated cirrhosis.

❖ Clinical features:

1. Type of bleeding :	
Hematemesis	vomiting blood; suggests upper GI bleeding (bleeding proximal to ligament of Treitz). Indicates moderate to severe bleeding that may be ongoing.
"Coffee grounds" emesis (5-10 ml)	suggests upper GI bleeding as well as a lower rate of bleeding (enough time for vomitus to transform into "coffee grounds (حثال القهوة)").
Melena *black, tarry, liquid, foul-smelling stool*	• Caused by degradation of hemoglobin by bacteria in the colon; presence of melena indicates that blood has remained in GI tract for several hours • The further the bleeding site is from the rectum, the more likely melena will occur. • Note that dark stools can also result from bismuth, iron, spinach, charcoal, and licorice. • **Melena suggests upper GI bleeding 90% of the time**. Occasionally, the jejunum or ileum is the source. It is unusual for melena to be caused by a colonic lesion, but if it is, the ascending colon is the most likely site.
Hematochezia	• This usually represents a lower GI source (typically left colon or rectum). Consider diverticulosis, arteriovenous malformations, hemorrhoids, and colon cancers. • **It may result from massive upper GI bleeding** that is bleeding very briskly (so that blood does not remain in colon to turn into melena). This often indicates heavy bleeding, and patient often has some degree of hemodynamic instability. An upper GI source is present in about 5% to 10% of patients with hematochezia.
Occult blood in stool	Source of bleeding may be anywhere along GI tract.
2. Signs of volume depletion (depending on rate and severity of blood loss).	
Increased pulse rate (>100 per minute), decreased blood pressure (systolic blood pressure < 100 mmHg), Increased respiratory rate, decreased urine output, decreased mental status.	
3. Symptoms and signs of anemia (e.g., fatigue, pallor, exertional dyspnea).	

Notes : (Step-up to medicine)

- A lower GI bleed (or positive occult blood test of stool) in patients over 40 is **colon cancer** until proven otherwise.
- About 80% of episodes of upper GI bleeding stop spontaneously and only need supportive therapy.
- Bleeding from the **small bowel** may manifest as melena or hematochezia.
- **Colonic** sources of bleeding present with either occult blood in the stool or hematochezia.
- Always ask patients with GI bleeding if they take any **NSAIDs/aspirin, clopidogrel or anticoagulants.**
- **Hematemesis and melena are the most common presentations of acute upper GI bleed**, and patients may have both symptoms. Occasionally, a brisk upper GI bleed presents as hematochezia.

Tests to Order in Patients With GI Bleeding:

Hematemesis—An upper GI endoscopy is the initial test.

Hematochezia—First rule out an anorectal cause (e.g., hemorrhoids). Colonoscopy should be the initial test because colon cancer is the main concern in patients over age 50.

Melena—Upper endoscopy is usually the initial test because the most likely bleeding site is in the upper GI tract. Order a colonoscopy if no bleeding site is identified from the endoscopy.

Occult blood—Colonoscopy is the initial test in most cases (colon cancer is the main concern). Order an upper endoscopy if no bleeding site is identified from the colonoscopy.

❖ Diagnosis :

For acute bleeding especially when the bleeding is severe it is far more important to replace fluids and check hematocrit, platelet count and coagulation tests as the prothrombin time or INR than it is to do an endoscopy

Nasogastric tube placement guide you where to start with endoscopy (upper or lower)

Angiography :

Specific vessel or site of bleeding needs to be identified prior to surgery or embolization of the vessel (used only in massive non-responsive bleeding)

Capsule endoscopy : small bowel bleeding (upper and lower endoscopy don't show the etiology)

Laboratory tests	• Stool guaiac for occult blood • Hemoglobin/hematocrit level (may not be decreased in acute bleeds): A **hemoglobin level >7 to 8 g/dL is generally acceptable in young, healthy patients** without active bleeding. However, most elderly patients (especially those with cardiac disease) should have a hemoglobin level >10 g/dL. • A low mean corpuscular volume is suggestive of iron deficiency anemia (chronic blood loss). Patients with acute bleeding have normocytic red blood cells. • Coagulation profile (platelet count, PT, PTT, INR) • LFTs, renal function • The BUN–creatinine ratio is elevated with upper GI bleeding. This is suggestive of upper GI bleeding if patient has no renal insufficiency. The higher the ratio, the more likely the bleeding is from an upper GI source.
Upper endoscopy	• **Most accurate diagnostic test in evaluation of upper GI bleeding** • Both diagnostic and potentially therapeutic (coagulate bleeding vessel) • Most patients with upper GI bleeding should have upper endoscopy **within 24 hours**.
Nasogastric tube	• This is often the initial procedure for determining whether GI bleeding is from an upper or lower GI source. • Use the nasogastric tube to empty the stomach to prevent aspiration. • **False-negative findings**: possible if upper GI bleeding is intermittent or from a lesion in the duodenum. • Evaluation of aspirate ➔ Bile but no blood—upper GI bleeding unlikely; source is probably distal to ligament of Treitz ➔ Bright red blood or "coffee grounds" appearance—upper GI bleeding ➔ Non-bloody aspirate (clear gastric fluid)—upper GI bleeding unlikely, but cannot be ruled out definitively (source may possibly be in the duodenum).
Anoscopy or proctosigmoidoscopy	Can exclude an anal/rectal source. Perform this if there is no obvious bleeding from hemorrhoids.
Colonoscopy	Identifies the site of the lower GI bleed in >70% of cases, and can also be therapeutic.
A bleeding scan (radionuclide scanning)	Reveals bleeding even with a low rate of blood loss. It does not localize the lesion; it only identifies continued bleeding. Its role is controversial, but it may help determine whether arteriography is needed.
Arteriography	Definitively locates the point of bleeding. ➔ Mostly used in patients with lower GI bleeding ➔ Should be performed during active bleeding ➔ Potentially therapeutic (embolization or intra-arterial vasopressin infusion).
Exploratory laparotomy	Last resort.

❖ Clinical approach to the patient :

A 69-year-old woman comes to the ER with multiple red/black stools over the last day. Her past medical history is significant for aortic stenosis. Her pulse is 115 per minute and her BP is 94/62 mm Hg. The physical examination is otherwise normal. What is the most appropriate next step in the management of this patient?

A. Colonoscopy
B. NGT
C. Upper endoscopy
D. Bolus of normal saline
E. CBC
F. Bolus of 5% dextrose in water
G. Consult gastroenterology
H. Check for orthostasis

Answer: D. The precise etiology of severe GI bleeding is not as important as a fluid resuscitation. There is no point in checking for orthostasis with the person's systolic BP under 100 mm Hg or when there is a tachycardia at rest. Endoscopy should be performed, but it is not as important to do first as fluid resuscitation. When BP is low, **normal saline or Ringer lactate** are better fluids to give than 5% dextrose in water (D5W). D5W does not stay in the vascular space to raise BP as well as NS.

Management: ..

Initial assessment

1. Risk assessment tools (Increased risk of further bleeding and death)

- ➔ Glasgow - blatchford score
- ➔ Rockall score
- ➔ **AIMS65 score**; (Albumin < 3.0, INR >1.5 , mental status, systolic BP < 90 and Age > 65)

AIMS65 Score

Risk Factor	Points
Albumin <3.0 g/dL	1
INR >1.5	1
Altered mental status	1
SBP ≤90 mm Hg	1
Age >65 y	1

Factors that increase mortality in GI bleeding:

- Age > 60 years
- Extensive comorbid illness
- Tachycardia (heart rate more than or equal to 100 beats/minute)
- Hypotension (Systolic BP less than or equal to 100 mmHg)
- Severity of initial bleed
- Onset or recurrence of bleeding while hospitalized for another condition
- Need for emergency surgery
- Significant transfusion requirements
- Diagnosis (Esophageal varices have a 30% mortality rate)
- Endoscopic stigmata of recent hemorrhage

2. Resuscitation **(If patient is hemodynamically unstable resuscitation is always top priority. Once the patient is stabilized obtain a diagnosis)**

a. **Supplemental oxygen**
b. **Hemodynamic status**

Adequate venous access, 2 large-bore peripheral venous lines (16 or 18 gauge). Isotonic intravenous fluids (Normal saline solution) for patients with evidence of hemodynamic instability. A bolus of 500 mL of IV isotonic fluid should be given and repeated as necessary to achieve hemodynamic stability. At the same time draw blood for hemoglobin and hematocrit, PT, PTT and platelet count.

- ➔ Packed red blood cells: If the hemoglobin level < 7 g/dL or If hemoglobin < 10 g/dL in patients with preexisting cardiovascular disease or patients with symptoms.
- ➔ Fresh frozen plasma: if PT or INR is elevated

Patients receiving anticoagulants correction of coagulopathy is recommended نعدلها كأن المريض ما ياخذ أدوية مسيلة للدم!

- ➔ Platelets: if the count is below 50,000

80% of GI bleeding will stop spontaneously if the fluid resuscitation is adequate and only need supportive therapy

Endoscopy The time of endoscopy is not significant in decreasing the mortality. Most IMPORTANT thing is to stabilize the patient.

- Within 24 hours after appropriate resuscitation and transfusion as needed, to a hemoglobin level greater than 7 g/dL
- Endoscopy may need to be delayed or deferred :
  1. Active acute coronary syndrome
  2. Suspected perforation (X-ray to exclude perforation)
- Within 12 hours in patients with high clinical risk may be associated with improved outcomes :
  1. Glasgow-blatchford score >=12
  2. Bloody nasogastric aspirate
  3. Hypotension
  4. Tachycardia
- In high-risk endoscopic findings > give IV PPI bolus (at a dose of 80 mg) followed by a continuous infusion (8 mg per hour) for 72 hours. This will reduce the risk of further bleeding and the need for surgery.
- If bleeding recurs after first scope repeat endoscopy if failed again Transarterial therapy (Injections, Clipping, Thermal therapy or powder spray) or surgery. Nobody knows about the mechanism of the powder. Also, it's not FDA approved drug.

Prevent recurrence

- Eradication of H.pylori infection and confirm eradication after therapy with breath test or stool test. Stop PPI for at least 2 weeks. Stop bismuth or antibiotics for at least 4 weeks. H2-receptor antagonists are permissible .
- Discontinue NSAIDs permanently if possible. If must be resumed a combination of COX-2 selective NSAID and PPI.
- For idiopathic ulcers continue on PPI daily

Management of Esophageal varices:

1. **Diet and exercise (shown to decrease Hepatic venous pressure gradient measurement)**
2. **Alcohol abstinence**
3. **Statins (decrease fibrogenesis, improve liver microcirculation and decrease portal pressure)**
4. **Depends on stage**

➔ **Compensated cirrhosis without clinically significant portal HTN:**
Directed toward the etiology of cirrhosis and anti-fibrogenic therapies

➔ **Compensated cirrhosis with clinically significant portal HTN who has not yet developed varices:** Treat the underlying cause

➔ **Cirrhosis and clinically significant portal HTN with gastroesophageal varices who has never bled:**

1. **Patients with medium/large varices:** Beta blockers (Propranolol, nadolol) or Endoscopic variceal ligation
2. **Patients with high-risk small varices (red wale marks** an endoscopic sign suggestive of recent hemorrhage, or propensity to bleed**):** Beta blockers
3. **Patients with small varices without signs of increased risk:** Beta blockers

➔ **Cirrhosis who presents with acute variceal hemorrhage:**

1. Airway, Breathing, and Circulation are followed to achieve hemodynamic stability
2. PRBC transfusion to reach hgb level of 7 to 8 g/dL
3. Antibiotic (ceftriaxone or norfloxacin) prophylaxis to decrease infection, early recurrence of hemorrhage and death
4. Vasoactive drugs
   - Somatostatin / Octreotide[4] / Terilipressin[5]
   - Continued for up to 5 days
   - Discontinued once the patient has been free of bleeding for at least 24 hours
5. Endoscopy (ASAP and not more than 12 hours after presentation)
   - Endoscopic variceal ligation
   - If all fails > Transjugular intrahepatic portosystemic shunt

➔ **Decompensated cirrhosis who has recovered from an episode of variceal hemorrhage:**

Beta blockers should be used cautiously in patients with refractory ascites

Dose reduced/Discontinued if :

1. Systolic blood pressure < 90 mmHg
2. Serum sodium < 130 mEq/L
3. AKI develops

[4] Octreotide is a somatostatin analogue
[5] Terilipressin is a vasopressin analogue

Cases

1. You see a 75-year-old man with an acute episode of haematemesis, who was admitted the night before and is awaiting an upper GI endoscopy. You are asked on the ward round about the common causes of upper GI bleeding. From the list below, which of the following is the most common cause of upper GI bleeding?

A. Mallory–Weiss tear
B. Peptic ulcers
C. Oesophageal varices
D. Drug induced
E. Malignancy

2. A 60-year-old man with alcoholic liver disease was admitted with an upper GI bleed secondary to oesophageal varices. The patient undergoes endoscopic variceal banding and is discharged after 2 weeks in-hospital stay. Which of the following medications would act as prophylaxis in preventing a rebleed from his oesophageal varices?

A. Frusemide
B. Amlodipine
C. Ramipril
D. Propranolol
E. Irbesartan

3. A 55-year-old woman is referred by her GP for upper gastrointestinal (GI) endoscopy following a four-month history of epigastric pain despite treatment with antacids and proton pump inhibitors (PPIs). The results demonstrate a duodenal ulcer coupled with a positive campylobacter-like organism (CLO) test. The patient has no past medical history and has no known drug allergies. The most appropriate treatment is:

A.Seven-day course of twice daily omeprazole 20mg, 1g amoxicillin and 500 mg clarithromycin
B. even-day course of twice daily omeprazole 20 mg
C. Seven-day course of twice daily omeprazole 20mg and 1g amoxicillin
D. Seven-day course of twice daily omeprazole 20mg and 500mg clarithromycin
E. Seven-day course of twice daily 1g amoxicillin and 500mg clarithromycin

4. A 30-year-old male smoker presents to the emergency room complaining of chest pain and hematemesis, having vomited up two cups of blood. He admits to drinking too much that same evening and having vomited repeatedly after drinking shots of vodka with his friends following a sporting event. His chest pain is worse after each episode of vomiting; he has never had a cardiac problem in the past. His past history is important for only for hypertension controlled with hydrochlorothiazide. He denies any previous history of alcohol abuse. On examination he is anxious and diaphoretic. His supine pulse is 90, with a blood pressure of 110/90. Heart and lungs are normal, and he has mild epigastric tenderness. His hemoglobin is 11. Stool is hemoccult positive. EKG and initial cardiac enzymes are normal. You admit the patient to the intensive care unit and consult a gastroenterologist. What is the most likely outcome of this patients gastrointestinal bleeding?

1. Spontaneous resolution of the acute upper GI bleeding within 24 to 48 hours
2. Recurrent massive upper GI bleeding within a few hours
3. Continued slow bleeding
4. Mental status deterioration within a few hours
5. Development of fever and intense right lower quadrant pain within a few hours

Answers

1. **B.** Approximately 80 percent of upper GI bleeds have known identifiable causes, some of which include:

- peptic ulcers – approximately 35–50 per cent of bleeds (B)
- Mallory–Weiss tears – 15 percent (A)
- oesophagitis – 5–15 per cent
- gastritis and gastric erosions – 5–15 per cent
- oesophageal varices – 5–10 per cent (C)
- drugs (e.g. NSAIDs, steroids, anticoagulants) – 5 per cent (D)
- upper GI malignancy – 5 per cent (E)
- rarer causes (<5 per cent) :
    - Dieulafoy's lesion
    - angiodysplasia
    - haemobilia
    - Aorto-enteric fistula

2. **D.** Oesophageal varices arise as a result of portal hypertension (>10mmHg) which leads to dilated collateral veins at sites of portosystemic anastomsis (e.g. the lower oesophagus). The causes of portal hypertension can be divided into: (1) Pre-hepatic: portal-vein thrombosis, splenic vein thrombosis; (2) Hepatic: cirrhosis (accounts for 80 per cent of causes of portal hypertension), shistosomiasis (most common cause worldwide), sarcoidosis, myeloproliferative disease, congenital hepatic fibrosis; and (3) Post-hepatic: Budd–Chiari syndrome, right heart failure, constrictive pericarditis, veno-occlusive disease. Once portal pressures are >12 mmHg, variceal bleeding may develop. Prophylaxis for the prevention of variceal bleeding can be divided into: (1) Primary: non-selective β-blockade (e.g. propranolol) and/or endoscopic banding ligation; (2) Secondary (i.e. after an initial variceal bleed: non-selective β-blockade, endoscopic banding ligation, transjugular intrahepatic portosystemic shunting (TIPPS) for varices resistant to banding or surgical shunts if TIPPS is not possible. Therefore, (D) is the most appropriate medication for prophylaxis in the prevention of variceal rebleeding.

3. **A.** This patient has been diagnosed with a duodenal ulcer secondary to *H. pylori* infection. The CLO test (also known as the rapid urease test) is positive, confirming the presence of the bacterium. A 7-day course of 'triple therapy' (PPI + two antibiotics) is recommended for patients with duodenal ulcers positive for *H. pylori*. The eradication therapy regimen is based on twice daily dosing and, as well as aiming to clear the *H. pylori* infection with the antibiotics, the PPI is used to enhance the healing of the ulcer. Therefore in this scenario, (A) is the most appropriate from the list. For patients who are allergic to penicillin, clarithromycin and metronidazole can be used instead.

4. **A.** This patient has a Mallory-Weiss tear, which is the cause of bleeding in approximately 5% of patients with an acute upper GI bleed. Most of these tears heal spontaneously within 24 to 48 hours with supportive therapy. If there is ongoing bleeding, IV vasopressin or injection of a sclerotic agent via endoscopy may be required. Surgical intervention with oversewing of the bleeder is rarely needed. The history is not suggestive of chronic alcoholism which may be associated with esophageal varices and hence a higher risk of recurrent massive bleeding as well as mental status deterioration. Acute appendicitis rarely presents with UGI bleeding.