GASTROINTESTINAL HEMORRHAGE

Anatomy

Bleeding can occur anywhere along the gastrointestinal (GI) tract from the oropharynx to the anus. Bleeding is the initial presentation in 1/3 of patients with gastrointestinal pathology, and the majority of GI bleeding cases stop spontaneously. Knowledge of the GI tract anatomy and blood supply is critical in locating and treating any GI bleed.

Upper GI Tract

Bleeding from the upper GI tract occurs anywhere between the oropharynx and ligament of Treitz which delineates the transition between the duodenum (foregut) and the jejunum (midgut). This encompasses the oral cavity, esophagus, stomach (fundus, cardia, body, and pyloric region) as well as the entirety of the duodenum. The duodenum is composed of 4 portions: the superior or duodenal bulb, descending, inferior, and ascending, termed 1-4 respectively.

The blood supply to the upper GI tract arises from the celiac trunk and includes the left gastric artery which supplies the cardia and lesser curve of the stomach, the splenic artery which has a tortuous course behind the stomach and gives rise to the short gastric arteries, as well as the left gastroepiploic artery on the greater curve of the stomach. The right gastric artery and gastroduodenal artery (GDA) both have their origin from the common hepatic artery which arises from the celiac trunk. The GDA passes just distal to the pylorus and posterior to the duodenum and splits into the anterior and posterior superior pancreaticoduodenal arteries as well as the right gastroepiploic artery along the greater curvature of the stomach.

Duodenal ulcers located on the posterior wall are more common than those found on the anterior wall. Posterior ulcers are more likely to erode through intestinal wall into branches of the GDA, resulting in massive bleeding. Ulcers located on the anterior side of the duodenal wall are more likely to perforate, and therefore, present with free air and peritonitis.

Lower GI Tract

The lower GI tract extends from the ligament of Treitz to the anus. Thus, the lower GI tract begins at the jejunum and includes the majority of the 600cm or 20 feet of the small bowel, colon, rectum, and anus. The ileocecal valve is located at the transition between small bowel and large bowel, or colon. The colon consists of cecum, ascending, hepatic flexure, transverse, splenic flexure, descending, and sigmoid before reaching the rectum. The transition between colon and rectum can be identified by fusion of the tenia of the sigmoid colon to form the circumferential longitudinal muscle of the rectum. This fusion is located about 12-15cm above the dentate line. Additionally, the rectum is covered anteriorly by the peritoneum on the middle and upper 2/3s. There is no peritoneal covering on the lower third of the rectum.

Important blood supply to the lower GI tract comes from the superior mesenteric artery which supplies the small bowel as well as the cecum, ascending, and proximal transverse colon via the ileocolic, right, and middle colic branches. The superior mesenteric vein drains the right side of the colon, joining the splenic vein to form the portal vein. The inferior mesenteric artery supplies blood to the distal transverse, descending, and sigmoid colon. The inferior mesenteric vein carries blood from the left side of the colon to the splenic vein. A rich network of vessels from the superior, middle, and inferior hemorrhoidal vessels supplies the rectosigmoid junction and rectum.

Definitions

Hematemesis Is defined as vomiting of blood and suggests an upper GI source. Bright red blood or clots indicates rapid bleeding while “coffee-ground” emesis signifies a slower, chronic bleed.

Melena is the passage of black, tarry stool and suggests an upper GI source. The source is likely distal to ligament of Treitz if not accompanied by hematemesis. The dark appearance is due to digested blood, and only 50-60cc of blood is required to cause melanotic stools and it can persist 5-7 days after the initial bleed.

Hematochezia is the passage of maroon blood and clots per rectum.

Occult bleeding is not visible to the patient or physician and is identified on stool guaiac testing for occult blood.

Pathophysiology

The first step in the diagnosis and treatment of GI bleed is to distinguish between an upper and lower source. Upper GI bleeds are more common than lower GI bleeds and account for about 70% and 30%, respectively, and patients over 60 years old represent about 60% of patients presenting with an upper GI bleed. Mortality in this older age group is as high as 20-25% but is much lower in younger patients at about 4%. Common etiologies and pathologies leading to both upper and lower GI bleeds are outlined below.

UPPER GI BLEED

Peptic Ulcers

Gastric and duodenal ulcers are the most common cause of upper GI bleeding and occur in 50-70% of patients. However, bleeding is the presenting symptom in only 10% of patients with peptic ulcers. Bleeding from duodenal ulcers is four times more common than from gastric ulcers. As described above, posterior duodenal ulcers are the most likely to bleed based on proximity to branches of the GDA. Significant bleeding occurs in 10-15% of peptic ulcers while 20% of these require surgical therapy for control.

Infection with the bacterium Helicobacter pylori (H. pylori) is the most common cause of peptic ulcers. These bacteria are known to colonize >50% of the population, with ultimately 10-20% of colonized individuals becoming symptomatic and developing ulcers. Chronic, slower bleeds tend to be associated with H. pylori. Another notable cause of peptic ulcers is chronic use of over the counter medications such as aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin, ibuprofen, and naproxen.

Medical therapy including H2 blockers and proton pump inhibitors for acid suppression has drastically improved the treatment of peptic ulcers. However, these patients can experience rebound with an increase in acid secretion with sudden cessation of medical therapy making medication adherence an important piece of information to obtain through history questions.

Stress Ulcers

These acute gastroduodenal lesions occur secondary to episodes of shock, sepsis, surgery, trauma, burns, or intracranial pathology. Curling's ulcers occur after burns, while Cushing's ulcers occur after intracranial processes. Risk factors for stress ulcers include multisystem trauma, hypotension, respiratory failure, sepsis, and jaundice.

Stress ulcers are possibly due to bile reflux causing damage to the gastric protective barrier along with decreased blood flow secondary to splanchnic vasoconstriction. Sepsis, coagulopathy, and activation of cytokines may also contribute. Patients with the afore-mentioned risk factors are often given stress ulcer chemoprophylaxis with an H2 blocker or proton-pump inhibitor, thereby, reducing the incidence of stress ulcers.

Esophageal and Gastric Varices

Varices are extremely dilated veins in the submucosa due to portal hypertension. Variceal hemorrhage is precipitated by ulceration of the varix due to reflux esophagitis or increased pressure within the varix.

Varices account for about 10% of upper GI bleeds in patients with liver disease. However, variceal hemorrhage accounts for 50-75% of upper GI bleeds in patients with advanced disease consisting of cirrhosis and portal hypertension. These bleeds are often life threatening. Patients with liver disease have a diminished ability to synthesize clotting factors increasing the risk of complication from bleeding. Knowing the status of liver disease helps direct therapy in these patients.

Erosive Gastritis

Diffuse gastritis causes 1/3 of all upper GI bleeds. Erosions are typically multiple and found primarily in the fundus and body of the stomach. Brisk bleeds are more likely due to NSAIDs, alcohol, or steroids which are harmful to the gastric mucosa. Chronic, slower bleeds are associated with H. pylori.

Mallory Weiss Syndrome

Mallory-Weiss syndrome is caused by prolonged or severe retching which results in a partial thickness mucosal tear at the gastroesophageal junction. This is in contrast to Boerhaave syndrome in which a full thickness tear results in mediastinitis. Mallory-Weiss tears account for about 5-10% of all upper GI bleeds.

Classically, patients will present with an episode of vomiting without blood. Further emesis leads to pain and the development of hematemesis due to a tear in the esophagogastric mucosa. Bleeding due to a Mallory-Weiss tear resolves spontaneously 90% of the time with no further intervention required. Endoscopic therapy with epinephrine injection or balloon tamponade may be necessary in the other 10% that don't resolve spontaneously.

Reflux Esophagitis

Esophagitis is more likely to result in chronic occult bleeding associated with grade II-III esophagitis with friable mucosa and is an uncommon cause of acute upper GI bleeds. Significant bleeding in this area is more likely due to complications from paraesophageal hernias.

Dieulafoy's Vascular Malformations

Dieulafoy's lesions are large, tortuous arterioles found within the stomach in the submucosa. The lesions are typically located in the fundus and body of the stomach along the lesser curvature. These dilated arterial lesions, up to 5mm, are uncommon accounting for less than 5% of all upper GI bleeds. Bleeding from Dieulafoy's lesions is due to a defect in the gastric mucosa likely due to the pressure from the underlying protruding, pulsatile arteriole.

Aortoenteric Fistulas

Aortoenteric fistulas are also uncommon causes of GI bleed, however, they are life threatening. In patients with prior aortic reconstructions, aortoenteric fistulas result from erosion of a prosthetic graft into nearby bowel typically in the setting of a perigraft infection. If no prior intervention occurred, the bleed is more likely from compression of an abdominal aortic aneurysm against the bowel. Patients typically present with a herald bleed which stops spontaneously followed by a massive bleed resulting in rapid hemodynamic deterioration requiring immediate intervention.

Gastric Neoplasms

Both malignant and benign lesions can cause upper GI bleeding. Bleeding from neoplasms is typically mild and chronic, commonly presenting with symptoms of anemia. These masses are usually diagnosed with endoscopy and biopsy.

LOWER GI BLEED

Small bowel

Pathology within the small bowel accounts for 10-15% of all lower GI bleeds. Small bowel bleeds are the most difficult to locate and diagnose. A list of common pathologies can be seen below in **Table 1**.

Colon

Colonic bleeds are the most common form of lower GI bleeds. Neoplastic disease can be present anywhere in the colon. Right sided lesions are typically associated with anemia and guaiac positive stools while left sided lesions present with obstructive symptoms. Similarly, ulcerative colitis leads to more chronic bloody diarrhea. However, massive bleeds may also occur. Diverticula and angiodysplastic lesions usually result in larger, more brisk bleeding.

Angiodysplastic lesions are degenerative as opposed to congenital or neoplastic and, therefore, increase with age. They are not associated with other vascular lesions. Angiodysplastic lesions are usually diagnosed by colonoscopy and are <5mm in size. Bleeding from these lesions spontaneously resolves in 80% of cases, although 50% will re-bleed within 3 years. A list of colonic causes of lower GI bleed can be seen below in **Table 1**.

Rectum and Anus

Rectal and anal bleeding present with fresh, red blood on the exterior of stool commonly called streaking. This is most likely associated with hemorrhoids, anal fissures, or proctitis. Additionally, patients may notice active bleeding into the toilet which is most commonly due to hemorrhoids or anal fissures. Hemorrhoid bleeding is often painless, while bleeding associated with anal fissures is quite painful.

Table 1

Lower GI Bleed Pathology		
Small Bowel	Colon	Rectum and Anus
Meckel's diverticulum Crohn's disease Neoplasm Vascular malformations Intestinal varices Non-Meckel's diverticulum Mesenteric thrombosis Mesenteric ischemia Drug reactions Enteric infections Polyps	Neoplastic disease Polyps Diverticula Angiodysplastic lesions Ulcerative colitis Infectious diarrhea	Proctitis Hemorrhoids Fissures

PEDIATRIC GI BLEED

Most common causes of GI bleeding in the pediatric population include Meckel's diverticulum and intussusception. Other UGI causes in this age group include gastric and duodenal ulcers, esophagitis, gastritis, varices, hereditary hemorrhagic telangiectasia, Ehlers-Danlos syndrome, and congenital or acquired coagulopathies. Lower GI bleeding can be due to juvenile polyps, vascular malformations, gastrointestinal duplication cysts, allergic colitis, infectious colitis, and lymphonodular hyperplasia.

Newborns with GI bleeding have a different set of potential diagnoses including swallowed maternal blood, necrotizing enterocolitis, malrotation with midgut volvulus, and Hirschsprung disease.

Diagnosis

Diagnosing a GI bleed is typically straightforward based on the patient presentation. In order to further identify the location and cause of the bleed within the GI tract, it is extremely important to obtain a thorough history and perform a complete physical examination.

HISTORY

The history of present illness should elicit details about the characterization of stool or emesis. This includes bright red versus dark, tarry stools or emesis and any alleviating or exacerbating factors. For instance, duodenal ulcers produce pain several hours after eating which is alleviated by further PO intake. Pain from gastric ulcers is typically exacerbated by eating. The length of symptoms and temporal sequence of events is also important, for instance, in the case of a Mallory-Weiss tear, non-bloody retching precedes bloody emesis.

Associated symptoms such as diaphoresis, dyspnea, lightheadedness, and orthostasis should be obtained. These constitutional symptoms are suggestive of anemia in slower, more chronic bleeds, or hypovolemia in larger, more brisk GI bleeding. Recent weight loss could indicate food aversion due to an ulcer or malignancy.

A review of systems should be thorough and identify any risk factors that could complicate an invasive procedure or surgery. This includes, but is not limited to, signs of active infection, angina, dyspnea, and poor exercise tolerance. A pertinent past medical history should inquire about peptic ulcer disease, cirrhosis, heartburn, and reflux. Past surgical history, including any abdominal surgeries and endoscopies, should be obtained. Review medications in particular NSAIDS and steroids including how much, how frequently, and for how long the patient has been taking these medications. Inquire specifically about medication adherence to identify possible rebound acid secretion. Obtain a relevant family history including colon cancer, inflammatory bowel disease, and any GI malignancies. A social history should include current and past alcohol consumption and smoking status. Additionally, sick contacts and recent travel outside of the country should be elicited.

PHYSICAL EXAMINATION

Physical exam should include up-to-date vital signs and a general assessment of the patient. This will help determine how sick or critical the patient is upon presentation. Hemodynamic instability with tachycardia (occurs first) or hypotension (occurs second) indicates a more urgent situation. Keep in mind older patients on beta blockade may not be able to mount a tachycardic response to massive GI bleeding. Resuscitation should begin immediately for unstable patients while further history and exam is being completed.

Always look for signs of anemia or dehydration including pallor, lethargy, dry mucous membranes, skin tenting, or flat neck veins. A thorough abdominal exam should be performed to locate any tenderness, rebound, guarding, or other signs of peritonitis. Pay attention to signs of cirrhosis including spider angiomata, prominent abdominal veins, caput medusa, and ascites. A rectal exam MUST be performed in patients presenting with a GI bleed in order to identify perianal causes of bleeding in addition to the presence of blood in the rectal vault. Perform an occult blood test at time of rectal exam. In women, include a pelvic examination.

LABORATORY TESTS

Following history and physical exam, laboratory tests should be the next step in the work-up for a GI bleed. Laboratory investigations should include CBC, CMP, coagulation studies, type and cross, occult blood test, and possibly an ABG if shock is suspected.

The CBC will reveal derangements in hemoglobin and hematocrit as well as potential platelet deficiency in patients with liver disease. A large drop in hgb/hct would not be expected immediately in an acute bleed until resuscitation is initiated and hemodilution occurs. Chronic GI bleeding will result in an iron deficiency anemia.

The CMP can reveal hepatic dysfunction and the renal status of the patient. A proportional elevation in BUN:Creatinine ratio can be a sign of prerenal azotemia. Isolated elevated of BUN can be the result of blood digestion and absorption of breakdown products in the GI tract. A ratio greater than 36:1 likely represents bleeding from an upper GI source. The BUN can be elevated as high as 30-50 mg/dL.

IMAGING STUDIES

Ideal imaging studies in the setting of a GI bleed include modalities that are also therapeutic. Therefore, imaging will be discussed further below under Management.

Management

ACUTE MANAGEMENT

Management should begin with the ABCs (airway, breathing, circulation). The airway needs to be secured if sensorium is altered or the patient is unable to protect the airway. Simultaneously, two large bore IVs (16 gauge or larger) should be placed for access. An

arterial line can be placed in those patients with deteriorating clinical status for dynamic blood pressure monitoring.

Fluid resuscitation should start with a 1L bolus of crystalloids, either NS or LR. If the patient responds well with improvement in hemodynamic parameters, a second 1L crystalloid bolus can be administered. If the patient remains unstable with a suspected GI bleed, resuscitation should be continued with packed RBCs.

Fluid balance should be monitored with strict ins and outs and a Foley catheter may be placed for monitoring urine output. An NGT should also be placed and gastric lavage performed. Bloody return indicates a gastric or upper GI bleed.

DIAGNOSTIC AND THERAPEUTIC INTERVENTIONS

In order to appropriately manage GI bleeding, it is important to identify the source of bleeding. Many GI bleeds can be managed with medical management, or with the help of endoscopy and interventional radiology. Whenever possible, it is important to identify a source of bleeding even if surgery is indicated.

Endoscopy

Endoscopy is typically the next step as it is both diagnostic and therapeutic. Esophagogastro-duodenoscopy (EGD) evaluates the esophagus, stomach, and duodenum. Several different interventions can be performed during EGD to control the bleeding. These include direct pressure, injection with vasoconstrictive properties (epinephrine, vasopressin), sclerotherapy, electrocautery, ligation, and clipping. Of note, ligation has been found to be as effective as sclerotherapy but with fewer complications and is practiced more commonly in the acute setting.

Failed therapy and re-bleeding occurs in 55% of patients found to have active pulsatile bleeding or oozing at the time of endoscopy. A nonbleeding, visible vessel has 43% risk of re-bleeding. Adherent clot carries a 22% risk of re-bleed, and a clean based ulcer has 0-5% chance of re-bleeding.

Endoscopic intervention in patients with known cirrhosis and liver failure needs to be well planned given the inability to synthesize clotting factors. For acute bleeding, initial therapy should include ligation and vasopressin. The use of a Sengstaken-Blakemore tube which provides balloon tamponade can help temporize bleeds in unstable patients or those in whom EGD has not been successful.

For lower GI bleeds, colonoscopy evaluates the rectum, colon, and distal ileum. The same interventions mentioned above are available during colonoscopic intervention. Anoscopy and proctoscopy can be done at the bedside or in the operating room accompanied by an exam under anesthesia (EUA).

Tagged red blood cell scan

If a source of bleeding cannot be identified by endoscopy, the next test is often a tagged RBC scan. This test is 91% sensitive and 100% specific for diagnosing a lower GI bleed. It can identify a bleed as slow as 0.005-0.1 mL/min compared to angiography at 0.5-1.0 mL/min. However, there must be active bleeding at the time of the scan in order to identify a bleed, and it is only about 50% active in identifying the precise location of the bleeding. The test will be positive wherever a critical mass of blood can be detected by the gamma camera, and for that reason, cannot be used in isolation to perform a segmental resection of the colon. However, it does provide very useful information for the interventional radiologist to target angiographic embolization.

Angiographic embolization

Angiography can be diagnostic and therapeutic. It can help localize a GI bleed where extravasation of contrast is seen, and can be used for coil embolization of the bleeding vessel. The risk of bowel necrosis is low with super selective embolization. However, the risk of renal failure related to dye load should be kept in mind, particularly in those patients who may be dehydrated or suffering from pre-existing renal compromise.

Transjugular intrahepatic portosystemic shunt (TIPS)

TIPS is used for bleeding secondary to portal hypertension that is not amenable to endoscopic intervention. It off-loads the pressure within the portal venous system. However, blood then bypasses the liver potentially leading to worsening of hepatic encephalopathy. In-hospital mortality of patients requiring an emergent TIPS procedure is high at about 35-55%. Stenosis or occlusion of the shunt occurs up to 50% of the time at one year. Therefore, patients may need a surgical portosystemic shunt, if their native liver function is reasonably good, or a liver transplant, if the functional reserve of the liver is critically low.

SURGICAL MANAGEMENT

Upper

Elective surgeries for gastric and duodenal ulcers have significantly decreased in frequency due to improved medical therapy with H2 blockers and proton pump inhibitors. However, the number of urgent or emergent surgeries for bleeding duodenal ulcers has remained somewhat stable.

Indications for surgical intervention include uncontrolled bleeding in a patient with a known ulcer after failure of endoscopic treatment of the bleed. Pre-operative preparation includes adequate fluid resuscitation with either crystalloid or blood pending the status of the patient.

The operative approach for an upper GI bleed is via an exploratory laparotomy through an upper midline incision. For a duodenal ulcer, dissection is carried out to expose the pylorus and first part of the duodenum. An anterior longitudinal duodenotomy is made extending

through the pyloric channel to the distal stomach. Bleeding from the GDA complex is controlled with a three-vessel ligation technique. This consists of a superior suture, inferior suture, and a horizontal mattress suture creating a “U” stitch for the transverse pancreatic artery. A Heineke-Mikulicz closure of the duodenotomy is then performed by closing the horizontal incision in a vertical fashion.

Bleeding gastric ulcers are best treated with surgical excision of the ulcer and repair of the remaining gastric defect. Given that 4-5% of benign appearing ulcers contain a malignancy, it is of utmost importance to send all gastric ulcers specimens for pathologic evaluation.

A truncal vagotomy can be added to the operation for long-term ulcer control. However, this is not appropriate in an unstable or under resuscitated patient. Additionally, this should only be performed if the patient was on adequate medical therapy prior to surgery. If a truncal vagotomy is performed, a 1 cm portion of each vagus nerve (anterior and posterior) is resected. It is necessary to send both of the vagal trunk specimens to pathology to document the vagotomy was performed successfully.

Unlike bleeding ulcers, operative therapy is first line treatment for an ulcer with an associated perforation. The operation of choice for a duodenal perforation is the Graham patch repair. In this maneuver, a portion of the omentum is placed over the perforation and is secured in place with interrupted silk sutures. The sutures should be placed quite wide of the ulceration to prevent tearing through the friable tissue. Perforated gastric ulcers can also be treated with a Graham patch or excision with repair of the defect as done for a bleeding duodenal ulcer. Again, the specimen of the gastric ulcer should be sent for pathology to rule out a malignancy.

Postoperatively, patients should be treated with acid suppression therapy and for H. Pylori infection, if positive. They should also be counseled on peptic ulcer disease.

Lower

Every effort should be made to locate the source of bleeding within the lower GI tract. If a source is identified in the colon and other less invasive modalities have failed, a partial colectomy can be performed. If colonic bleeding persists and cannot be located with less invasive techniques, a total abdominal colectomy is the indicated operation. This is a morbid operation and associated with an ileostomy.

PREVENTION

Medication

NSAIDs should be avoided by patients with peptic ulcer disease. Additionally, the importance of aspirin and clopidogrel in patients with cardiovascular disease and bleeding from peptic ulcer disease should be discussed with the cardiologist to weigh the risks of bleeding versus the risk of a cardiac event.

Alcohol and Caffeine

Both alcohol and caffeine impair the ability to heal an already present peptic ulcer. Smoking cessation and moderation of alcohol should be recommended. Adequate resources should to be offered to help patients with these addictive behaviors.

H. pylori treatment

Eradication of H. pylori is essential to improve ulcer healing and reduce the risk of re-bleeding. H. pylori is treated with triple therapy including clarithromycin, a PPI, and amoxicillin or metronidazole or quadruple therapy consisting of bismuth, metronidazole, tetracycline, and a PPI. Effective treatment can be confirmed with a biopsy urease test, urea breath test, or a stool antigen test. Repeat EGD should also be performed in 8-12 weeks after an upper GI bleed to evaluate healing and perform biopsies to exclude malignancy.

Questions

1. Compared to the anterior wall, ulcers located on the posterior wall of the duodenum are more likely to:
   A. Contain H. pylori
   B. Bleed
   C. Perforate
   D. Obstruct

2. Identify the most common location and etiology of gastrointestinal bleeding.
   A. Lower, colon cancer
   B. Lower, diverticulosis
   C. Upper, gastric cancer
   D. Upper, peptic ulcer disease

3. What is the first step in the management of a gastrointestinal bleed?
   A. Exploratory laparotomy
   B. Esophagogastroduodenoscopy
   C. Volume resuscitation
   D. Acid suppression with H2 blocker

Answers

1. Compared to the anterior wall, ulcers located on the posterior wall of the duodenum are more likely to:
   A. Contain H. pylori
   B. Bleed
   C. Perforate
   D. Obstruct

   The posterior wall of the duodenum is in close proximity to the gastroduodenal artery (GDA). Therefore, ulcers in this location are more likely to bleed. Ulcers located on the anterior duodenal wall are more likely to perforate. These patients with perforation present with peritoneal signs and typically require a trip to the operating room.

2. Identify the most common location and etiology of gastrointestinal bleeding.
   A. Lower, colon cancer
   B. Lower, diverticulosis
   C. Upper, gastric cancer
   D. Upper, peptic ulcer disease

   Upper gastrointestinal bleeding is more common than lower gastrointestinal bleeding, 70% and 30% respectively. The upper GI tract reaches from the oropharynx to the Ligament of Treitz. The most common cause of upper GI bleeding is peptic ulcer disease.

3. What is the first step in the management of a gastrointestinal bleed?
   A. Exploratory laparotomy
   B. Esophagogastroduodenoscopy
   C. Volume resuscitation
   D. Acid suppression with H2 blocker

   Volume resuscitation via large bore peripheral intravenous catheters is the first step in management of a patient with gastrointestinal bleeding. Once stabilized, further diagnostic imaging to characterize the bleed can be performed. Acid suppression is effective in the setting of peptic ulcer disease as the etiology.

Problems

For each of the following problems, answer the following questions:

- What further data should be obtained from the patient's history?
- What findings would you look for on physical exam?
- What is your differential diagnosis?
- What work-up would you recommend (include laboratory tests and diagnostic interventions)?
- What therapy or treatment would you recommend?

1. A 25-year-old, otherwise healthy, medical student presents with acute abdominal pain, nausea, vomiting, and bright red blood per rectum.

2. A 65-year-old man presents with hypotension and bright red blood and clots per rectum. Two months ago, he had a similar episode of massive bleeding for which he did not seek medical advice.

3. A 62-year-old woman is referred with chronic anemia.

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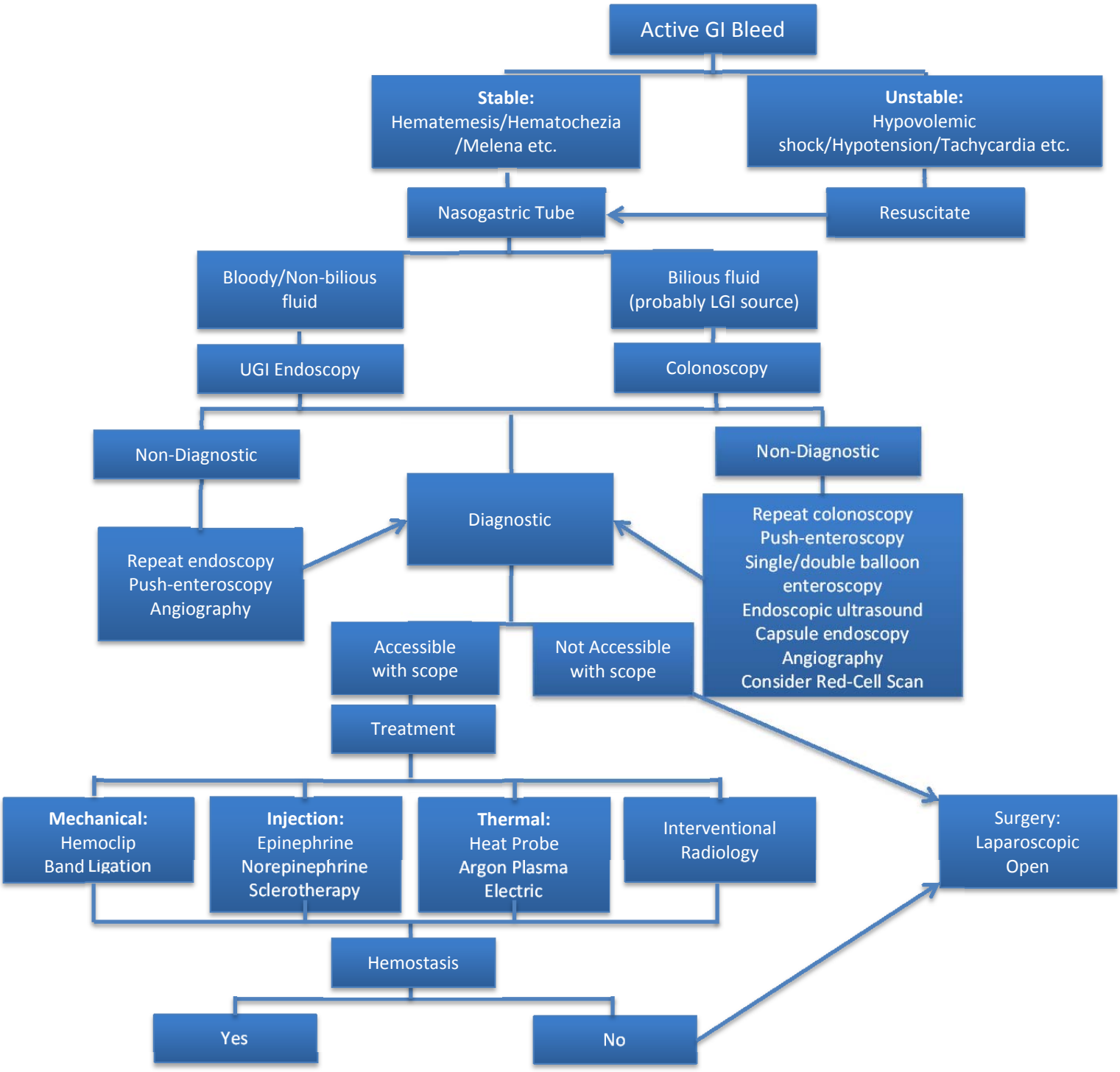
Figure


Active GI Bleed
Stable: Hematemesis/Hematochezia /Melena etc.
Unstable: Hypovolemic shock/Hypotension/Tachycardia etc.
Nasogastric Tube
Resuscitate
Bloody/Non-bilious fluid
Bilious fluid (probably LGI source)
UGI Endoscopy
Colonoscopy
Non-Diagnostic
Non-Diagnostic
Diagnostic
Repeat endoscopy
Push-enteroscopy
Angiography
Repeat colonoscopy
Push-enteroscopy
Single/double balloon enteroscopy
Endoscopic ultrasound
Capsule endoscopy
Angiography
Consider Red-Cell Scan
Accessible with scope
Not Accessible with scope
Treatment
Mechanical: Hemoclip Band Ligation
Injection: Epinephrine Norepinephrine Sclerotherapy
Thermal: Heat Probe Argon Plasma Electric
Interventional Radiology
Surgery: Laparoscopic Open
Hemostasis
Yes
No

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