HOSPITAL PHYSICIAN®

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Acute Gastrointestinal Bleeding

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Table of Contents

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INTRODUCTION

Acute gastrointestinal (GI) bleeding is defined as gross bleeding into the enteric tract. It is a common and potentially life-threatening condition, affecting between 50 and 100 persons per 100,000 in the United States each year. It can occur at all ages but is more prevalent in patients older than 50 years. Upper GI bleeding is more common than lower GI bleeding.

Emergency medicine physicians must be familiar with the current diagnostic modalities and treatment options available for GI bleeding. Hospital stay, morbidity, and number of transfusions can all be minimized by proper initial diagnosis and treatment. The following article discusses the pathophysiology of upper and lower GI bleeds and reviews the diagnosis and treatment of GI bleeding since the advent of improved endoscopy and pharmacotherapy.

APPROACH TO THE PATIENT

TYPICAL PRESENTATION

Patients with GI bleeds reliably present with gross hematemesis, melena, or hematochezia and usually have evidence of hemodynamic compromise. Hematemesis is gross bloody emesis; it indicates a recent or ongoing GI bleed. Coffee ground emesis contains gross blood that has been coagulated and oxidized by stomach acid and appears as "coffee grounds" floating in stomach aspirate. It usually indicates a significant bleed that has stopped. Melena is tarry black stool produced by gross blood oxidized in the enteric tract with a transit time of more than 8 hours. It may indicate bleeding that occurred up to 3 to 4 days prior. Hematochezia is maroon or bright red gross blood per rectum. It can be present in lower GI bleeding or in upper GI bleeding with rapid transit time.

HISTORY AND PHYSICAL EXAMINATION

When approaching a patient with a complaint of hematemesis or melena, one must first exclude epistaxis, pharyngeal bleeding, or hemoptysis. Once these have been ruled out, a careful history and physical examination should focus on locating the source of the bleeding.

Frequently, a careful history can pinpoint the etiology of the bleeding. Classically, hematemesis or coffee ground emesis suggests upper GI bleeding, and hematochezia indicates lower or distal GI bleeding; however, exceptions do occur. The patient should be questioned about alcohol use because alcohol is strongly associated with a number of causes of GI bleeding, and about drug ingestions, including aspirin, nonsteroidal anti-inflammatory drugs

(NSAIDs), steroids, cocaine, and anticoagulants. Iron and bismuth can simulate melena by causing stool to turn black, and certain food products can simulate hematochezia by causing red stools; however, results of fecal occult blood testing will be negative in both cases. Medications and food products that can cause false-positive and false-negative results on stool guaiac testing are shown in **Table 1**. Change of stool caliber and weight loss associated with signs of GI bleeding should raise suspicion of a malignancy.

LABORATORY EVALUATION

Order a rapid measurement of hemoglobin or a spun hematocrit, a complete blood count, a prothrombin time/partial thromboplastin time, and an electrolyte panel. Type and crossmatch for 2 to 4 units of packed red blood cells. Obtain an upright chest radiograph if perforation is suspected. Consider electrocardiogram for patients older than 55 years, as coronary ischemia can be a significant sequela to acute blood loss.

INITIAL MANAGEMENT

Aggressive resuscitation with continuous cardiac monitoring and appropriate and timely consultation are key to emergent management of GI bleeding. A nasogastric tube should be placed, and 2 large-bore intravenous access sites should be obtained. Warm isotonic fluids should be infused as needed for tachycardia, orthostatic hypotension, or other signs of hypovolemia. Patients should receive nothing by mouth. Definitive treatment is based on the etiology of the bleed. The algorithm **Figure 1** presents a general approach to acute GI bleeding.

UPPER GASTROINTESTINAL BLEEDING

The upper GI tract is the most common source of significant bleeding into the enteric lumen. Upper GI bleeding is defined as any bleeding proximal to the ligament of Treitz. The diagnosis, treatment, and prognosis of this condition have changed greatly over the past 20 years due to the advent and refinement of endoscopy, new pharmacotherapy agents, better understanding of the physiology of portal hypertension, and knowledge of the role of *Helicobacter pylori*.

PRESENTATION

The presentation of upper GI bleeding varies greatly, from simple tachycardia to profound shock, and patients may present with a variety of complaints. In a series of studies summarized by Peter et al, 40% to 50%

Table 1. Substances That Can Interfere with Tests for Fecal Occult Blood

May cause false-positive results:

Red meat (beef, lamb, and liver)

Aspirin (> 325 mg/day) and nonsteroidal anti-inflammatory drugs such as ibuprofen, indomethacin, and naproxen*

Corticosteroids, phenylbutazone, reserpine, anticoagulants, antimetabolites, and cancer chemotherapeutic drugs

Alcohol in excess

The application of antiseptic preparations containing iodine (povidone/iodine mixture)
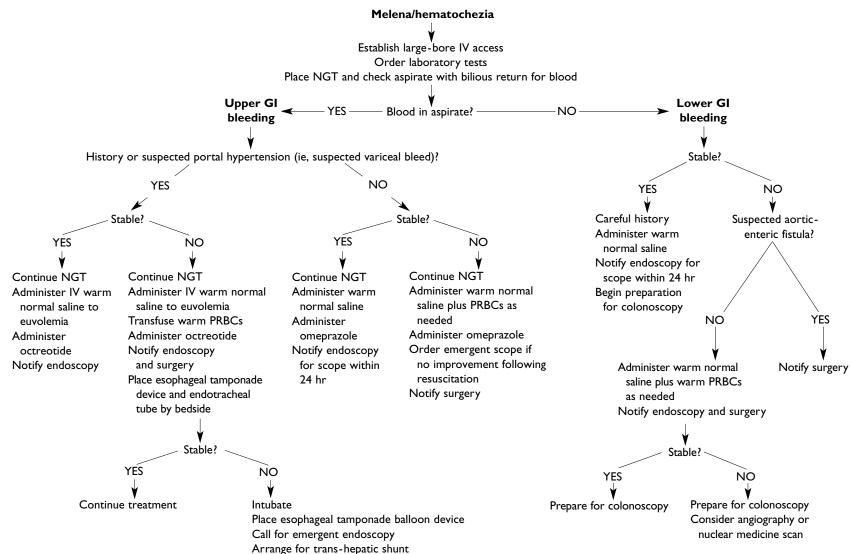
May cause false-negative results:

Ascorbic acid (vitamin C) in excess of 250/mg day

Excessive amounts of vitamin C enriched foods (citrus fruits and juices)

Iron supplements that contain quantities of vitamin C in excess of 250/mg day

*Acetaminophen is not expected to affect test results.

of patients with significant upper GI bleeding complained of hematemesis (including coffee ground emesis) and 70% to 80% complained of melena.[1] Melena or hematochezia is found on examination in 90% to 98% of patients with significant upper GI bleeding. Therefore, rectal examination should be done in all patients presenting with symptoms of GI bleeding. Melena can be produced by 50 to 100 mL of blood introduced experimentally into the stomach; hematochezia is usually apparent if more than 1000 mL are introduced.

Syncope and presyncope were the presenting complaints in 14.4% and 43% of patients with upper GI bleeding in 2 studies.[1,2] Jaundice was found only 5% of the time but was associated with an increased mortality of 38% from a baseline of 10%. Further history gathering may elicit symptoms that occurred in the preceding 30 days, including epigastric pain (41% of patients), dyspepsia (18%), heartburn (21%), and diffuse abdominal pain (10%).[1,3] Massive hemorrhage may be associated with symptoms of shock, heart rate greater than 100 bpm, systolic blood pressure less than 90 mm Hg, and altered mental status. Hemoglobin level less than 10 g/dL increases mortality from 10% to 18.9% and the occurrence of rebleeding from 11% to 23.3%.[3] Therefore, blood should be typed and crossmatched in preparation for transfusion in patients presenting with a low hemoglobin level, hypotension, or syncope. Other helpful laboratory tests include renal function tests and a

Melena/hematochezia

Establish large-bore IV access
Order laboratory tests
Place NGT and check aspirate with bilious return for blood

Upper GI bleeding ← YES — Blood in aspirate? — NO → **Lower GI bleeding**

History or suspected portal hypertension (ie, suspected variceal bleed)?

YES — Stable? — NO

Stable? (YES / NO)

YES:
Continue NGT
Administer IV warm normal saline to euvolemia
Administer octreotide
Notify endoscopy

NO:
Continue NGT
Administer IV warm normal saline to euvolemia
Transfuse warm PRBCs
Administer octreotide
Notify endoscopy and surgery
Place esophageal tamponade device and endotracheal tube by bedside

Stable?

YES → Continue treatment

NO → Intubate
Place esophageal tamponade balloon device
Call for emergent endoscopy
Arrange for trans-hepatic shunt

Stable? (YES / NO)

YES:
Continue NGT
Administer warm normal saline
Administer omeprazole
Notify endoscopy for scope within 24 hr

NO:
Continue NGT
Administer warm normal saline plus PRBCs as needed
Administer omeprazole
Order emergent scope if no improvement following resuscitation
Notify surgery

Lower GI bleeding

Stable?

YES:
Careful history
Administer warm normal saline
Notify endoscopy for scope within 24 hr
Begin preparation for colonoscopy

NO: Suspected aortic-enteric fistula?

NO → Administer warm normal saline plus warm PRBCs as needed
Notify endoscopy and surgery

Stable?

YES → Prepare for colonoscopy

NO → Prepare for colonoscopy
Consider angiography or nuclear medicine scan

YES → Notify surgery

Figure 1. Emergency management of acute gastrointestinal bleeding. GI = gastrointestinal; IV = intravenous; NGT = nasogastric tube; PRBCs = packed red blood cells; Scope = endoscopic procedure.

coagulation panel. A BUN-to-creatinine ratio greater than or equal to 36 without renal failure is suggestive of upper GI bleeding, whereas a lower ratio is not helpful.[1]

ETIOLOGY
Esophageal Varices

Esophageal varices are not the most common cause of upper GI bleeding, even in cirrhotic patients. However, they are considered the most potentially lethal source of upper GI bleeds. Actively bleeding varices are associated with a 35% to 50% mortality and 60% chance of rebleed within 24 hours.[4–6] Death from acute bleeding usually occurs from secondary injuries such as aspiration, acute respiratory distress syndrome, sepsis, or cardiac demise rather than exsanguination; thus, early recognition and aggressive treatment are essential.

Esophageal varices develop when the portal circulation is hypertensive (> 10 mm Hg) for a prolonged period. The portal circulation normally flows through a low-pressure, low-resistance path that can tolerate large fluctuations in volume (eg, postprandial blood flow can increase flow by 150% after a meal) and still maintain pressures below 8 mm Hg.[5] Any pathologic entity causing resistance along the pathway from gut to vena cava will lead to increased portal pressure and a shunting of the venous flow to collateral veins. The leading cause of portal hypertension in the United States is cirrhosis due to alcoholism and hepatitis C. Liver schistosomiasis is the etiologic agent of cirrhosis worldwide. Other causes include cardiac disease (congestive hepatitis), veno-occlusive disease, sarcoidosis, splenomegaly, and arteriovenous malformations. Varices inevitably develop in 30% to 70% of all cirrhotic patients. Variceal bleeding should be considered the cause of upper GI bleeding in all patients with a history of cirrhosis until proven otherwise because of the associated high morbidity.

Patients with variceal bleeding will present with high-volume hematemesis or melena. A history of conditions leading to cirrhosis, prior diagnosis of cirrhosis, or prior history of variceal bleeding usually can be elicited. Hemodynamic compromise is commonly evident. It is important to note that many cirrhotic patients are on β blockers for their portal hypertension and may not exhibit the expected tachycardia due to volume loss that typically occurs with upper GI bleeding.

Peptic Ulcer Disease

Peptic ulcer disease covers the spectrum of pathology from gastric and duodenal mucosal erosive lesions to diffuse gastritis and focal ulcerations. Peptic ulcer disease is the most common cause of upper GI bleeding (even in cirrhotic patients), accounting for 50% of all cases. The overall mortality of an acute bleed from peptic ulcer disease is 10%; this rate has remained unchanged for the past 20 years. The mortality and risk of rebleed increases significantly with comorbid disease and age greater than 60 years. For otherwise healthy patients younger than 60 years of age, the mortality associated with peptic ulcer disease drops to 1%.

Bleeding occurs from mucosal erosion into underlying submucosal vessels. Predisposing factors include aspirin use, NSAID use, alcoholism, and smoking. Calcium channel blockers have been implicated in one study.[7] Corticosteroids alone have not been shown to increase the risk of upper GI bleeding due to peptic ulcer disease, but these agents can double the NSAID-associated risk.[8] Any patient with a history of hematemesis, coffee ground emesis, or melena should be suspected of upper GI bleeding from peptic ulcer disease until proven otherwise. Peptic ulcer disease is the leading cause of upper GI bleeding in cirrhotic patients but carries a far lower mortality than esophageal variceal bleeding.

Eighty percent of cases of GI bleeding caused by peptic ulcer disease will stop spontaneously. Rebleeding within the first 48 hours significantly increases mortality. The risk of rebleeding is increased with comorbid disease, increasing age, and a presenting hemoglobin level less than 10 g/dL.[3,9]

Mallory-Weiss Tears

Mallory-Weiss tears are superficial mucosal tears into underlying vessels thought to be secondary to violent Valsalva or trauma. They are associated with alcohol use in up to 80% of patients. The most common scenario is an upper GI bleed following a retching episode, but Mallory-Weiss tears have been reported after coughing and seizures. Most tears stop bleeding spontaneously, heal within 3 days, and do not recur. Rarely, they can present as large-volume bleeds. The prognosis is worse in patients with portal hypertension.[10] Treatment is supportive care, with blood transfused as needed.

Other Causes

Dilefoy's arteries are blind-end arteries that arise from the submucosa, protrude into the gastric mucosa, and can bleed. Arteriovenous malformations associated with congenital vascular diseases and chronic renal failure can also cause upper GI bleeding. These miscellaneous etiologies are rare, but nonetheless are part of the differential diagnosis for upper GI bleeding.

MANAGEMENT
Emergency Intervention

A gastroenterologist should be consulted immediately

on presentation of a patient with upper GI bleeding. Emergency department (ED) personnel should wear protective coverings because upper GI bleeds, especially bleeding esophageal varices, are typically very messy. Two intravenous access sites should be obtained using 18-gauge or larger-bore catheters, and the patient should be resuscitated with warm saline to euvolemia. Warm fluids (40° to 41°C) help to prevent worsening coagulopathy associated with massive transfusions in patients who are already coagulopathic. In patients with variceal bleeding, avoid overhydration because it exacerbates the portal hypervolemia, which increases intravariceal pressure, resulting in further bleeding. A nasogastric tube should be placed to confirm upper GI bleeding and to monitor ongoing blood loss. Nasogastric aspirate should be considered negative only if there is return of bilious fluid that is negative on occult blood testing. Aspirate from the nasogastric tube is falsely negative in up to 15% of cases, so upper GI bleeding must still be suspected in patients who present with melena even if the nasogastric aspirate is negative. Consider early intubation in any patient who is in shock, has an altered mental status, is massively hemorrhaging, or may need balloon tamponade. Any patient presenting with shock, active hematemesis, or a hemoglobin level less than 10 g/dL should be typed and crossmatched for at least 2 units of blood. Patients with variceal bleeding should be typed and crossmatched for at least 4 units of packed red blood cells. Consider a transfusion with fresh frozen plasma or cryoprecipitate in massive exsanguination situations. Other requisite laboratory tests include a measure of lactate level to assess for subclinical shock as patients on β blockers may not have tachycardia when in shock. In patients with peptic ulcer bleeding, *H. pylori* titer level may be measured to guide future treatment, and the gastroenterologist may do a urease breath test.

The 2 primary goals of treatment of upper GI bleeds are to prevent secondary events (ie, myocardial or cerebral ischemia) by maintaining blood pressure and hematocrit and to prevent rebleeding episodes. Rebleeding during hospitalization has been associated with a 6- to 12-fold increase in mortality.

Pharmacotherapy

The newest and most effective pharmacologic treatment in controlling esophageal variceal bleeding is octreotide. Octreotide is a somatostatin analogue with a half-life of 2 hours. It decreases splanchnic blood flow and gastric secretions. Early studies on the benefit of octreotide were equivocal,[4] but the new dosing parameters (ie, 100 μg bolus followed by a constant infusion of 50 μg per hour for 48 hours) have been shown to decrease rebleeding in the first 48 hours from 40% to 10%, decrease the number of transfusions, and decrease mortality from 20% to 10%.[5] When octreotide is not available, a vasopressin infusion at a rate of 0.1 to 0.9 U per minute with a concomitant nitroglycerin drip can be used. Nitroglycerin must be used in conjunction with vasopressin because vasopressin alone can have serious cardiovascular side effects, such as hypertension, coronary artery spasm, arrhythmias, and sudden death.

In the nonacute setting, nonselective β blockers can prevent 40% of variceal bleeds, but they do not have a role in the management of acute variceal bleeding. Prophylactic banding of esophageal varices is superseding the role of nonselective β blockers in decreasing the occurrence of acute bleeds.[5,11]

Emergent administration of H_2 blockers in patients with peptic ulcer bleeding has not been proven to affect the outcome of an acute bleed. In several studies, however, proton pump inhibitors have been shown to decrease rebleeding and the need for surgical intervention.[7] Proton pump inhibitors are not available in intravenous form in the United States, so these agents must be administered through the nasogastric tube. The tube should be clamped for 30 minutes after administration to allow for absorption of the medication.[7]

The identification and eradication of *H. pylori* infection in peptic ulcer disease essentially has usurped the role of surgery in the treatment of this condition. Several studies found that patients who tested positive and were treated for *H. pylori* infection had an annual rebleed rate of 0% compared with the untreated group, where the rebleed rate remained 27% to 30%.[7] This finding has been repeated in other studies.[12]

Endoscopy

An endoscopist should be notified immediately in cases of variceal bleeding. In all cases of acute upper GI bleeding from peptic ulcer disease, an endoscopist should be notified and endoscopy performed within 24 hours, but emergent intervention is not necessary except for active bleeding. Locating the source lesion within 24 hours has been associated with improved mortality and lower rate of rebleeding.[7] In upper GI bleeding, endoscopy can be used to treat the localized lesion using a variety of tools, including injecting sclerotic agents, tissue glue, laser, and thermoablation.[11] Some authors believe that in a low-risk patient with no comorbid disease, endoscopic intervention in the ED can preclude admission to the hospital.[3,9] Currently this is not the standard of care, but it may become the standard.
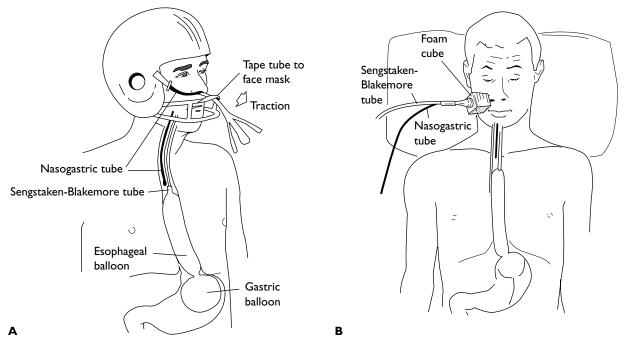
Figure 2. Methods for maintaining traction on tube during esophageal balloon tamponade. Adapted with permission from Roberts JR, Hedges JR, editors. Clinical procedures in emergency medicine. Philadelphia: WB Saunders; 1991:653.

Esophageal Balloon Tamponade

An esophageal balloon tamponade device should be placed by the bedside of any patient who is suspected of having variceal bleeding and is massively exsanguinating. The 2 models most commonly used, the Sengstaken-Blakemore tube and the Minnesota tube, have essentially the same features. It is imperative that the treating physician be familiar with the device used in the facility they practice in. The basic maneuver is to place the tube, entirely deflated and lubricated, through a naris into the stomach. It may also be placed through the mouth with a bite block in place. The gastric balloon should be inflated with 50 to 100 mL of air and then a radiograph should be done to confirm its appropriate placement in the stomach. The radiograph is important because further inflating a balloon erroneously placed in the esophagus may lead to esophageal rupture and death. After appropriate tube placement is confirmed, the gastric balloon should be fully inflated and the tube tugged up upon to occlude the gastric esophageal feeding veins. The tube should then be anchored externally to something, such as a football helmet or weight-and-pulley system (**Figure 2**). Placement of a balloon in this manner is usually adequate for halting variceal bleeding. If blood continues to flow through the esophageal port, the balloon can be inflated to 40 mm Hg. This pressure must be closely monitored by a manometer to avoid pressures

that can cause esophageal rupture (ie, > 40 mm Hg). After successful tamponade, arrangements should be made for surgical or fluoroscopic trans-hepatic shunt placement.

The balloon tamponade device is only a temporary measure and must be released within 24 hours to avoid ischemic damage to the upper GI tract. Definitive treatment for esophageal bleeding is endoscopic band ligation of the varices, although injection therapy with sclerotic agents, tissue glue, fibrin, and other agents is undergoing investigation.

Role of the Surgeon

It is widely recommended that a surgeon be notified about any patient with a significant upper GI bleed. Surgeons, however, rarely intervene because endoscopy has become the primary modality for treating even ongoing bleeding. Surgical intervention is warranted after 2 failed endoscopic attempts to stem ongoing bleeding or in the event of suspected visceral perforation.

RISK STRATIFICATION AND DISPOSITION

Several scoring systems have been developed to guide disposition of patients who present with nonvariceal bleeding (**Tables 2** and **3**). The lower the risk score, the lower the overall mortality.

Table 2. Risk Scoring System to Stratify Patients with Nonvariceal Gastrointestinal Bleeding

	Score			
Variable	**0**	**1**	**2**	**3**
Age, yr	< 60	60–79	≥ 80	
Shock	None	Pulse > 100 beats per minute	SBP ≤ 100 mm Hg	
Comorbidity	None		CHF, IHD, other	Renal or liver failure; disseminated malignancy
Diagnosis	MWT, no lesion found, no SRH	All other diagnoses	Malignancy in UGI tract	
Major SRH	None or dark spot only	Blood in UGI tract, adherent clot, visible or spurting vessel		

CHF = congestive heart failure; IHD = ischemic heart disease; MWT = Mallory-Weiss tear; SBP = systolic blood pressure; SRH = stigmata of recent hemorrhage; UGI = upper gastrointestinal. Reprinted with permission from Rollhauser C, Fleischer DE. Upper gastrointestinal nonvariceal bleeding: a review covering the years 1996-97. Endoscopy 1998;30:117.

Table 3. Outcomes of Patients with Nonvariceal Gastrointestinal Bleeding Stratified by Risk Score

		n (%)		
Risk Score	**No. of Patients (%)**	**Rebleeding Episodes**	**Mortality in Patients with Rebleeding**	**Overall Mortality**
≤ 2	744 (30%)	32 (4.3%)	0 (0%)	1 (0.1%)
3–5	1219 (48%)	173 (14%)	30 (2.5%)	56 (4.6%)
6 to ≥ 8	58 (22%)	211 (37%)	80 (14%)	126 (22%)

Reprinted with permission from Rollhauser C, Fleischer DE. Upper gastrointestinal nonvariceal bleeding: a review covering the years 1996-97. Endoscopy 1998;30:117.

LOWER GASTROINTESTINAL BLEEDING

Lower GI bleeds are defined as bleeding into the enteric lumen that originates distal to the ligament of Treitz. These are less common, are more elusive in their origin, and carry a lower mortality than upper GI bleeds. In a summary of 6 epidemiological studies, lower GI bleeding was found to occur with a 5- to 10-fold lower incidence than upper GI bleeding.[13] Lower GI bleeds are associated with an overall mortality of 5% per episode. The majority (85% to 90%) of lower GI bleeds stop spontaneously and usually do not warrant hospital admission.[14] The diagnosis and treatment of lower GI bleeds have changed greatly with the refinement of endoscopy.

PRESENTATION

Hematochezia is the presenting complaint in 90% of patients with lower GI bleeding, making a rectal examination imperative in these patients.[1] In studies of verified lower GI bleeding, 68% of patients presented with bright red blood per rectum, 43% with maroon stools, 12% with melena, 10% with syncope, and 30% with orthostatic hypotension. Abdominal pain is rare, with only 12% of patients presenting with this complaint. Use of color cards versus verbal description of patients' stool is recommended because a visual choice correlates with a higher predictive value for lower GI bleeding.[13]

Aspirin and NSAIDs have been found to increase the risk of lower GI bleeding. As in upper GI bleeds, increased age and presence of comorbidity increase the risk of lower GI bleeding and influence the prognosis.

ETIOLOGY

The challenge of treating patients with a lower GI

bleed is locating the source of the bleeding. Criteria for verifying the source of bleeding have not been standardized, and the source is often surmised from luminal stigmata found on endoscopy. Due to this lack of standardization, study results vary widely.[13]

Diverticulosis

Diverticulosis is often implicated as the leading cause of lower GI bleeding; it is an incidental finding in a large majority of patients with lower GI bleeding. The incidence ranges from 20% to 87%. Endoscopic diagnosis of diverticular bleeding is less certain in studies that use predetermined criteria such as active bleeding, adherent clot, or local blood around an exposed vessel.

Diverticuli bleed because the vasa recta vessels at the base or dome of the diverticula are attenuated and the overlying mucosa is thinned. It is thought that trauma from hard stool causes the bleed; inflammation and diverticulitis have not been shown to be causal. Diverticular bleeding is usually arterial and eventually requires colonoscopic intervention. A well-designed study of patients with active diverticular bleeding on angiography showed that 60% of the lesions were located in the right colon.[15] The majority of diverticular bleeds stop spontaneously.[13] Up to 38% of patients rebleed during hospitalization, but less than 2% of these require massive transfusions (ie, more than 4 units of packed red blood cells).[15] Patients not treated with an endoscopic intervention have a higher rate of rebleeding. Mortality at 4 years is 20%, although the cause of death is usually not exsanguination from diverticulosis but rather other disease processes.[13,15]

Angiodysplasia

Angiodysplasia is an arteriovenous malformation in the submucosa of the enteric lumen; it is found on autopsy in 1% to 2% of the general population. Angiodysplasia is associated with aortic stenosis, chronic obstructive pulmonary disease, chronic renal insufficiency, atherosclerotic disease, and collagen vascular disease. Early studies implicated arteriovenous malformation in 3% to 12% of all significant lower GI bleeds, but later studies with more sophisticated endoscopy and predetermined criteria found these malformations to be causal in up to 41% of severe bleeds.[16] Cautery ablation is usually required to prevent further bleeding episodes.

Neoplasm

Neoplasm is most commonly associated with microscopic blood in the stool, but these lesions can occasionally be the cause of gross hematochezia when they erode into underlying vessels. Studies found neoplasm to be the cause of significant lower GI bleeds in 2% to 26% of all cases.[15] Post-polypectomy bleeding accounts for up to 4% of all significant lower GI bleeds. Because bleeding due to this procedure may occur up to 15 days after surgery, the physician should ask about recent surgical/endoscopic procedures during the history. With improving endoscopic polypectomy techniques, the incidence is decreasing.

Inflammatory Diseases and Infections of the Colon

Inflammatory diseases of the colon account for 6% to 30% of all cases of lower GI bleeding. Although ulcerative colitis is found to be the cause in only 2% to 8% of all lower GI bleeds, severe lower GI bleeds account for 10% of emergent colectomies in ulcerative colitis patients.[15,16] Significant bleeding is found in only 1% of patients with Crohn's disease.

Infectious causes such as typhoid, *Escherichia coli,* and pseudomembranous colitis may cause gross hematochezia, but these cases are rarely severe.

Radiation Proctitis

Radiation proctitis is a painful, persistent cause of lower GI bleeds in persons receiving radiation therapy. In these patients, the integrity of the mucosa is often permanently altered and has very friable neovascularization that bleeds with minimal trauma. In a study of 192 patients with prostate cancer who received pelvic radiation therapy, 5% of patients experienced daily hematochezia and 9% had at least 1 episode per week.[15] Radiation proctitis can present in patients 9 to 14 months after radiation therapy. Hematochezia with this etiology usually requires admission for pain management and endoscopic treatment.

Ischemic Colitis

Ischemic colitis occurs secondary to vasospasm. It can occur in patients with a history of hypotensive episodes, atherosclerosis, dilated cardiomyopathies, atrial fibrillation, and cocaine abuse. Between 3% and 9% of all cases of lower GI bleeding can be attributed to mesenteric ischemia. Treatment is usually surgical because the bleeding is from necrotic segments of bowel that must be resected.

Aortic-Enteric Fistulas

Aortic-enteric fistulas are rare but can cause fatal massive hematochezia. These fistulas usually occur in patients with a history of vascular surgery or trauma and can present up to 14 years after surgery. They occur when compromised areas of bowel erode into the aortic wall. Classically, patients will present with a self-limiting

"herald" bleed prior to subsequent massive hemorrhage. Treatment is emergent surgery, but death is the typical outcome.

Meckel's Diverticulum

Meckel's diverticulum is an unusual but important cause of lower GI bleeding to keep in mind. The presence of gastric mucosa in the diverticulum may give rise to an ulcer in adjacent ileum, which may cause symptoms such as painless rectal bleeding. Bleeding is usually brisk and bright red.

Anal Lesions

Although anal fissures and hemorrhoids are the leading cause of bright red blood per rectum, they are not considered causes of lower GI bleeding in most studies except in HIV-positive patients. They rarely cause significant blood loss and can usually be handled with outpatient medical or surgical care.

The differential diagnosis of lower GI bleeding is different in HIV-positive patients. Hemorrhoids, which are usually excluded from the roster of etiologies of lower GI bleeding, can cause considerable blood loss in HIV-positive patients because of the high prevalence of dysfunctional platelets; they account for up to 23% of severe lower GI bleeding in the HIV patient.[15] These patients should be admitted for observation and treatment. In addition, HIV-positive patients have unique enteric lesions such as cytomegalovirus ulcers, HIV ulcers, and Kaposi's sarcoma that must be considered in the differential.[15]

Small Bowel Lesions

Small bowel lesions account for 1% to 9% of all cases of lower GI bleeding. Endoscopic techniques that use long, small-caliber fiber optics that extend the length of small bowel are replacing surgical and radiologic investigations for diagnosis.[11]

DIAGNOSTIC WORKUP

Upper GI bleeding is far more common than lower GI bleeding and must be excluded. Lack of blood in the nasogastric aspirate excludes upper GI bleeding. The nasogastric aspirate must contain bilious material to ensure that the tube has advanced past the stomach into the small bowel. Thereafter, history will help form the differential. Requisite laboratory evaluation includes a baseline hematocrit level, blood type and screening, coagulation profile, and chemistry tests. History should include questions about aspirin and NSAID use, history of diverticulosis, recent colonoscopy, recent hypovolemic shock, history of radiation therapy, atrial fibrillation, peripheral vascular disease, abdominal aneur-

ysm, penetrating abdominal wounds or vascular surgery, history of travel, and HIV status. After obtaining historical data, the next step is to locate the lesion; this information will be used to guide appropriate therapy.

The diagnostic modalities available for locating the source are colonoscopy, nuclear medicine, and angiography. Colonoscopy is increasingly becoming the most favored diagnostic modality. It not only will successfully locate the source the majority of the time, but it also can be used to treat, giving it an advantage over the other diagnostic modalities. Colonoscopy in a prepared colon is usually more successful than in an unprepared colon, so early administration of a colloid cathartic such as polyethylene glycol is recommended. Tagged red blood cell studies can detect active bleeds flowing at a rate greater than 1 mL/min. Nuclear medicine studies usually are not recommended because they take time, take patients away from the critical care environment, and are sensitive but not specific. Angiography can detect active bleeding greater than 0.5 mL per minute and can be used to stop active bleeding through embolization or vasoactive infusions. However, angiography is associated with a 9% to 10% overall complication rate, including reactions or allergies to contrast dye, femoral artery thrombosis, acute renal failure, and ischemic attacks.[13]

TREATMENT

The fundamentals of resuscitation as reviewed previously are key in treating patients with lower GI bleeding. Large-bore intravenous access should be obtained, and the patient should be resuscitated with warm saline. Hemodynamically unstable patients should receive transfusions. There is no pharmaceutical treatment for lower GI bleeding that has proven helpful, but early bowel cleansing with a colloid cathartic may aid in the early and accurate diagnosis of the offending lesion.

Jensen et al summarized several investigations and concluded that the indications for surgery typically include (1) hypotension/shock despite resuscitation efforts; (2) continued bleeding with transfusion of 6 units of blood, with or without specified source of bleeding by colonoscopy, angiography, or scintigraphy; and (3) a specific diagnosis made by colonoscopy but persistent bleeding.[16]

SUMMARY

Management of GI bleeding in the ED is centered around appropriate resuscitation and supportive care (Figure 1). Most GI bleeds stop spontaneously but merit endoscopic investigation for source within 24 hours of

presentation to minimize morbidity and mortality; therefore, a gastroenterology consult is imperative. Patients older than 65 years and with other illnesses are at higher risk for rebleed and death. The most life-threatening sources of GI bleeding are esophageal variceal bleeding and aortic-enteric fistulas. These sources must be considered and treated empirically if suspicion exists. The timely use of octreotide, proton pump inhibitors, and colonic cleaning can maximize good outcome. Endoscopy is becoming the diagnostic and treatment modality of choice in all acute GI bleeds. Surgery has an increasingly minimal role, with the exception of perforated peptic ulcers, aortic-enteric fistulas, and mesenteric ischemia.

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