Gastrointestinal Bleeding

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KEYWORDS

• Gastrointestinal bleeding • Emergency department
• Gastrointestinal hemorrhage • Peptic ulcer disease

Gastrointestinal bleeding (GIB) is a common problem encountered in the emergency department (ED) and a significant cause of morbidity and mortality. The overall mortality rate is approximately 10% and has not changed significantly in the past several decades.[1,2] The significant morbidity and mortality associated with GIB requires clinicians to be equipped with the skills to promptly diagnose, aggressively resuscitate, risk stratify, and request timely consultations. For hemodynamically or clinically unstable patients, early resuscitative measures focus on infusion of intravenous fluids or transfusion of blood products to reverse the direct consequences of bleeding; prevent end-organ damage, such as hypoxia or prerenal azotemia; and promote hemostasis.[3] Although controversy exists regarding the management of gastrointestinal hemorrhage, the use of a treatment algorithm enables emergency medicine physicians to effectively care for these patients. This article reviews the initial assessment, management, differential diagnosis, and treatment modalities available for patients presenting to an ED with GIB.

EPIDEMIOLOGY

Gastrointestinal hemorrhage may be divided into upper GIB (UGIB) and lower GIB (LGIB) as defined by bleeding originating proximal or distal to the ligament of Treitz. Both UGIB and LGIB are more common in men and older adults.[2,4,5] In general, UGIB is more common, accounting for a greater proportion of admissions in adults. The annual incidence for UGIB is estimated at 50 to 150 per 100,000 population,[2,6] whereas that for LGIB is lower, at approximately 20 to 27 per 100,000 population.[5,6] The mortality associated with UGIB is estimated to be anywhere from 6% to 13%, and even with the advent of endoscopic intervention, this mortality rate has not substantially decreased within the past 30 years.[7] This is believed secondary to the increased number of older adults presenting with GIB, who are often on antiplatelet and anticoagulation agents and often have other comorbidities associated with their disease.[3]

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Other independent markers of increased morbidity and mortality associated with GIB include the requirement of more than 5 units of packed red blood cells, hemodynamic instability, recurrent bleeding, endoscopic stigmata of recent hemorrhage, melena or hematochezia, esophageal varices, and bloody nasogastric aspirate.[1,8,9] In general, most cases of GIB are self-limited, with the majority of patients having only one episode of bleeding.[6] Compared with UGIB, LGIB has a decreased mortality rate of approximately 4%.[5] Bleeds originating from the colon require fewer blood transfusions than those originating from the small intestine (36% vs 64%).[10] In general, patients with LGIB are more likely to have higher hemoglobin levels (84% vs 61%) and less likely to go into shock (19% vs 35%).[10,11]

ETIOLOGY OF UGIB

There are several causes of UGIB, with age playing a role in determining potential etiologies. The elderly are more likely to present with bleeds secondary to peptic ulcer disease, esophagitis, and gastritis. Together these account for 70% to 90% of hospital admissions for UGIB in this age group.[12] Younger patients account for a larger percentage of cases secondary to causes such as Mallory-Weiss tears, gastrointestinal varices, and gastropathy, which are all less likely in older adults. Common causes of UGIB with prevalence are summarized in **Table 1**.

Peptic ulcer disease (PUD) is the most common cause of UGIB, accounting for approximately half of all cases.[3] PUD occurs secondary to erosion of the gastric or duodenal tissue with symptoms of a gnawing epigastric discomfort and pain that worsens after eating and with lying down. Nausea and vomiting, along with anorexia and concomitant weight loss, are symptoms that are consistent with a diagnosis of PUD. Symptoms are often treated with the use of a proton pump inhibitor or H_2 receptor antagonist. Infection with helicobacter pylori is the most common cause of PUD, with nonsteroidal anti-inflammatory drug (NSAID) use coming in second. Aspirin use, history of PUD, smoking, and alcohol use are all risk factors for PUD.[13]

Table 1
Major causes of upper gastrointestinal bleeding

Causes	Prevalence
Peptic ulcer disease	55%
Gastric ulcer	21.3%–23.1%
Duodenal ulcer	13.9%–24.3%
Esophageal varices	10.3%–23.1%
Esophagitis	3.7%–6.3%
Duodenitis	3.7%–5.8%
Gastritis	4.7%–23.4%
Mallory-Weiss tears	5%–10.2%
Angiodysplasia	6%
Neoplasm	2%–4.9%
Stomal ulcer	1.8%
Esophageal ulcer	1.7%
Dieulafoy lesion	1%

Data from Cappell M, Friedel D. Initial management of acute upper gastrointestinal bleeding: from initial evaluation up to gastrointestinal endoscopy. Med Clin North Am 2008;92:491–509.

Zollinger-Ellison syndrome, a disorder resulting in excess production of the hormone gastrin, is an uncommon cause of PUD. Although the overall prevalence of PUD as a cause of UGIB has decreased, an increase in PUD incidence secondary to NSAID use is noted in the elderly.[14]

Varices are responsible for approximately 10% to 25% of UGIB overall and 60% of UGIB in patients with cirrhosis.[15] Cirrhotic patients develop portal hypertension secondary to blockage of the portal venous system, which lends itself to portosystemic collaterals, such as varices, and variceal bleeding. In patients with liver disease, the incidence of new esophageal varices is linear over time with a rate of approximately 9% per year.[16] In addition, 30% of patients with portal hypertension and cirrhosis have bleeding secondary to these varices. Patients with portal hypertension–related bleeding, which includes esophageal and gastric varices and portal hypertensive gastropathy, have mortality rates of greater than 50% as compared with a 4% rate with bleeding from PUD.[2] Other common causes of UGI bleeds are inflammatory pathologies, such as gastritis and duodenitis, and Mallory-Weiss tears. Less common causes include angiodysplasia and Dieulafoy lesions, which are large tortuous arterioles in the gastric wall that can erode and bleed.[3]

ETIOLOGY OF LGIB

LGIB has decreased morbidity in comparison to UGIB and is often self-limited. Similar to UGIB, there are several factors that may be responsible for the bleed. The most common cause of LGIB is colonic diverticulosis, which presents with painless hematochezia. It is estimated that more than two-thirds of the population over the age of 80 are affected by diverticular disease. Approximately 60% of diverticular bleeds are found in the left aspect of the colon on colonoscopy.[17] The recurrence rate of diverticular bleeds is 25% after 4 years.[5]

After colonic diverticulosis, angiodysplasia, colitis, and postpolypectomy bleeding follow in frequency of LGIB causes. Angiodysplasia is responsible for both acute and chronic LGIB but those are often asymptomatic because they do not frequently bleed. Patients taking NSAIDs, aspirin, and anticoagulants as well as coagulopathic patients or patients with platelet dysfunction are more likely to present with LGIB from angiodysplasia.[17]

Although there are poor data on the incidence of ischemic colitis, it is proposed that the disease is becoming more prevalent secondary to an increase in elderly patients with cardiovascular disease.[17] Ischemic colitis is caused by a decrease in mesenteric blood flow as a result of hypotension or vasospasm, and patients often present with sudden onset of abdominal pain followed by diarrhea mixed with blood or hematochezia. LGIB is also a common manifestation of colitis secondary to inflammatory bowel disease but uncommonly leads to acute major gastrointestinal hemorrhage, with ulcerative colitis and Crohn's disease responsible for 0.1% and 1.2% of massive bleeds, respectively.[18] **Table 2** lists common causes of LGIB with prevalence.

Determining the source of LGIB can be a challenging task for clinicians. Although most lesions responsible for LGIB are due to colonic or anorectal disease, diagnosis may be difficult because LGIB may be intermittent and may originate from the small intestine or an upper tract source of brisk bleeding. Lower tract sources of GIB have a rebleeding rate of approximately 10% to 20%, require operative intervention in 10% to 15% of cases, and have a mortality rate of 4%.[2,5,6] Chronic LGIB is responsible for 18% to 30% of patients with iron deficiency anemia presenting to the ED.[17]

Table 2
Major causes of lower gastrointestinal bleeding

Causes	Prevalence
Diverticular disease	17%–40%
Angiodysplasia	9%–21%
Colitis	2%–30%
Inflammatory bowel disease	—
Ischemia	—
Infectious	—
Radiation	—
Postpolypectomy bleeding	11%–14%
Anorectal disease	4%–10%
Hemorrhoids	—
Rectal varices	—
Fissures	—
Small bowel bleeding	2%–9%
Upper gastrointestinal bleeding	0–11%

Data from Barnert J, Messmann H. Diagnosis and management of lower gastrointestinal bleeding. Nat Rev Gastroenterol Hepatol 2009;6:637–46.

INITIAL EVALUATION

The initial evaluation of patients presenting with GIB should focus on assessment of vital signs, the obtainment of a thorough yet focused medical history, a physical examination with particular attention paid to evidence of GIB and hemodynamic compromise, and laboratory diagnostic testing. Prompt and accurate assessment of these factors guides medical decision making, allowing for early diagnosis, aggressive resuscitation, and timely consultations. Vital signs, in particular heart rate and blood pressure, provide clinicians with diagnostic clues regarding the stability of patients. The loss of approximately less than 250 mL of blood does not usually affect heart rate or blood pressure. Greater than 800 mL of blood loss, however, may cause a drop in blood pressure of 10 mg Hg and a rise in heart rate of 10 beats per minute.[17] Significant tachycardia, tachypnea, hypotension, decreased mental status, and shock may result from blood loss totaling more than 1500 mL.[17] Although abnormal vital signs are concerning, normal vital signs do not preclude the presence of a significant bleed.

NATURE OF THE BLEED

The medical history can provide important diagnostic information with regards to the severity of disease and help focus triage and treatment of patients. Pertinent historical questions are detailed in **Box 1**. Historical questions include defining the nature of the bleed as hematemesis, hematochezia, or melena. Hematemesis usually signifies a UGIB and can be defined as bright red blood or darker coffee ground emesis. Approximately 50% of patients with UGIB present with hematemesis.[6] The return of bright red blood or coffee grounds through the passage of a nasogastric tube and subsequent nasogastric lavage has high predictive value for a bleed proximal to the ligament of Treitz; however, a negative lavage does not exclude UGIB.[3] Hematochezia is characterized by bright red or maroon-colored blood per rectum and suggests

Box 1 Pertinent historical questions
Bleeding location (distinguish upper from lower)
Bleeding severity
Nature, duration, and frequency of bleed
Risk factors (history of coagulopathy, prior GIB, alcohol intake, and liver disease)

a source distal to the ligament of Treitz. Approximately 14% of bleeds presenting with hematochezia are caused by a brisk upper source with rapid transit.[19] Hematochezia due to an upper tract source of bleeding has been shown associated with a higher transfusion requirement, need for surgery, and mortality rate.[6,19] Dark tarry or melanotic stools, which may also signify either an upper or lower source for the bleed, are associated with a lower mortality rate compared with hematochezia.[6] It is estimated that 90% of melanotic stools arise proximal to the ligament of Treitz and take on a dark tarry appearance secondary to prolonged transit and degradation of blood.[3] Approximately 70% of patients with UGIB present with melena in contrast to only 20% to 30% of patients with LGIB.[6,11] In addition to characterizing the nature of the bleed, it is important to define the duration, quantity, and frequency of bleeding episodes.

CLINICAL FEATURES

Although many patients may present with a chief complaint of hematemesis or blood in the stool, clinical suspicion for GIB must remain high in patients with signs or symptoms of hypovolemia or more subtle presentations, which may include hypotension, tachycardia, dizziness, angina, confusion, or syncope. Additional clinical features that should be ascertained as part of the medical history include questions pertaining to hypovolemia, including lightheadedness and dizziness, and symptoms suggestive of anemia, such as chest pain, dyspnea, and fatigue. Gastrointestinal historical features themselves may help delineate the possible etiology of the bleed. For example, epigastric pain is often associated with duodenal ulcers, whereas pain relieved with food intake is suggestive of a gastric ulcer. Mesenteric ischemia may present with significant abdominal pain out of proportion to physical examination findings. If a patient complains of streaks of blood present after vomiting, retching, or coughing, a Mallory-Weiss tear should be considered.

Medications and past medical, family, and social histories are also useful tools to help diagnose patients presenting with signs and symptoms suggestive of GIB. Medication history includes patient use of NSAIDs, aspirin, glucocorticoids, anticoagulants, and antiplatelet agents. Both aspirin and NSAID use have been shown to contribute to UGIB and LGIB, which are dependent on dose and duration of use.[11,20,21] Additionally, the use of aspirin (<100 mg/d), anticoagulation agents in the therapeutic range, and antiplatelet agents, such as clopidogrel, all increase the risk of UGIB threefold.[22,23] Important past medical history includes episodes of prior gastrointestinal hemorrhage, because up to 60% of UGI bleeds arise from prior gastrointestinal lesions that have bled.[24] Other relevant medical history includes presence of hemorrhoids, hepatic disease, coagulopathies, vascular disease, HIV infection, prior radiation therapy for prostate or pelvic cancer, inflammatory bowel disease, and recent colonoscopy with polypectomy. Family history of colon cancer and social history, specifically pertaining to alcohol and tobacco intake, also help to risk stratify patients. Both alcohol

and cigarette smoking are associated with gastrointestinal malignancies.[25] Alcohol abuse is independently associated with an increased incidence of PUD and may also result in cirrhosis and varices.[25]

PHYSICAL EXAMINATION

Physical examination should include assessment of general appearance, mental status, examination of the conjunctiva (with pallor suggesting anemia), and skin characteristics, including color and temperature. Presence of petechiae or ecchymoses on skin examination may indicate a coagulopathy. Extremities should be evaluated for character of pulses and adequacy of capillary refill. Cool, clammy, pale extremities often indicate hypovolemic shock. Patients with cirrhosis may present with stigmata of liver disease, which include jaundice, caput medusa, palmar erythema, ascites, and hepatomegaly. Examination of the nasopharynx and oropharynx may reveal a source of blood that is being swallowed. A thorough abdominal examination is important and includes auscultation for bowel sounds and assessment of tenderness, masses, and signs of peritonitis. Blood from a proximal source can be irritating to the gastrointestinal tract and thereby stimulate peristalsis, leading to hyperactive bowel sounds. On the converse, LGIB presents with normoactive bowel sounds. Significant abdominal tenderness, involuntary guarding, or rebound tenderness should raise concern for a perforation, which needs to be excluded before endoscopy or colonoscopy is performed. Rectal examination should include inspection for anal fissures or hemorrhoids, assessment for rectal masses, and evaluation of stool for color and occult blood.

INITIAL DIAGNOSTIC TESTING

Laboratory studies to obtain in patients with GIB include a type and crossmatch, hemoglobin and hematocrit levels, blood urea nitrogen (BUN) and creatinine, coagulation profile, platelet count, and liver function tests. The initial hemoglobin level may not adequately reflect the true amount of blood loss in an acute bleed, because it often takes more than 24 hours to manifest a change in hemoglobin level. Infusion of intravenous fluids and movement of extracellular fluid into intravascular spaces can also confuse the initial and subsequent serial analyses of hemoglobin levels. If available, prior hemoglobin levels for comparison are helpful in determining the severity of the bleed. A hemoglobin level of less than 10 g/dL has been associated with increased rebleeding and mortality rates.[8] Because an elevation of the BUN concentration is associated with UGIB, the BUN-to-creatinine ratio has been used to distinguish UGIB from LGIB. In patients without renal failure, a BUN-to-creatinine ratio greater than 36 has a sensitivity of 90% to 95% in predicting UGIB.[26,27] Coagulation profile and platelet count may be beneficial in patients with liver disease or on anticoagulation therapy.

Because significant GIB may lead to decreased oxygen delivery and resultant myocardial ischemia and infarction, an electrocardiogram and cardiac markers should be obtained in patients at risk for acute coronary syndrome.[28,29] Patients with GIB who are diagnosed with concurrent myocardial infarction often do not complain of chest pain but rather dyspnea, dizziness, or abdominal pain.[30,31] In patients with GIB, routine abdominal radiographs have been shown of limited value.[32] In addition, routine chest radiographs have not been found to alter clinical outcomes or management decisions in the absence of pulmonary examination findings or known pulmonary disease.[33]

NASOGASTRIC ASPIRATION

Nasogastric aspiration detects active bleeding and may be helpful in the management of some patients with GIB. Once considered important in the initial management of GIB, there is now controversy surrounding its clinical utility, especially in light of the significant patient discomfort with its use. In patients with hematemesis and a clear upper source of GIB, nasogastric aspiration with lavage may be used to quantify active bleeding. In patients presenting without hematemesis and an unclear source of bleeding, a bloody nasogastric aspirate indicates an upper source of bleeding and may help direct management. In these patients without hematemesis, nasogastric aspiration has a limited sensitivity (42%) and accuracy (66%) in detecting UGIB, although a positive aspirate has been demonstrated in 23% of cases with a likelihood ratio of 11.[34] Bright red blood from the nasogastric lavage suggests an active bleed, whereas darker coffee grounds suggest a recent bleed and slow rate of bleeding. A massive hemorrhage should be suspected in patients with continued bright red blood in the aspirate. A bloody aspirate has been shown associated with the presence of a high-risk lesion at endoscopy.[35]

In contrast, a negative nasogastric aspirate is not helpful because it may miss various sources of UGIB and has been shown to miss up to 50% of patients with recent duodenal bleeding.[34,36] A nonbloody nasogastric aspirate may indicate that the source of the bleed is distal to the ligament of Treitz or that the bleeding has ceased. Although nasogastric tube placement is one of the more painful commonly performed procedures in the ED, use of lubrication, proper positioning (sitting up with head tilted forward), and patient involvement (swallowing during placement) may facilitate insertion.[37] In addition, topical anesthesia can decrease pain by two-thirds.[38] Complications of nasogastric tube insertion include epistaxsis, aspiration, pneumothorax, perforation, and gastric lesions.[39,40]

OTHER CAUSES OF GASTROINTESTINAL BLEEDING

Some patients who complain of vomiting blood or passing blood in the stool may not have GIB. The source of suspected UGIB may actually be due to blood from the nasopharynx or oropharynx, which is being swallowed. Vomitus may appear as hematemesis due to red colored food products. Iron or bismuth ingestion may lead to the appearance of melena, whereas ingestion of beets may result in the appearance of hematochezia. In these cases, fecal occult testing is heme negative.

EMERGENCY DEPARTMENT MANAGEMENT

ED care encompasses timely assessment with immediate resuscitation and management of patients with GIB. Because the loss of oxygen-carrying capacity from hemorrhaging can lead to hypoxia and tissue ischemia, supplemental oxygen and pulse oximetry are recommended. If indicated, definitive airway management may be required in patients unable to support their airway and to prevent aspiration. It is also recommended that patients be placed on cardiac monitoring secondary to the risk of demand ischemia from decreased oxygen delivery to cardiac tissue. Patients with significant GIB should be kept nothing by mouth to facilitate the need for emergent endoscopy or surgical intervention.

FLUID RESUSCITATION AND TRANSFUSION OF BLOOD PRODUCTS

Two peripheral large-bore (18-gauge or larger) intravenous catheters are recommended with aggressive resuscitation initiated with crystalloid infusion. Assessment of

volume status and hemodynamic stability should dictate the amount of fluid transfused. In hemodynamically unstable patients or patients with large hemorrhage, boluses of 500 mL of normal saline or lactated Ringer solution with continuous reassessment are appropriate. Transducing a central venous pressure via a central line or the use of a Swan-Ganz catheter is a more accurate method of determining volume status; however, determining the need for invasive monitoring should be based on the clinical presentation and need for close assessment and management of volume status.

Transfusion of blood products is recommended in those patients with hemodynamic instability despite crystalloid resuscitation and in those with continuous bleeding. A patient's age, comorbid conditions, briskness of bleed, baseline hemoglobin and hematocrit levels, and evidence of cardiac, renal, or cerebral hypoperfusion should all be taken into consideration when determining the quantity of blood to transfuse. Although there are no hard-set transfusion parameters for GIB, it has been suggested that patients with variceal bleeding do not benefit from aggressive transfusion, because this increases portal pressure and may lead to more bleeding.[41] A small study comparing 25 cirrhotic patients transfused with 2 units or more of packed red blood cells with 25 cirrhotic patients transfused only for a hemoglobin level of less than 8 g/dL or hemodynamic compromise demonstrated a higher risk of rebleed in the patients who were aggressively transfused.[42] Achieving a goal hematocrit level of 27 is recommended for patients with variceal bleeding.[3] Although a hematocrit level of 25 to 27 may be adequate to maintain perfusion in younger GIB patients with no comorbid conditions, older patients, in particular those with cardiovascular disease, may require more aggressive resuscitation and a higher hematocrit level.[43] Attention should be paid to the precipitation of flash pulmonary edema or worsening congestive heart failure in patients with pre-existing congestive heart failure, and blood should be transfused slowly if possible. Patients requiring more than 5 units of packed red cells have a much higher mortality rate and often require surgery.[1]

Coagulopathic patients may require platelets or fresh frozen plasma as appropriate. To replace coagulation factors, it is recommended that for every 4 units of packed red blood cells transfused, a patient be given 1 unit of fresh frozen plasma.[44] Patients with platelet counts in the range of 50,000 to 90,000 platelets/μL do not require platelet transfusion, whereas those with counts less than 50,000 platelets/μL and active bleeding may require transfusion.[45] Clinical parameters, including age, comorbid conditions, and severity of bleed, should be used to determine whether or not platelets should be administered to thrombocytopenic patients.

MEDICAL THERAPIES

Various medications have been demonstrated to improve outcome in GIB. Somatostatin and octreotide, its longer-acting derivative that inhibits mesenteric vasodilation induced by glucagon, have been shown to decrease the risk for persistent bleeding and rebleeding in patients with an upper source of bleed in both variceal and nonvariceal bleeding.[46,47] A large systematic review of the use of somatostatin analogs for acute esophageal variceal bleeding revealed no significant decrease in mortality but did demonstrate a reduction in bleeding and transfusion requirement.[48] The recommended dose for octreotide is a 50-μg bolus intravenously followed by a continuous infusion of 50 μg per hour.

Proton pump inhibitors have been shown to reduce the risk of rebleeding and the need for surgical intervention and blood transfusions, with conflicting evidence for mortality reduction, in patients with UGIB due to PUD.[49,50] Proton pump inhibitors are recommended before endoscopy, because they reduce the likelihood of bleeding

or need for intervention (cauterizing of vessels or injection of medications) during endoscopy.[51] Vasopressin has also been used for GIB, most often for variceal bleeding, but is associated with a significant rebleeding rate and a high rate of complications, which include hypertension, dysrhythmias, myocardial and peripheral ischemia, and decreased cardiac output.[52] The use of H_2-receptor antagonists in UGIB has not been shown to be of significant value, with a lack of benefit in bleeding duodenal ulcers and a possibly weak benefit in bleeding gastric ulcers.[53,54]

CONSULTATION AND DISPOSITION

Gastroenterology should be consulted promptly for significant GIB. The decision for emergent versus nonemergent endoscopic evaluation may be based on clinical parameters, including severity of the bleed and hemodynamic stability of the patient. Unstable patients, including those with hemodynamic instability, patients in shock, patients demonstrating a change in mental status, and those with signs of end-organ ischemia secondary to volume loss, may require intensive care unit admission and intensivists should be involved early in the care. Surgical consultation for continued hemorrhage may be necessary in those patients with UGIB or LGIB who fail to respond to standard therapies. Patients with signs of cardiac ischemia or infarction may require cardiology consultation. Hospital admission is needed for patients with significant GIB. Various studies have demonstrated a subset of low-risk patients who may be discharged safely with UGIB, although short observation and endoscopy were performed in all of these study patients before discharge home. These low-risk patients were under 60 years of age with follow-up care and had no significant comorbid conditions, no signs of shock, no history of liver disease or varices, no severe anemia, and no frequent hematemesis or melena.[55–57] Risk stratification for patients with lower sources of bleeding has not been well studied; therefore, most patients with significant LGIB are admitted for further management.

UPPER ENDOSCOPY

Although endoscopy is often performed once resuscitation is achieved, urgent endoscopy may be necessary if clinically warranted. After hemodynamic stabilization, upper endoscopy is the most accurate intervention in UGIB patients and allows for the diagnosis of the bleeding site with achievement of hemostasis in more than 90% of cases.[58] Endoscopic therapies, including sclerotherapy and band ligation, are useful in UGIB due to varices. Early esophagoduodenoscopy (EGD), performed within 12 to 24 hours of bleeding, reduces the risk of rebleeding and hospital length of stay for patients with UGIB.[59] Endoscopic findings are also useful in risk stratification and aid in the decision-making process regarding patient disposition.[59] Balloon tamponade with a Sengstaken-Blakemore tube is rarely used due to its high complication rate, but it may be helpful in exsanguinating patients due to active variceal bleeding when endoscopy is not available.

COLONOSCOPY

In patients with acute LGIB, the treatment goal is localization of the bleeding site for therapy, to ultimately avoid a subtotal colectomy. As a brisk UGIB may be the source of hematochezia, EGD may be performed before colonoscopy in unstable patients with bright red blood per rectum.[17] Otherwise, colonoscopy is indicated to attempt to identify the source of the LGIB. Colonoscopy is often performed in stabilized patients with self-limited bleeding or in those with a higher likelihood of a localized lesion. Although

urgent colonoscopy requires both rapid bowel preparation and availability of the endoscopist, it may be both diagnostic and therapeutic in cases of LGIB.[5,60] Severe hemorrhage may necessitate an urgent colonoscopy without bowel preparation, but it is recommended that the bowel be purged before evaluation to facilitate visualization and decrease the incidence of bowel perforation from poor visualization. Bowel preparation consists of administering 3 to 6 L of polyethylene glycol solution, which may be difficult for patients to tolerate. Administration of the solution through a nasogastric tube or adding an antiemetic with promotility properties, such as metoclopramide (10 mg intravenously), may be helpful. Urgent colonoscopy without bowel preparation is diagnostic approximately 89% to 97% of the time.[61,62] Colonoscopy is usually performed 24 to 48 hours after initial presentation.[61] The risk of serious complications for patients undergoing colonoscopy is 1:1000.[17] If neither EGD nor colonoscopy allows for visualization of the source of bleeding, other endoscopic techniques may be used. These include push enteroscopy, where approximately 100 cm of the proximal jejunum may be visualized, and wireless video capsule endoscopy, where 80% of the small bowel may be seen.[63] Patients with clinical signs of severe hemorrhage may be more likely to undergo radiographic interventions than urgent colonoscopy.[64]

NUCLEAR SCINTIGRAPHY

Nuclear scans, in particular those involving technetium Tc 99m–labeled red blood cells, are a noninvasive technique that may be used in localizing an obscure source of LGIB. Nuclear scintigraphy is less specific but more sensitive than angiography for detecting bleeds and requires active hemorrhage at a rate of at least 0.1 mL per minute.[6,65–67] On average, approximately 45% of technetium Tc 99m–labeled red blood cell scans performed for LGIB are positive.[10] The source of the bleed is identified correctly approximately 95% to 100% of the time with a positive scan within 2 hours of erythrocyte injection, with the accuracy dropping to 57% to 67% with a positive scan 2 hours after the injection.[10] Nuclear scintigraphy is useful for bleeds that are difficult to visualize using EGD or colonoscopy and in recurrent bleeds. Technetium scans are then used to direct therapy through the use of angiography or surgery.

ANGIOGRAPHY

Angiography may also be used to localize obscure lower bleeding but requires a brisker rate of hemorrhage of at least 0.5 to 1 mL per minute. The sensitivity of angiography has been demonstrated to be 46% for acute bleeds and 30% for recurrent bleeds, whereas the specificity approaches 100%.[68] Although angiography has the advantage of allowing for therapeutic intervention, it also has the disadvantage of complications, which include acute renal failure, contrast reactions, arterial thrombosis or dissection, and bowel infarction. The complication rate for visceral angiography is approximately 9.3%.[68] Angiography is often reserved for massive, continuous LGIB, particularly when endoscopy is unfeasible or difficult secondary to the briskness of the bleed.[6,15] The management of LGIB is controversial as to which diagnostic test should be performed initially, and management may be influenced by institutional availability and expertise, and the preference of the consulting physician.

OTHER STUDIES

Barium studies do not play a role in the evaluation of LGIB. CT or plain film radiography might be indicated when an acute abdomen is suspected, such as with bowel

perforation, or for further evaluation of bowel pathology, such as inflammatory bowel disease. Chronic bleeds may benefit from CT colonography, but lesions such as angiodysplasia are not visualized using this method.

SUMMARY

GIB is a common complaint encountered in an ED and frequent cause of hospitalization. Important diagnostic factors that increase morbidity and mortality include advanced age, serious comorbid conditions, hemodynamic instability, esophageal varices, significant hematemesis or melena, and marked anemia. Because GIB carries a 10% overall mortality rate, emergency physicians must perform timely diagnosis, aggressive resuscitation, risk stratification, and early consultation for these patients.

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